Post-exercise oxygen consumption and heart rate recovery as possible measures of the homeostatic stress of an exercise bout

BY

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DECLARATION

PhD THESIS TITLE:

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As I near the end of this thesis journey, I am slowly starting to see and appreciate all the good that has come out of it and in how many ways I have been fortunate. In acknowledging how I have been fortunate, I should really start with my parents, who have loved me, supported me and made significant sacrifices to provide me with my first few years of tertiary education. I deeply appreciate how they have never exerted the slightest pressure on me to choose a more "reliable" career path but have let me choose to do what interests me most. Nevertheless, I predict great relief on their part when I finally a) graduate and b) find a job.

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Finally, by far the most valuable part of this thesis experience has been a deeper understanding of James 1 v 2-5

"*Consider it a sheer gift, friends, when tests and challenges come at you from all sides. You know that under pressure, your faith-life is forced into the open and shows its true colours. So don't try to get out of anything prematurely. Let it do its work so you become mature and well-developed, not deficient in any way. If you don't know what you're doing, pray to the Father. He loves to help.*" (*Message* translation)

I hope to remember this lesson long after I have forgotten the finer details of this thesis. Therefore, my greatest thank-you goes to my God and Father, who loves me and helps me far more than I can understand. He has used every aspect of this thesis to do my faith and my character much good.

TABLE OF CONTENTS

PUBLICATIONS AND PRESENTATIONS ASSOCIATED WITH THE THESIS

Thesis Publications

Mann T, Lamberts RP, Lambert MI *Methods of prescribing relative exercise intensity physiological and practical considerations* Sports Medicine 2013 43(7):613-25. PMID: 23620244

This paper developed from discussions between the PhD candidate and her two supervisors, Professor Mike Lambert and Dr Robert Lamberts. The PhD candidate conceived the idea, reviewed the literature thoroughly and wrote the first draft of the paper. Professor Lambert and Dr Lamberts provided feedback and editorial support for the final version.

Mann T, *"Mean response" disregards the importance of individual variation* South African Journal of Sports Medicine 2011 23(1) 30

Thesis conference presentations

(Scheduled) 44th German Congress of Sports Medicine, 6-7th September 2013, Frankfurt, Germany. Invited speaker on the topic of "*Pro's and con's of lactate vs. cardiopulmonary measurements for prescribing training intensity*"

17th Annual Congress of the European College of Sports Sciences (ECSS), 4-7th July 2012, Bruges, Belgium. Oral presentation "*Factors explaining training-related differences in* EPOC *halflife*"

14th Biennial Congress of the South African Sports Medicine Association (SASMA), 18-20th October 2011, Johannesburg, South African. Oral presentation "*Variation in measurements of recovery following a standardized exercise bout in trained and untrained individuals*"

THESIS ABSTRACT

Several methods have been used to quantify the internal training load of a bout of exercise. However, a recent novel approach to quantify the internal training load has been to investigate the dynamic return towards resting homeostasis at the cessation of exercise. Objective and non-invasive methods of monitoring the return towards resting homeostasis include measures of heart rate recovery (HRR) and excess post-exercise oxygen consumption (EPOC). However, the relative potential of autonomic- vs. metabolic-type recovery measurements to represent the internal training load or homeostatic stress of the preceding exercise bout has not been established. Therefore, the broad aim of this thesis was to investigate the magnitude of EPOC ($EPOC_{MAG}$), the time constant of the EPOC recovery curve ($EPOC\tau$), HRR within the first minute post-exercise (HRR_{60s}) and the time constant of the HRR curve ($HRR\tau$) as measures which might reflect the homeostatic stress of an exercise bout. It was hypothesized that a measure representing the homeostatic stress of an exercise bout could have the following possible applications;

- to identify inter-individual variation in the homeostatic stress of a standardized exercise bout
- to detect intra-individual variation in the homeostatic stress of different exercise bouts
- to detect intra-individual variation in "readiness to train", based on the response to a standardized exercise bout

Therefore, the investigations of this thesis aimed to assess the relative potential of $EPOC_{MAG}$, $EPOC\tau$, HRR_{60s} and $HRR\tau$ in these different roles. The experimental work was divided into 4 studies.

The aim of the first study was to investigate which of the 4 recovery outcome measures was most closely associated with Borg's Rating of Perceived Exertion (RPE) during standardized exercise among participants with a range of fitness levels. Although RPE may be influenced by subjective factors, it is widely acknowledged as an integrated measure of the homeostatic stress of an exercise bout and it was anticipated that a recovery measurement sensitive to the internal training load or homeostatic stress of an exercise bout would be associated with inter-individual differences in RPE. A heterogeneous group of trained and untrained participants (n = 36, 14 Males (M), 22 Females (F)) completed a bout of exercise on the treadmill (3 km at 70% of maximal oxygen uptake (VO_2max)) followed by 1 hour of controlled recovery. Expired respiratory gases and heart rate (HR) were measured throughout the exercise and recovery phases of the trial with recovery measurements used to calculate the $EPOC_{MAG}$, $EPOC\tau$, HRR_{60s} and $HRR\tau$ responses for each participant. It was found that RPE taken in the last minute of exercise had 48% of variation in common with HRR_{60s}, 23-26% of variation in common with $EPOC\tau$ or $HRR\tau$ ($p < 0.05$) and no significant association with $EPOC_{MAG}$. This finding suggests that, of the 4 recovery measurements under investigation, HRR_{60s} showed the most potential to represent inter-individual variation in the homeostatic stress of a standardized exercise bout, in a group with a wide range of fitness levels.

In the second, third and fourth studies, the context of the investigations shifted from inter-individual variation in recovery outcomes to intra-individual variation in recovery outcomes. With this in mind, the aim of the second study was to determine the day-to-day variation of each recovery measurement. It was anticipated that determining the day-to-day variation in each measurement would improve the interpretation of the individual changes in recovery responses in the third and fourth studies. Twelve moderately-trained runners (4 M, 8 F) completed 3 repetitions of a submaximal treadmill and recovery protocol on consecutive days. This protocol was the same protocol to be used in the third and fourth studies and consisted of a 5 min warm-up, a 20 min constant-intensity exercise at 70% VO_2max and 15 min of controlled recovery. Expired respiratory gases and HR were measured throughout the 20 min exercise and recovery period and recovery measurements were used to calculate $EPOC_{MAG}$, EPOCτ, HRR_{60s} and HRRτ. The typical error of each measurement (expressed as a coefficient of variation with the associated 90% confidence limits) was 8.0 % (6.7-10.3 %) for $EPOC_{MAG}$, 12.9 % (10.6-16.4%) for EPOCτ, 8.7 % (7.2-11.2 %) for HRR_{60s} and 10.0 % (8.2-12.8 %) for HRRτ. It was anticipated that, in the third and fourth studies, individual changes in excess of the typical error of a measurement could be interpreted as having practical significance whereas changes less than- or equal to- the day-to-day variation could be interpreted as having little practical significance.

The aim of the third study was to investigate which of the 4 recovery outcomes was most sensitive to changes in exercise intensity. It was anticipated that increased exercise intensity would increase the homeostatic stress of the exercise bout which, in turn, would result in slower recovery towards resting homeostasis. Therefore, a recovery measurement suitable to represent the homeostatic stress of an exercise bout would be expected to show slower recovery with increased exercise intensity, sensitivity to both smaller- and larger- changes in exercise intensity and consistent responses on an individual level as well as on a group mean level. Thirty two moderately-trained runners (20 M, 12 F) completed 20 min of treadmill exercise at 60%, 70% and 80% VO_2max, on separate days and in random order. Each exercise bout was followed by 15 min of controlled recovery and expired respiratory gases and HR were measured throughout the exercise bout and recovery period. When recovery responses at 60%, 70% and 80% of VO_2max were compared at the group mean level, $EPOC_{MAG}$, EPOCτ and HRRτ all reflected slower recovery with increased exercise intensity. However, only $EPOC_{MAG}$ was significantly different across all 3 exercise intensities (d = 0.5 to 1.2)($p < 0.05$). In contrast, HRR_{60s} reflected faster recovery in the 70% vs. 60% VO_2max and 80% vs. 60% VO_2max trials (d = 0.6 to 0.7)($p < 0.05$) but was not different between the 70% and 80% VO_2max trials. At an individual level, changes in $EPOC_{MAG}$ were meaningful and in a consistent direction in the majority of individuals whereas other recovery measurements did not respond as consistently to changes in exercise intensity. The main finding of the study was that only $EPOC_{MAG}$ was sensitive to both larger- and smaller-changes in exercise intensity and showed the slower recovery responses with increased exercise intensity that were anticipated from a possible measure of the homeostatic stress of an exercise bout.

The aim of the fourth and final study was to compare recovery responses before and after an acute training "overload" in the form of an ultra-marathon road race. It was anticipated that the capacity to respond to training or "readiness to train" would be impaired in the days after the race and that a recovery measure with potential to monitor changes in readiness to train would be sensitive to this change. Ten runners (8 M, 2 F) completed a standard protocol of 20 min treadmill running at 70% VO_2max followed by 15 min of controlled recovery 7 days before the 87 km race and again 3 days after the race. Although there was an increase in perceived muscle soreness and RPE after the race (p <0.05), there was no significant change in either $EPOC_{MAG}$ or EPOCτ. In contrast, both HRR_{60s} and HRRτ reflected significantly faster HRR after the race at the group mean level (d = 0.9 to 1.0)(p < 0.05). On an individual level, 7 out of 10 runners showed faster HRR_{60s} responses after the race whereas 5 out of 10 runners showed faster HRRτ responses. Therefore, of the 4 recovery measurements investigated, HRR_{60s} was the most sensitive to the effect of the ultra-marathon. However, the main finding of the study was that changes in HRR should be considered along with other measurements (e.g. muscle soreness, RPE) as faster HRR may not necessarily indicate an increased readiness to respond to training.

In conclusion, both EPOC and HRR represent the return towards resting homeostasis at the end of a bout of exercise. However, the findings this thesis show that different forms of these metabolic- or autonomic-recovery measurements respond differently with regards to sensitivity to inter-individual differences in the homeostatic stress of an exercise bout, intra-individual differences in the homeostatic stress of an exercise bout or intra-individual differences in readiness to train. Therefore, in response to the title of the thesis, *Post-exercise oxygen consumption and heart rate recovery as possible measures of the homeostatic stress of an exercise bout*, it can be concluded that both post-exercise oxygen consumption and HRR have potential as measures of the homeostatic stress of an exercise bout, depending on the form in which it is measured and the context of the application.

LIST OF FIGURES

CHAPTER 1

CHAPTER 2

CHAPTER 4

CHAPTER 5

CHAPTER 6

CHAPTER 7

CHAPTER 8

APPENDIX

LIST OF TABLES

CHAPTER 2

CHAPTER 3

CHAPTER 4

CHAPTER 5

CHAPTER 6

CHAPTER 7

.

LIST OF ABBREVIATIONS

ACSM	American College of Sports Medicine
AerT	Aerobic Threshold
AMP	Adenosine Monophosphate
AnT	Anaerobic Threshold
AP	All Participants
ATP	Adenosine Triphosphate
BF	Breathing Frequency
BMI	Body Mass Index
BP	Blood Pressure
CL	Confidence Limits
CV	Coefficient of Variation
CV_{TEM}	Typical Error of Measurement as a Coefficient of Variation
DALDA	Daily Analysis of Life Demands for Athletes
DXA	Dual X-ray Absorptiometry
EE	Energy Expenditure
$EE \cdot kg^{-1}$	Energy Expenditure per kilogram
$EE \cdot kgFFM^{-1}$	Energy Expenditure per kilogram Fat Free Mass
EPOC	Excess Post-exercise Oxygen Consumption
$EPOC_{MAG}$	Magnitude of Excess Post-exercise Oxygen Consumption
$EPOC\tau$	Time Constant of Excess Post-exercise Oxygen Consumption Recovery Curve
F	Female
HR	Heart Rate
HRmax	Maximal Heart Rate
HRR	Heart Rate Recovery

HRR_{60s}	60 second Heart Rate Recovery
HRRτ	Time Constant of the Heart Rate Recovery Curve
HRres	Heart Rate reserve
IAT	Individual Anaerobic Threshold
LT	Lactate Threshold
M	Male
MLSS	Maximal Lactate Steady State
n	Number of participants
n/a	Not applicable
OBLA	Onset of Blood Lactate Accumulation
PAR-Q	Physical Activity Readiness Questionnaire
PO	Power Output
PTRS	Peak Treadmill Running Speed
RER	Respiratory Exchange Ratio
RPE	Rating of Perceived Exertion
SD	Standard Deviation
TEM	Typical Error of Measurement
TP	Trained Participants
TRIMP	Training Impulse
VE	Minute Ventilation
V_E	Ventilatory Equivalent
VO_2	Oxygen Uptake
VO_2max	Maximum Oxygen Uptake
VO_2R	Oxygen Consumption Reserve
VT	Ventilatory Threshold

MEASURING UNITS

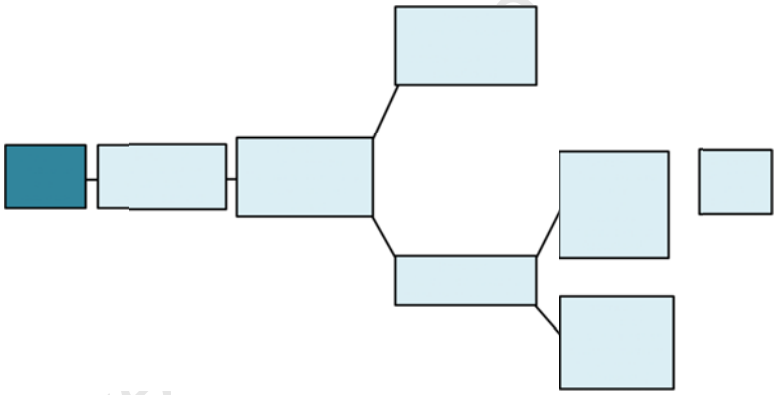
bpm	beats per minute
cm	centimetre
h	hour(s)
kcal	kilocalories
kg	kilogram
$kg \cdot m^2$	kilograms times metre squared
km	kilometres
$km \cdot h^{-1}$	kilometres per hour
$km \cdot wk^{-1}$	kilometres per week
L	litres
$l \cdot min^{-1}$	litres per minute
m	metres
min	minute(s)
$m \cdot min^{-1}$	metres per minute
ml	millilitre
$ml \cdot kg^{-1}$	millilitres per kilogram
$ml \cdot kg^{-1} \cdot min^{-1}$	millilitres per kilogram per minute
$ml \cdot min^{-1}$	millilitres per minute
mmHg	millimetres of mercury
$mmol \cdot l^{-1}$	millimoles per litre
$pmol \cdot l^{-1}$	picomoles per litre
$runs \cdot wk^{-1}$	runs per week
s	second(s)
$Sessions \cdot wk^{-1}$	Sessions per week
W	Watts
°C	degrees Celsius

DEFINITION OF TERMS

Training status	The integration of physiological, morphological, autonomic and metabolic adaptations following exercise training. Although the training status can be classified anywhere on a spectrum, for the purpose of this thesis the categories are defined as untrained, moderately trained, well-trained.
Training response	The relative or absolute change in one or more target outcome measures as a result of adaptations in response to a period of exercise training (e.g. change in submaximal heart rate).
Exercise response	The relative or absolute change in one or more physiological, metabolic or autonomic parameters during an exercise bout e.g. increased heart rate, increased metabolic rate.
Homeostatic stress	The overall disturbance to resting homeostasis arising from the interaction of the intensity, duration and mode of the exercise bout. This disturbance or "stress" can also be viewed as a biological stimulus to induce exercise-specific adaptations, provided that the individual has an adequate "readiness to train" (*see definition below*). This definition of homeostatic stress will be used synonymously with the "training load" of an exercise bout.
Training load	Overall disturbance to resting homeostasis arising from the interaction of the intensity, duration and mode of the exercise bout, also known as the "internal" training load. "Training load" will be used synonymously with the term "homeostatic stress" (*see definition above*).
Readiness to train	The extent to which an individual has recovered from recent training sessions and is able to tolerate and adapt to the next training session.

CHAPTER 1

INTRODUCTION AND OVERVIEW



1.1 BACKGROUND

The response to a particular exercise intervention is often described in general terms, with the assumption that the group average represents a typical response for most individuals. In reality, however, it is common for some individuals to show responses much larger or much smaller than the group average response [1–7]. This phenomenon of "high responders" and "low responders" following a standardized training intervention has important implications for training prescription, particularly when the training has been prescribed to treat or prevent lifestyle diseases and/or to produce an improvement in exercise performance. There are a number of factors that may explain individual variation in response to training and these factors are reviewed in Chapter 2.

In the relatively small number of studies which have focused on individual variation in response to endurance-type training program [2–5,7–10], training was standardized according to the relative intensity, frequency and duration or caloric expenditure of the training sessions with the intention that all participants would receive a similar exercise stimulus. However, a common theme among these studies has been the use of percentage (%) maximal oxygen uptake (VO_2max) or % maximal heart rate (HRmax) to prescribe the relative intensity of the training sessions [2,3,5,7–9] and these methods may in fact contribute to individual variation in training response. To be specific, at a fixed %VO_2max (or %HRmax) of moderate-to-high intensity, some individuals may be exercising above- and others below- metabolic thresholds such as the aerobic threshold and anaerobic threshold [11–13]. With this in mind, it has been suggested that individual variation in the homeostatic stress of the exercise bout may contribute to individual variation in the exercise stimulus associated with each exercise bout and, over time, contribute to individual variation in the adaptive response to standardized training [7,12–14]. Authors that have criticized the use of %VO_2max or %HRmax to standardize exercise intensity have argued that exercise prescribed relative to the aerobic or anaerobic threshold would be a more effective means of prescribing an equivalent relative intensity among different individuals [7,12,14]. Therefore, to investigate this premise, the physiological and practical implications of prescribing exercise intensity relative to VO_2max or HRmax vs. the aerobic threshold or anaerobic threshold were reviewed in Chapter 3.

1.2 THESIS TOPIC

While each method of relative exercise intensity prescription may have pro's and con's, one limitation that generally affects all approaches is that methods of relative exercise intensity prescription tend to standardize one aspect of the exercise response while other aspects of the

exercise response are free to vary between individuals. It follows that, on some level, individual variation in the homeostatic stress of each exercise bout is likely to contribute to individual variation in training response regardless of which method of relative exercise intensity prescription is used.

Although it is difficult to prescribe an equivalent homeostatic stress prospectively, it may be possible to quantify the homeostatic stress of an exercise bout retrospectively through measures of (internal) training load. It was hypothesized that a measure of training load, representing the homeostatic stress of an exercise bout, could have a number of possible applications. For example, a measure of training load could potentially be used to detect inter-individual variation in the overall homeostatic stress of a standardized exercise bout and perhaps help to predict relatively high- or relatively low- responders to further training of a similar kind. Furthermore, a measure of training load could potentially be used to detect intra-individual variation in the homeostatic stress of different exercise bouts and provide insight into the relationship between exercise dose and adaptive response for a particular individual. Finally, intra-individual variation in the training load associated with a standardized exercise bout, performed at regular intervals, could potentially have application as a monitoring tool, perhaps identifying when an individual shows signs of fatigue and reduced training tolerance or signs of adaptation and possible readiness for an increase in training load.

At present, there is no gold standard measure of training load and measures that show potential as possible measures of training load warrant further investigation. For example, in a recent novel approach, Kaikkonen *et al.* investigated recovery measurements as possible measures of training load and found that changes in heart rate variability within the first minutes after exercise were sensitive to the intensity of the preceding exercise bout and, in some instances, to changes in exercise duration [15,16].

It is intuitive that the return towards resting homeostasis at the cessation of exercise would be related to the homeostatic stress or training load of the preceding exercise bout, although recovery measurements have not necessarily been applied in this context. For example, excess post-exercise oxygen consumption (EPOC) has been investigated primarily in the context of weight loss [17–24]. However, this measurement is sensitive to the intensity and duration of the preceding exercise bout [20,25] and may show potential as a measure of training load. In a different example, heart rate recovery (HRR) has been investigated as a predictor of mortality [26,27] or as possible tool to monitor training status [28] but also appears to be somewhat sensitive to the intensity of the preceding exercise bout [29] and has the potential to be related to training load.

Both EPOC and HRR represent objective, non-invasive measures of the return towards resting homeostasis at the end of an exercise bout. However, EPOC and HRR may have different relative potential as measures of training load due to differences in the physiological determinants and day-to-day variation of metabolic- vs. autonomic- type recovery measurements. Another factor to consider is that there are different forms of EPOC and HRR and these different forms may also differ in relative potential as measures of training load. For example, EPOC can be reported as a magnitude in the form of the area under the oxygen consumption (VO_2) recovery curve [30–32], or as a time-based measurement such as the half life or time constant of the VO_2 recovery curve [33,34] or the total time taken to return to baseline metabolic rate [31,32]. These different forms of EPOC can be dissociated in that trained and untrained individuals show no difference in the magnitude of EPOC after exercise at the same %VO_2max but show significant differences in EPOC duration or VO_2 recovery rate [31,33,35].

In the case of HRR, the return towards resting heart rate has been calculated as the absolute change in heart rate within the first minute post-exercise (HRR_{60s}) [26,36] as well as the time constant of the heart rate recovery curve (HRRτ) [37,38]. At submaximal intensities, HRR_{60s} is determined primarily by parasympathetic reactivation whereas HRRτ is influenced by both parasympathetic reactivation and sympathetic withdrawal [29,37,39,40]. The responses of these two forms of HRR can be differentiated under certain conditions [41,42], supporting the premise that each form represents a somewhat different measure of autonomic regulation.

Although it is common for authors to include different forms of EPOC or different forms of HRR within their findings, it is rare for metabolic and autonomic recovery responses to be reported and compared within the same study. As a result, the relative sensitivity of these measurements to factors such as training status, training load or training fatigue is not clear. It was anticipated that comparing the relative sensitivity of these measurements would help to identify which measurements show potential as markers of, for example, training load or "readiness" to train. Furthermore, it was anticipated that the relative potential of different recovery measurements as markers of training load may differ according to whether the measures are used to compare responses between individuals or within the same individual. Therefore, Chapters 4 to 7 describe original investigations aimed at addressing the main topic of this thesis: *"Post-exercise oxygen consumption and heart rate recovery as possible measures of the homeostatic stress of an exercise bout"*.

The 4 recovery measurements that served as the main outcomes of each study were the magnitude of EPOC ($EPOC_{MAG}$), the time constant of the VO_2 recovery curve (EPOCτ), HRR

within the first minute post-exercise (HRR_{60s}) and the time constant of the HRR curve (HRRτ) As mentioned previously, HRR_{60s} and HRRτ represent established methods of quantifying heart rate recovery with HRR_{60s} thought to be primarily mediated by parasympathetic reactivation and HRRτ by a combination of parasympathetic reactivation and sympathetic withdrawal [40]. Although EPOC is typically reported as a magnitude [15,20,25,30], it has also been reported as a time-based variable [31,33,34] and both magnitude-based ($EPOC_{MAG}$) and time-based EPOC responses were included. EPOCτ was chosen as the time-based form of EPOC so as to be more comparable with HRRτ.

The specific questions posed in the thesis were as follows.

Which outcome measurement is;

- most closely related to inter-individual variation in the homeostatic stress of an exercise bout?
- most sensitive to intra-individual variation in the homeostatic stress of an exercise bout?
- most sensitive to intra-individual variation in "readiness to train"?

A summary of the thesis topic, outcome measurements and specific research questions along with an overview of the chapters of the thesis appear in Figures 1.1 and 1.2, respectively.

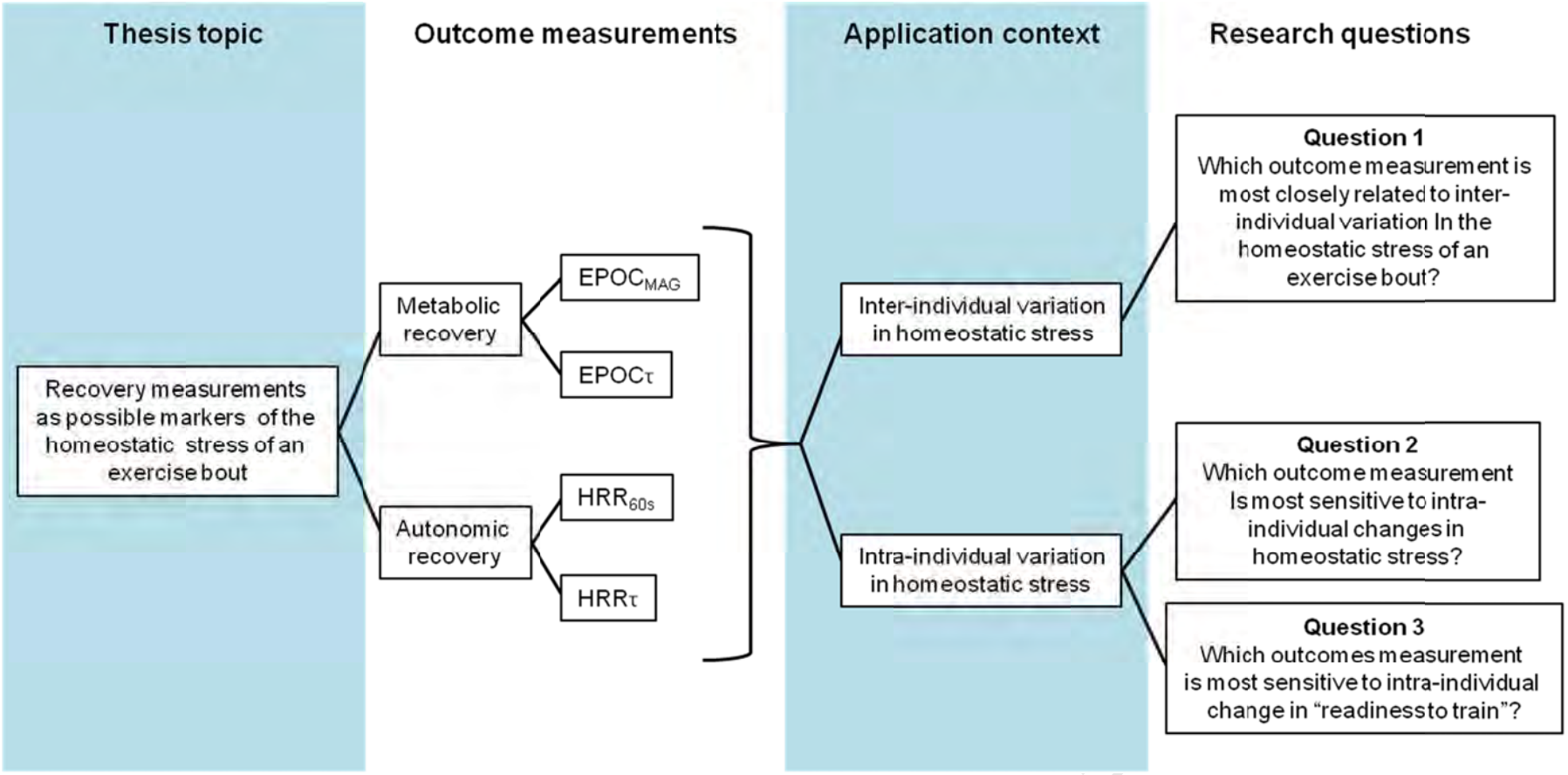


Fig 1.1 Overview of thesis topic

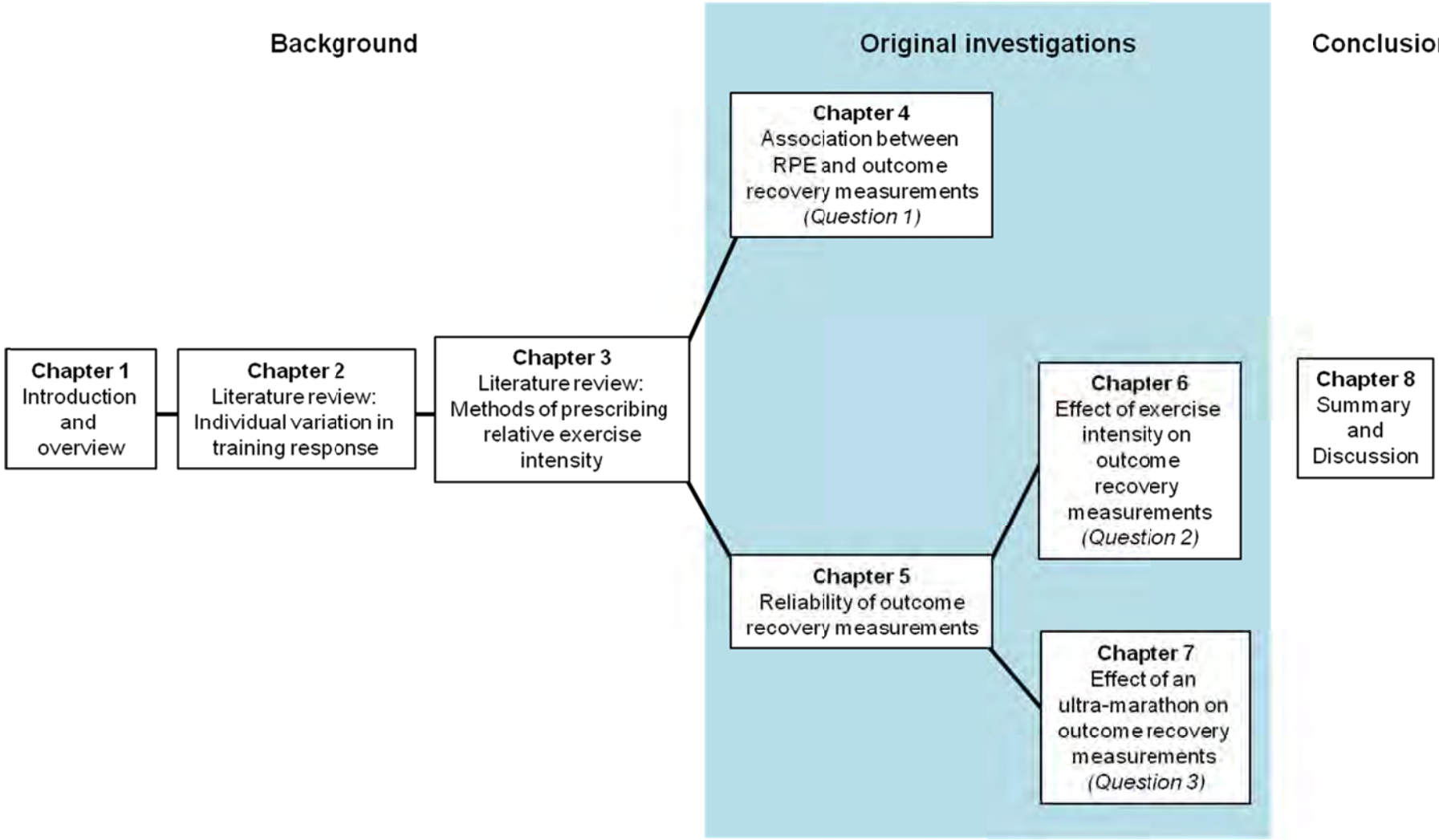
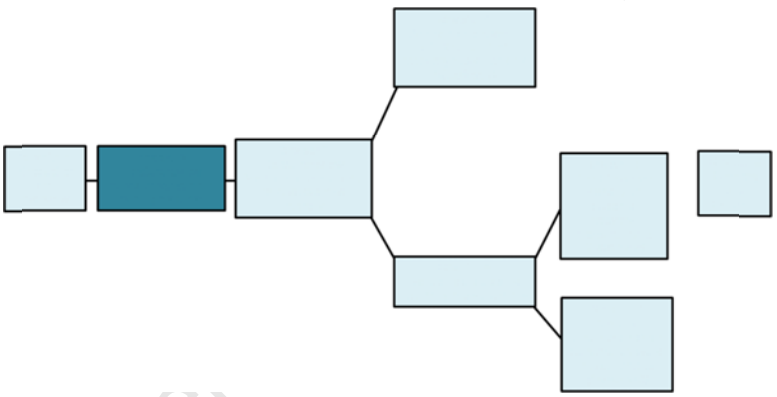


Fig 1.2 Overview of thesis chapters

CHAPTER 2

FACTORS THAT MAY EXPLAIN INDIVIDUAL VARIATION IN RESPONSE TO STANDARDIZED TRAINING



2.1 INTRODUCTION

2.1.1 Individual variation in response to standardized training

In the exercise science literature, it remains conventional to report only the group mean and standard deviation for exercise- or training-responses even though ~32% of normally-distributed measurements fall more than 1 standard deviation from the mean [43]. This variation in response around the mean alludes to the individual variation in exercise- and training- responses that are ubiquitously observed but only specifically described in a comparatively small number of studies. Parameters for which individual variation in training response have been highlighted include maximal oxygen uptake (VO_2max) [1–3,5–8,10,44], resting heart rate (HR) [10], exercise HR [1,10], exercise systolic blood pressure [1], the aerobic threshold [45], the anaerobic threshold [10,45], resting muscle glycogen [7], muscle enzyme activity [7,44,46] and total work done in a performance trial of fixed duration [7].

2.1.2 Non-response or adverse response to training

Although individual variation in training response provides helpful insight into the mechanisms of training adaptation, it also raises the practical concern of poor response to exercise interventions. To be specific, at each end of a range of individual training responses, there are individuals who show exceptionally large responses (high responders) and exceptionally small responses (non-responders) to a training program. The non-responders are of particular concern in the context of exercise for health promotion. By definition, these individuals show a worsened or unchanged response after training [5], or more accurately, exhibit a training response that does not exceed the day-to-day variation of that particular measurement [10].

The presence of at least some non-responders for VO_2max training response is a common occurrence following endurance training in previously untrained individuals [1,2,5,7,8,10]. However, as authors have included more measurements of training response, rather than measuring VO_2max alone, it has become apparent that non-responders for VO_2max are not necessarily non-responders in other markers of training adaptation. For example, Scharhag-Rosenberger *et al.* [10] found that after a year of endurance training, 4 of 18 participants had training-induced changes in exercise HR and the individual anaerobic threshold but no change in VO_2max whereas 3 different participants improved their VO_2max and individual anaerobic threshold but showed no change in exercise HR. The authors described this phenomenon as “individual patterns of non-response”. Individual patterns of non-response have also been demonstrated at a molecular level by the work of Vollaard *et al.* [7], who showed that some individuals with no change in aerobic enzyme activity post-training were among the highest responders for VO_2max.

Although individuals who show no improvement in certain parameters after training may be a concern for coaches or health professionals, an even greater concern are individuals who show a categorically adverse response in certain exercise-related parameters. The prevalence of adverse responses was recently investigated by Bouchard *et al.* [47] in a combined sample of 1,687 men and women from 6 different training studies. The authors defined an adverse response as a change of twice, or more the within-subject typical error of a measurement in an adverse direction. Examples of adverse changes for typical measurements include; ≥ 10 mm Hg increase in systolic blood pressure, ≥ 0.42 $mmol \cdot l^{-1}$ increase in triglyceride levels, 24 $pmol \cdot l^{-1}$ increase in fasting insulin levels and ≥ 0.12 $mmol \cdot l^{-1}$ decrease in high density lipoprotein cholesterol levels. Based on these criteria, 12% of the participants were adverse responders for systolic blood pressure, 10% for triglyceride levels, 8% for fasting insulin levels and 13% for high density lipoprotein cholesterol levels [47]. In total, 31% of the sample showed 1 adverse metabolic response, 6% showed 2 adverse responses and 0.8% showed 3 or 4 adverse responses. The 6 studies from which the total sample was composed were heterogeneous with respect to participant age, health status and training methods. Nevertheless, the prevalence of adverse responses appeared to be consistent across the different participant groups [47]. Adverse metabolic responses were not related to a smaller increase in VO_2max. Furthermore, the incidence of adverse metabolic responses was not different in subgroups who had performed different training volumes [47].

2.1.3 Purpose of review

It is clear that individual variation in response to training is a highly prevalent phenomenon and may have important implications when prescribing training for both health- and performance-related purposes. A better understanding of this phenomenon may provide helpful insight into the mechanisms underpinning training adaptation and could identify possible strategies to predict relatively high- or low-training response at an early stage. With this in mind, the purpose of this review was to discuss factors that may contribute to individual variation in response to endurance-type training.

2.2 FACTORS THAT MAY EXPLAIN INDIVIDUAL VARIATION IN RESPONSE TO TRAINING

2.2.1 Genotype, heredity and baseline phenotype

The link between genetic variation and heterogeneous training responses was first investigated in the mid 1980's by comparing the within-pair and between-pair training responses of monozygotic twins [44,45,48,49]. There was less variation within-pairs of twins than between-pairs of twins for several response

phenotypes, suggesting that certain training responses were indeed genotype-dependent [44,45,48]. However, these findings were somewhat limited by small participant numbers.

In response to the need for a much larger study, a 5 laboratory consortium recruited more than 90 Caucasian families and more than 40 African American families (both parents and 3 or more adult offspring) and studied their responses to a 20 week, standardized endurance training program - an undertaking known as the HERITAGE Family study [50]. The study examined the cardiovascular and metabolic response to exercise training, with particular emphasis on the role of major gene effects [51,52], specific polymorphisms [53,54], heritability [9,55–58] and familial aggregation [9,59]. For clarity, heritability is reported as a maximal estimate based on the correlations between family members who share genetic variance (parent-offspring and siblings) and family members who do not (spouses) whereas familial aggregation is determined by comparing variance within- and between- family units.

2.2.1.1 Genotype and training response

Gene variants associated with certain training response phenotypes are gradually being identified. For example, ~50% of the variance in VO_2max response to training can be explained by variance in 21 single-nucleotide polymorphisms [53] and ~36% of the training response in exercise heart rate at 50 W can be explained by 9 single-nucleotide polymorphisms [54]. Other training response phenotypes that may be associated with specific gene variants include peak power output, the anaerobic threshold and running economy (see review by Rankinen *et al.)* [60]. Preliminary evidence suggests that specific gene variants may facilitate high- or low- response to training through the expression profile of certain RNA's at baseline [61] and the expression profile of certain micro RNA's in response to training [62].

Nevertheless, several findings have yet to be replicated elsewhere and many aspects of the relationship between genotype and training response remain unclear [63]. For example, it is not known whether the genetic variance associated with variance in training response remains consistent across different exercise modes, training program structures and training program durations. Furthermore, it is unclear whether genomic predictors of training response are the same in healthy and at-risk or diseased populations. In light of this uncertainty, the clinical value of genomic predictors of exercise response for individualized exercise prescription remains under debate [64–67].

2.2.1.2 Hereditary factors and baseline phenotype

Due to the challenges of investigating heritability and training response (e.g. need for large sample sizes, recruitment of family units), the findings of the HERITAGE study feature prominently in the following discussion. However, heritability estimates may vary with race [55], the relative intensity of the exercise response measurement [56] and the duration of the training program [44]. Furthermore, the mode

of training and the training load would also be expected to affect heritability estimates [63]. It follows that genetic- and hereditary- estimates included in this section and section 2.1.3. serve as examples rather than established effects.

Many exercise-related parameters have a considerable hereditary or familial influence in the untrained or "baseline" state (Fig 2.1). For example, hereditary factors explain up to 50% of the variance in VO_2max [68], up to 58% of the variance in the VO_2 at the ventilatory threshold [55] and up to 59% of the variance in HR when exercising at 50 W in the untrained state [69,*]. Furthermore, there is significant familial aggregation of the maximal enzyme activities for the phosphagen, glycolytic and oxidative pathways in the untrained state [59]. Hereditary influences and specific gene variants have also been linked to a number of other exercise-related phenotypes including fat-free mass, forced expiratory volume and muscle strength (see review by Puthucheary *et al.*) [70].

In theory, differences in the pre-training or "baseline" phenotype should be compensated for by the use of relative rather than absolute exercise intensity prescription. However, in some cases, the pre-training value of a measurement may influence the magnitude of the training response. For example, in the HERITAGE study, baseline HR at 50 W accounted for ~40% of the variation in the training-induced change in HR at 50 W [1]. In a similar way, baseline systolic blood pressure at 50 W accounted for 32% of the variation in the training-induced change in systolic blood pressure at 50 W [1]. In contrast, baseline measurements explained only ~ 1% of the training-induced change in VO_2max and high density lipoprotein cholesterol levels [1]. When modelling the heritability of training responses, it is conventional to correct for differences in baseline measurements as well as factors such as age and gender. However, in a practical context, baseline factors such as these would contribute to individual variation in exercise response.

2.2.1.3 Hereditary factors and training response

Heritable factors have been linked to variation in training response for a number of phenotypes although the variance explained is typically smaller when corrected for factors such as baseline values, age and gender. There are also examples of parameters for which a hereditary influence is present at baseline but not in the subsequent training response (Fig 2.1).

In the HERITAGE study, heritable factors explained up to 47% of the VO_2max training response [9], 22% of the ventilatory threshold training response [55], 34% of the HR at 50 W training response [69] and 22% of

[*] An *et al.* (2003) cite the afore-mentioned heritability estimate for exercise heart rate response in the untrained state as the work of Leon *et al.* currently under review. However, there appears to be no record of this work having been published in the interim.

the systolic blood pressure and 50 W training response [69] when corrected for baseline influences. However, no significant hereditary influences were detected for the training-induced change in diastolic blood pressure at 50 W [69], fibre-type proportion [59] and muscle capillarization [59]. This implies that the latter training responses were determined primarily by environmental factors.

Some of the afore-mentioned training response heritability estimates from the HERITAGE study are in accordance with earlier observations in monozygotic twins by Prud'homme *et al.* [45]. These authors reported within-pair similarities of 55% and 18% for VO_2max and ventilatory threshold training responses following a 20 week standardized training program. In addition, the authors showed a reduced hereditary influence on training responses at higher exercise intensities (ventilatory threshold response vs. anaerobic threshold response). A similar effect was observed when comparing the hereditary influence on systolic blood pressure response at 50 W and at 60% VO_2max in the HERITAGE study (Fig 2.1).

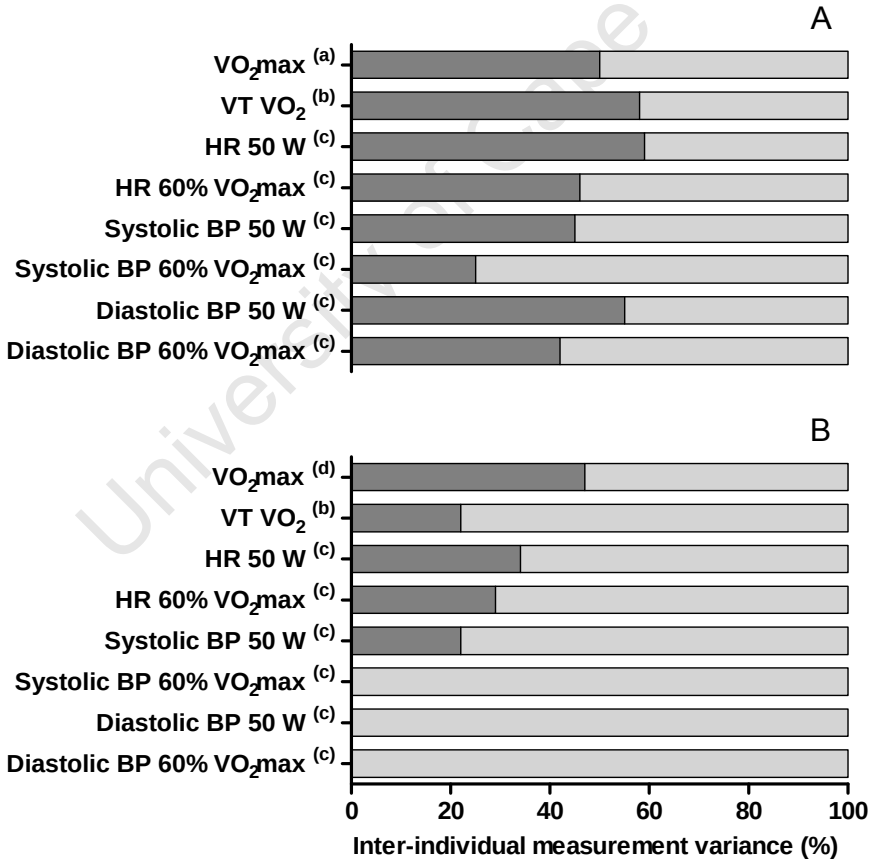


Fig 2.1 Maximum heritability (dark grey bars) and minimum environmental influence on inter-individual variation in measurement variance from the HERITAGE Family study. A = Baseline measurements B = Baseline-corrected training response.

VT = ventilatory threshold, VO_2 = oxygen consumption, HR = heart rate, BP = blood pressure.

[a] = Bouchard *et al.* [68] , [b] = Gaskill *et al.* [55] , [c] = An *et al.* [69], [d] = Bouchard *et al.* [9].

2.2.1.4 Predicting training response

There are a small number of examples in which authors have used baseline measurements as well as factors such as age and gender in multiple regression models to predict training response [1,6,10,71]. Further details of these studies is shown in Table 2.1.

Table 2.1 Multiple regression models to predict training response from baseline phenotypes in untrained individuals

Predicted variable	Participants	Training program	Predictive variable/model	Variance explained (R^2)	Reference
ΔVO_2max	n =17 (M & F)	3 sessions·wk^{-1} for 50 weeks	Age, gender, compliance and baseline VO_2max	16 %	Scharhag-Rosenberger *et al.* [10]
	n = 720 (M & F)	3 sessions·wk^{-1} for 20 weeks	Age, gender, race and baseline VO_2max	11 %	Bouchard & Rankinen [1]
	n= 39 (M)	6 sessions·wk^{-1} for 8 weeks	Nocturnal heart rate variability	27 %	Hautala 2003 [6]
	n = 16 (F)	3 sessions·wk^{-1} for 12 weeks	Resting heart rate variability	34 %	Boutcher *et al.* [71]
Δ exercise heart rate*	n = 18 (M & F)	3 sessions·wk^{-1} for 50 weeks	Age, gender, compliance and baseline exercise heart rate	21 %	Scharhag-Rosenberger *et al.* [10]
Δ heart rate at 50 W	n = 720 (M & F)	3 sessions·wk^{-1} for 20 weeks	Baseline heart rate 50 W, gender, race and age	47 %	Bouchard & Rankinen [1]
Δ HDL-C	n = 720 (M & F)	3 sessions·wk^{-1} for 20 weeks	Baseline HDL-C, gender and race	2 %	Bouchard & Rankinen [1]
Δ systolic blood pressure at 50 W	n = 720 (M & F)	3 sessions·wk^{-1} for 20 weeks	Systolic blood pressure at 50 W and gender	33 %	Bouchard & Rankinen [1]
Δ individual anaerobic threshold (W)	n = 15 (M & F)	3 sessions·wk^{-1} for 50 weeks	Age, gender, compliance and baseline individual anaerobic threshold	11 %	Scharhag-Rosenberger *et al.* [10]

Δ = pre-post training change. Hautala *et al.* used relative change in the predictive variable whereas the other authors appear to have used absolute change. HDL-C = high density lipoprotein cholesterol, M = males, F = females.

Although it is difficult to form conclusions based on such a small number of studies, it would appear that factors such as age, gender and the baseline value of a measurement explain only a small amount of the subsequent training response for VO_2max and the individual anaerobic threshold (11-16%) and a somewhat larger amount of variation in exercise HR and blood pressure training responses (21-47%).

In a different approach, growing evidence suggests that markers of autonomic activity, measured at baseline, could predict subsequent training responses [6,71–73]. For example, Hautala *et al.* [6] and Boutcher *et al.* [71] found that resting heart rate variability measured in the untrained state was able to explain ~ 30% of the variation in VO_2max response to a subsequent training program. Furthermore, both Hedelin *et al.* [72] and Buchheit *et al.* [73] reported that resting heart rate variability was associated with subsequent improvements in performance among trained individuals.

With further investigation, it may be possible to establish which variable or combination of variables can be used to predict relatively high- or low- training responses for a particular training response parameter. However, unlike existing studies, these investigations should also consider whether predictive models or variables are practically meaningful and not just statistically significant.

2.2.2 Homeostatic stress of each training session

It is inevitable that individuals who complete different training regimens will show individual variation in the adaptive responses incurred as a result of the training. For this reason, many authors reporting individual variation in response to training have taken care to ensure that the training received can be considered approximately equal across all individuals in the participant group. To be specific, training is prescribed at a relative, rather than absolute intensity, with a fixed session duration and frequency and is often supervised [2,3,7,8,10,74]. Although training prescribed according to relative exercise intensity is designed to produce a comparable homeostatic stress among individuals, it is, nevertheless, challenging to standardize all the components of an exercise stimulus simultaneously. It follows that inter-individual differences in the exercise stress of the training may occur if the method of prescribing relative exercise intensity was not sufficiently effective [3].

Studies that have reported individual variation in response to training have generally prescribed relative exercise intensity as a %VO_2max or a %HRmax [2,5,7–9]. However these methods of prescribing exercise intensity have been criticized on the basis of large inter-individual variation in blood lactate responses at a fixed %VO_2max or %HRmax of moderate-to-high intensity [11–13]. It has been argued that inter-individual variation in blood lactate response is indicative of inter-individual variation in the “metabolic stress” of the exercise bout and that, for this reason, the use of %VO_2max or %HRmax does not standardize the relative intensity of the exercise bout effectively.

When extrapolated to the context of a training program, variation in the homeostatic stress of each training session would result in variation in the exercise stimulus incurred and hence variation in the nature of the transcriptional and translational response after each training session. Adaptation to exercise training is the accumulated effect of the transcriptional- and translational "micro-adaptations" that occur after each exercise bout [75], therefore variation in the homeostatic stress of each training session may account for some of the variation in training responses. Gaskill *et al.* provided some evidence of this effect when they retrospectively analyzed the effect of training intensity relative to the ventilatory threshold on pre vs. post training changes in the ventilatory threshold and in VO_2max among HERITAGE study participants [76]. The HERITAGE study training sessions were initially prescribed at 55% VO_2max and progressed to 75% VO_2max over the course of the 20 week training intervention. At the initial training intensity of 55% VO_2max, baseline ventilatory threshold values ranged from 34 to 83 %VO_2max, indicating large individual variation in the training intensity relative to the ventilatory threshold [76]. Variation in training intensity relative to the ventilatory threshold was subsequently shown to account for 26% of the improvement in the VO_2 at the ventilatory threshold with higher relative intensities associated with greater improvements in ventilatory threshold VO_2. Conversely, there was no significant relationship between training intensity relative to the ventilatory threshold and improvements in VO_2max. This implies that individual variation in training intensity relative to threshold values does not necessarily contribute to the individual variation in VO_2max response reported in the HERITAGE study [1] or elsewhere [2,3,5–8,10,44], although the authors described the lack of effect as "surprising" [76].

Authors that have criticized the use of %VO_2max and %HRmax for exercise intensity prescription have argued that training prescribed relative to threshold measurements would be better suited to eliciting a similar relative exercise stress among individuals [7,11–13,77] and the afore-mentioned findings of Gaskill *et al.* would appear to support this premise. However, we are not aware of any studies that have reported individual variation in training response following training prescribed relative to threshold measurements. Therefore the reduced individual variation in training response following threshold-related training when compared to training at a %VO_2max or %HRmax remains an assumption. Nevertheless, it is reasonable to conclude that individual variation in the homeostatic stress of each training session is likely to make a significant contribution to individual variation in training responses after a standardized training program. It follows that the method of relative exercise intensity prescription may require careful consideration, taking into account the main outcomes targeted for improvement after the training intervention. For example, the findings of Gaskill *et al.* [76] suggest that exercise prescribed relative to a threshold measurement may reduce inter-individual variation in the ventilatory threshold training response but not necessarily in VO_2max training response.

2.2.3 Recovery and "readiness" to train

Another factor that may contribute to individual variation in response to training is individual variation in recovery from previous training sessions and, by association, "readiness" to adapt to subsequent training sessions. The importance of recovery between training sessions and possible strategies to enhance the recovery process have been discussed in detail elsewhere [78–80].

The potential influence of quality of recovery on training response would be expected to be more prominent at higher training loads or frequencies and with increased duration of the training program. This effect is somewhat illustrated by the findings of Lamberts *et al.*, who monitored well-trained cyclists over the course of a 4 week high intensity interval training program [81]. Heart rate recovery was measured bi-weekly and cyclists were retrospectively grouped according to those who had shown improved heart rate recovery over the course of the training program and those who had shown a decrease in heart rate recovery. However, these differences emerged primarily in the final week of the program as both groups had improved heart rate recovery after 3 weeks of training. The authors found that the group with an overall decrease in heart rate recovery had a blunted improvement in post vs. pre- training 40 km time trial average power output compared to the group with a continuous increase in heart rate recovery and speculated that accumulated fatigue and decreased tolerance to the training may explain these observations [81]. It follows that accumulated fatigue and decreased tolerance to the training could be said to have contributed to the variation in performance improvements after 4 weeks of training. Furthermore, this effect may have been smaller had performance been re-measured after only 3 weeks of training, given that both groups showed favourable changes in heart rate recovery at this time point.

Variation in the ability to recover between training sessions may to a certain extent arise from lifestyle factors such as sleeping patterns and mental stress [82]. These factors may be difficult to quantify and/or to interpret practically, however it is reasonable to suggest that they influence adaptation to training. For example, sleep debt may decrease glucose tolerance and increase sympathetic activity at rest [83], sleep deprivation may reduce anabolic activity through a decrease in circulating testosterone [84] and mental stress may promote catabolic activity through the associated increase in circulating cortisol levels [85]. Based on the latter observations, Dattilo *et al.* [86] hypothesized that the accumulation of sleep debt may impair muscle recovery following exercise through promoting a catabolic environment within the muscle. Furthermore, Samuels presented case studies relating sleep disturbances to poor training tolerance and fatigue in athletes and commented that "ongoing sleep debt is likely a critical factor affecting post-exercise recovery, performance and susceptibility to the overtraining syndrome" [87]. Although these findings provide some support for the influence of quality of recovery on adaptation to

training, other findings have not supported this relationship [88]. It follows that larger sample sizes and appropriately designed studies are required to more conclusively establish the link between recovery and training response.

Although there is no gold standard measure for assessing an individual's overall adaptive status, questionnaires such as the Daily Analysis of Life Demands for Athletes (DALDA) [89] and measures of autonomic nervous system activity such as heart rate recovery and heart rate variability incorporate both training and lifestyle stress and have potential for detecting fatigue or overreaching [28,90–92]. It is not clear whether adjusting individual exercise prescription based on "readiness to train" would decrease overall individual variation in response to training *per se*. However, it could be speculated that this approach might reduce the likelihood of low training responses.

2.2.4 Nutritional status

It is increasingly evident that endogenous and exogenous substrate availability can modulate the transcriptional and translational response to an exercise bout [93,94], suggesting that variation in the typical timing and composition of dietary intake may also contribute to individual variation in training responses. For example, ingestion of carbohydrates or a carbohydrate-protein mixture attenuates the mRNA expression of genes involved in lipid metabolism [95] and protein degradation [96], respectively. In addition, several studies have demonstrated significant differences in training adaptation following short-term dietary interventions (e.g. training with low muscle glycogen levels) (see review by Hawley *et al.* [94]). Nevertheless, the mechanisms linking acute, substrate-related differences in gene regulation and accumulated training adaptations remain under investigation and it is not clear what magnitude of variation in typical nutritional status would be required to make a meaningful contribution to individual variation in training responses.

2.3 SUMMARY AND CONCLUSION

Although it is conventional to focus on the group mean response following a particular training intervention, individual responses typically show considerable variation including particularly "high responders" and particularly "low responders" or "non-responders" for a certain training response parameter [1,4,7]. A high responder for one form of training response (e.g. change in submaximal heart rate) may not necessarily be a high responder for a different form of training response (e.g. change in VO_2max) [7,10], implying that the same individual could potentially be described as both a "responder" or

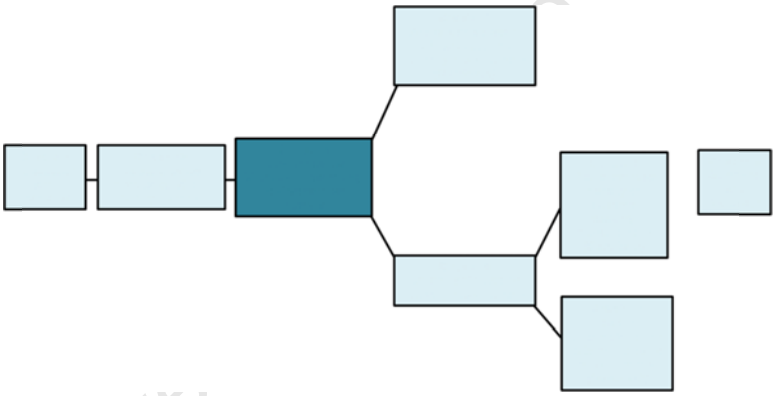
a “non-responder”, depending on the outcome variable of interest. It follows that factors that explain or predict individual variation in training response may vary with different training response phenotypes.

Identifying which genetic or environmental factors contribute to variation in a particular training response could have application to predict in advance which individuals might have particularly high or low training responses. Furthermore, some environmental contributors to individual variation in training response could potentially be adjusted to decrease individual variation in response to training or reduce the likelihood of low training response. For example, one environmental factor likely to contribute to individual variation in response to standardized training programs is individual variation in the homeostatic stress of each training session. It is difficult to prescribe an equivalent overall homeostatic stress among different individuals and there is, at present, no gold standard for this type of measurement. In theory, exercise prescribed relative to threshold measurements may result in less individual variation in the homeostatic stress of the training session than when exercise is prescribed according to %VO_2max or %HRmax. However, when the practical application of threshold measures vs. %VO_2max or %HRmax to prescribe relative intensity is taken into account, it is apparent that there are advantages and disadvantages to both of these approaches (*see Chapter 3*)[97]. Another strategy that may promote less individual variation in training response is to monitor fatigue or “readiness to train” on an individual basis and then adjust training sessions accordingly, however this effect has yet to be demonstrated and individual monitoring would not be practical for large participant groups.

In conclusion, this review discussed a number of factors that may contribute to the individual variation in adaptive responses observed after standardized training programs. In each case, the discussion included a rationale as to why a particular factor would be expected to contribute to individual variation in response to training. Nevertheless, appropriate research studies supporting the role of factors such as the homeostatic stress of each training session, sleep patterns and nutritional status to individual variation in training response, are currently lacking. Future studies addressing such topics may aid in the early prediction of high- or low- training responses and provide further insight into mechanisms of training adaptation.

CHAPTER 3

METHODS OF PRESCRIBING RELATIVE EXERCISE INTENSITY: PHYSIOLOGICAL AND PRACTICAL CONSIDERATIONS



3.1 INTRODUCTION

It is widely understood that exercise standardized according to an absolute external workload may produce large inter-individual differences in internal cardiovascular and metabolic stress. For this reason, it is more common to "individualize" exercise prescription according to relative intensity [98–100]. This approach is intended to account for differences in physiological and functional capacity, producing an approximately equivalent exercise stress among individuals despite differences in phenotype. For research purposes, controlling the relative intensity of an exercise bout allows for the interpretation of other exercise-related responses, while for health and performance purposes prescribing training according to relative intensity allows for more predictable adaptive responses.

The traditional approach when prescribing relative intensity has been to use a % of maximal oxygen consumption (VO_2max) or maximal heart rate (HRmax) and many recent publications continue to favour these methods [35,46,101–106]. However, a number of authors have argued against the use of %VO_2max or %HRmax for exercise intensity prescription, recommending other methods as more meaningful for equating exercise stress [7,11–13,77,107–109]. For example, Swain *et al.* have reasoned that use of %VO_2max does not account for differences in resting metabolic rate and that it is preferable to prescribe exercise relative to an individual's oxygen consumption reserve (VO_2R) (VO_2max minus resting oxygen consumption(VO_2)) [107–109]. Use of %VO_2R has the advantage of placing individuals at an equivalent intensity above resting levels. Furthermore, several studies have found that %VO_2R and % heart rate reserve (HRres) (HRmax minus resting heart rate (HR)) can be considered equivalent methods of exercise intensity prescription whereas %VO_2max and %HRres may differ noticeably at lower exercise intensities [108–110]. Based on the convenience of the %VO_2R-%HRres relationship for HR-based monitoring of training, these methods of exercise intensity prescription were recommended by the American College of Sports Medicine (ACSM) in 1998 [111]. However, the %VO_2R-%HRres relationship has since been questioned [112–115] and the 2011 ACSM guidelines included %VO_2max and %HRmax along with %VO_2R and %HRres as recommended methods of exercise intensity prescription [100].

A separate criticism of the use of %VO_2max or %HRmax for exercise intensity prescription is that these methods fail to account for differences in metabolic stress [7,11–13,77]. Authors highlighting this discrepancy have advocated the use of metabolic thresholds such as the aerobic threshold (AerT) and anaerobic threshold (AnT) as preferable "anchors" for relative exercise intensity prescription and there are indeed numerous examples of this approach [116–122]. However, there has been little consistency in methods of threshold calculation and the theoretical basis of the thresholds remains controversial [123–125].

In summary, there appear to be some discrepancies in the methods of relative exercise intensity prescription recommended by different authors. Furthermore, there are discrepancies between methods of exercise intensity prescription that have been recommended and those methods that continue to be used by researchers. Although different methods of relative exercise intensity prescription have been reviewed on previous occasions, Hills *et al.* [98] focused on the development of equations for prescribing exercise intensity and Carvalho *et al.* [99] focused on the implications of prescribing exercise intensity in clinical populations. In contrast, the aim of the current review was to compare the physiological and practical implications of prescribing exercise relative to VO_2max, HRmax, VO_2R or HRres, the AerT or the AnT in healthy, active or athletic populations.

3.2 PHYSIOLOGICAL RESPONSES TO EXERCISE STANDARDIZED BY RELATIVE INTENSITY

3.2.1 Acute exercise responses

3.2.1.1 Systemic responses

Most authors citing poorly standardized metabolic stress at a fixed %VO_2max or %HRmax have based this view on the individual variation in blood lactate accumulation that may occur when using these methods [7,11–13,77]. For example, Dwyer and Bybee [11] observed that for any intensity between 58 and 75% VO_2max, some of their participants were below, and others above the AnT. Conversely, Meyer *et al.* [12], showed that the workload associated with 75% VO_2max corresponded to 86-118% of the individual anaerobic threshold and blood lactate concentrations of 1.4-4.6 mmol·l^{-1} in different individuals following an incremental test. Scharhag-Rosenberger *et al.* [13] reported similar findings with 4- and 14- of 18 participants exceeding the individual anaerobic threshold during constant intensity exercise at 60% and 75% of VO_2max, respectively. Furthermore, in comparing the blood lactate ranges at 60% of VO_2max (0.7-5.6 mmol·l^{-1}) and 75% VO_2max (2.2-8.0 mmol·l^{-1}), Scharhag-Rosenberger *et al.* demonstrated increased variation in blood lactate response with increasing exercise intensity by %VO_2max as well as increased variation in blood lactate response in a heterogeneous group when compared to the more homogenous group of Meyer *et al.* [12] at the same intensity. Although blood lactate variation at a %VO_2max would be expected to be larger in heterogeneous groups, some variation may occur even when individuals have a similar VO_2max [126].

Even though exercise prescribed as a %VO_2R or %HRres may place individuals at a similar intensity above resting metabolism, these methods have also been linked to individual variation relative to

threshold concepts [13,127–130]. For example, Scharhag-Rosenberger *et al.* reported that some participants were above and others below the AnT at an intensity corresponding to 71±1% VO_2R [13] and Acevedo *et al.* reported that the AerT occurred at 70±10% HRres in men of high cardiorespiratory fitness, implying that a fixed %HRres could be above the AerT for some individuals and below the AerT for others [130].

Notably, it is individuals exercising in different exercise intensity domains at the same %VO_2max, %HRmax, %VO_2R or %HRres that may be cause for concern rather than heterogeneous blood lactate concentrations as such- it has been documented that the AnT can be associated with blood lactate concentrations of 2-9 mmol·l^{-1} in different individuals [131–133]. As is well known, different exercise intensity domains are associated not only with a shift in blood lactate responses but also with changes in ventilation [134], oxygen uptake kinetics [135] and catecholamine responses [136,137]. For example, constant-intensity exercise within the "severe" exercise intensity domain (> AnT) is characterized by a continuous increase in ventilation and VO_2, progressive acidosis and metabolite accumulation whereas constant intensity exercise equal to or below the AnT is associated with a physiological steady state [138–141]. The metabolic characteristics of the AnT are analogous to those of critical power in that both measurements are intended to represent the highest workload at which it is possible to achieve a steady state [138,142]. It follows that many of the discussion points regarding exercise prescription relative to the AnT also apply when prescribing exercise intensity relative to critical power. On a practical level, however, the AnT has been associated with a significantly lower workload and increased time to exhaustion when compared to critical power [132,138,143].

3.2.1.2 Time to exhaustion during constant intensity exercise

If exercise intensity prescribed as a %VO_2max, %HRmax, %VO_2R or %HRres results in different metabolic and respiratory profiles among individuals, these differences would be expected to contribute to individual variation in the time to exhaustion at a constant exercise intensity. For example, those individuals exercising above the AnT might be expected to terminate exercise earlier than those exercising below the AnT at the same %VO_2max due to an increased depletion of anaerobic energy reserves and an increased accumulation of metabolites[144]. However, Scharhag-Rosenberger *et al.* found that exercise above or below the AnT did not appear to explain premature exercise cessation when individuals of varied aerobic capacity attempted 60 min of exercise at 75% VO_2max [13]. This finding highlights both the complex nature of fatigue [145] and the discrepancies that can exist between theoretical expectations and individual responses in practice. Nevertheless, at the cross-sectional level, lactate threshold concepts have been related to metabolic activity in the muscle through significant correlations with muscle capillarization (r = 0.59-0.77)[146,147], percentage of slow twitch fibres (r = 0.74-0.78)[147,148], oxidative capacity (r = 0.94)[148] and muscle enzyme activity (r = 0.54-0.68) [149,150] and it is not

surprising that blood lactate markers have been shown to explain more variation in performance than is explained by VO_2max e.g. (r = 0.83 vs. 0.91)[151], (r = 0.55 vs. r = 0.61 to 0.84)[152] and (r = 0.51 vs. r = 0.76)[153].

3.2.1.3 Molecular responses

While it is clear that the skeletal muscle's transcriptional response to exercise is sensitive to increases in relative exercise intensity [104,154,155], the effect of different methods of relative exercise intensity prescription does not appear to have been directly investigated. The biochemical signals that activate adaptive cellular pathways include an increased ratio of adenosine monophosphate (AMP) to adenosine triphosphate (ATP), increased levels of reactive species of oxygen and nitrogen, depleted levels of muscle glycogen and decreased oxygen tension [75,156,157] and it could be speculated that changes in blood lactate accumulation would be associated with some of these changes. Nevertheless, without evidence it is not clear whether exercise above- and below- a threshold concept would be more significant for training adaptation that any other increase in exercise intensity. Future investigations could address this topic by comparing transcriptional and translation responses of individuals exercising above and below a threshold measurement but at the same %VO_2max.

3.2.2 Training responses

3.2.2.1 Individual variation in training responses

Adaptation to training can be understood as the accumulated effect of micro-adaptations that occur in response to the stimulus of each training session [75]. With this in mind, it has been suggested that differences in acute metabolic stress during exercise prescribed relative to %VO_2max (i.e. individuals exercising above and below threshold levels) may explain the large inter-individual variation in response that has been reported following training programs using this method of exercise prescription [12–14]. Although many studies allude to variation in response by way of the standard deviation around the mean response, only a small number of studies deliberately highlight these individual differences. One prominent study that highlighted individual differences is the HERITAGE Family study where 20 weeks of endurance training standardized by %VO_2max produced a mean increase in VO_2max of 384 ml oxygen with a range of ~0 to 1000 ml oxygen for individual responses [1]. Large ranges in individual VO_2max response have also been reported following other training programs based on %VO_2max [5,7] as well as following training programs prescribed by %HRmax [2,8] and %HRres [45]. Furthermore, a range of individual responses following training based on maximal measurements is not restricted to VO_2max but includes other measurements such as submaximal HR [1,7], the AerT [45], the AnT [45], submaximal blood lactate concentration [7], muscle glycogen [7], muscle enzyme activity [7,46] and performance [7]. While

Bouchard and Rankinen are confident that individual differences in training responses are "biologically meaningful" [1], it is rare for authors to discuss individual variation in response in the context of within-subject variability [10]. It could be argued that only individual differences in response that exceed the biological variation of a measurement represent differences that are truly meaningful.

If different individual responses following training at a %VO_2max, %HRmax or %HRres can be explained by differences in metabolic stress, it follows that training standardized with respect to metabolic stress should produce more homogenous individual responses. However, it is rare to find studies addressing individual variation when exercise has been prescribed relative to a threshold concept. Karavirta *et al.*[4] demonstrated substantial individual variation in VO_2max responses (approximately -10% to +58%) following 21 weeks of endurance training prescribed relative to threshold concepts. However, the authors described the prescribed intensities as simply "below", "between" or "above" the aerobic and anaerobic thresholds and it is unclear whether inadequate standardization of the exercise prescription may have contributed to the heterogeneous responses. In another example, McLellan and Skinner[14] investigated whether inter-individual variability in response would be reduced in a group trained relative to the AerT compared to a group trained relative to VO_2max following 8 weeks of cycling training. Contrary to their expectations, no group differences in individual variation were observed. However, the small participant numbers (n = 6 and 8 per group) and the manipulation of individual training loads to match overall intensity between the groups (%VO_2max group = 50 to 58 %VO_2max and +3.6 to +8.5% AerT) make these results similarly difficult to interpret. Until a training study strictly standardized relative to a threshold measurement clarifies the individual responses, it remains an assumption that the use of threshold-related training will produce less individual variability in training effects than training prescribed by %VO_2max.

3.2.2.2 The contribution of genetics to individual variation in training response

An individual's response to exercise training is determined not only by the physiological stress of the exercise bouts but also by the individual's genotype. This is an important reason why reduced individual variation following threshold-related training would need to be demonstrated, rather than assumed. A detailed discussion of genomic predictors of trainability is beyond the scope of the present review and can be found elsewhere [63]. However, by way of illustration, the HERITAGE Family study reported that the VO_2max training response showed 2.5 times more variance between families than within families following 20 weeks of endurance training [9]. Hereditary factors explained up to 47% of VO_2max "trainability" in Caucasian participants [9] and a subsequent genome-wide analysis identified 21 single-nucleotide polymorphisms that explained 49% of the variance in VO_2max response [53]. In a similar sample, the hereditary contribution to submaximal heart rate response was calculated to be about 30%

[57] and very significant familial aggregation has also been reported for the training response of enzymes in the phosphagen, glycolytic and oxidative pathways [59]. Genetic factors may also affect changes in body temperature, norepinephrine and blood lactate during exercise among untrained individuals [158]. It is not yet known how the amount of variance explained by hereditary factors is affected by differences in exercise intensity or exercise mode [63]. Nevertheless, it is possible that genotype may account for a significant proportion of the variation following training studies based on %VO_2max. The relative contribution of hereditary factors vs. differences in metabolic stress during exercise is some way from being understood and provides a large scope for further study.

3.3 PRACTICAL ADVANTAGES AND DISADVANTAGES OF DIFFERENT ANCHOR MEASUREMENTS

When making the case for one method of relative exercise prescription over another, it is typical for the recommendation to centre on the physiological basis of each method. Often little consideration is given to the day-to-day application of each method. It follows that an essential aspect of comparing and contrasting the afore-mentioned anchor measurements is to consider questions such as the ones addressed below.

3.3.1 Can the measurement be verified?

Although the measurements under discussion are routinely determined, the characteristics of the incremental protocol may differ depending on the requirements of the study and the preferences of the research group. In a similar way, there may also be variation in the method of data processing and, where appropriate, the model of graphical analysis. These differences in the nature of the protocol and method of graphical analysis can have a large effect on the measurement value under some circumstances [159]. It follows that if the measurement is to be used as the basis for prescribing relative intensity or to monitor changes in performance, verifying the measurement value would be an important precaution.

3.3.1.1 VO_2max and HRmax verification

Failure of participants to reach maximal exertion would result in VO_2max or HRmax being underestimated and it is common to apply certain checks to evaluate whether the measurement was truly maximal. For example, criteria such as respiratory exchange ratio (RER) > 1.1 or 1.15, a “plateau” in VO_2 with an increased workload and blood lactate > 8 mmol·l^{-1} are often used to verify a true VO_2max. However, using these criteria not only allows for the significant underestimation of VO_2max [160]

but may also result in some genuinely maximal efforts being discounted [160–162]. In recent years, a solution to the limitations of these secondary criteria has emerged in the form of a square wave "verification" bout, performed to exhaustion shortly after the graded exercise protocol [163]. There is, as yet, no consensus as to the duration of the rest interval nor the use of a submaximal or supramaximal workload and a recent report has suggested that these factors might be adjusted based on the length of the preceding protocol [164]. However, it would appear that a 10 min rest period prior to a supramaximal verification bout is sufficient [165,166]. The duration of the supramaximal verification bout itself is reported to be approximately 2 min with the verification VO_2max expected to differ from the incremental VO_2max by no more than 3% [161,167,168] or 5.5% [166] according to different recommendations.

A HRmax is generally considered accurate if it falls within 10 bpm of the "220 minus age" predicted HRmax. This formula is so widely used that an original reference is very rarely cited but Robergs and Landwehr [169] have attributed the original reference of Fox *et al.* [170]. Robergs and Landwehr calculated that the standard error of the estimate around Fox's original data was certainly greater than 10 bpm, thus proximity to that particular age-predicted HRmax does not constitute a meaningful criterion for having attained HRmax. Although other, more accurate formulas have been developed, the use of any standard formula does not consider the variation in HRmax that can occur across different exercise modes. As an alternative, Midgley *et al.* [165] have incorporated HRmax verification within the VO_2max verification bout and proposed a verification criterion of ≤ 4 beat/min difference between the initial HRmax and the verification HRmax of an individual. Notably, the protocol from which this criterion was developed involved 3 min of submaximal exercise before the supramaximal workload, resulting in a total verification exercise time of ± 4.5 min. It follows that this criterion may not be as successful for shorter verification trials that allow less time for heart rate to reach maximum.

3.3.1.2 VO_2R and HRres verification

As VO_2R is based on both resting VO_2 and VO_2max, verifying both of these measurements should ensure that a true VO_2R value has been obtained. Although many authors take precautions of some form to verify that a true VO_2max has been obtained (see section 3.1.1.), a recent review highlighted poor standardization of resting VO_2 measurements and the error that variation in resting VO_2 can introduce into %VO_2R exercise prescription [113]. A number of studies reporting VO_2R have inferred a standard resting VO_2 of 3.5 ml·kg^{-1}·min^{-1} for all individuals [115,171,172] whereas others have determined resting VO_2 individually but have failed to fulfill "best practice" criteria for resting VO_2 measurements [108–110]. These best practice criteria for resting metabolic measurements are based on a systematic review by Compher *et al.* and include a ≥ 5 h fast and restrictions on physical exertion in the hours prior to the assessment among other evidence-based recommendations [173]. Recent studies reporting VO_2R have

adopted these best practice criteria for determining resting VO_2 but did not verify VO_2max at supra-maximal intensities [112,114]. An approach incorporating both of these elements might reasonably be assumed to produce an accurate VO_2R, just as the corresponding values for resting heart rate and HRmax might reasonably be assumed to produce an accurate HRres.

3.3.1.3 AerT and AnT verification

Although it is possible to verify AerT and AnT, doing so would require a minimum of 2 subsequent laboratory visits and several blood lactate measurements. For AerT, workloads less than or equal to the AerT would be expected to produce blood lactate concentrations not different from baseline whereas workloads greater than AerT would be expected to produce a stable blood lactate concentration that was significantly elevated above baseline [174].

Similarly, the AnT could be verified by demonstrating that workloads less than or equal to the AnT workload produced blood lactate responses that were elevated but stable, perhaps showing a slight decrease towards the end of the exercise bout [138,174], whereas workloads greater than AnT should result in a progressive increase in blood lactate concentration [138,174,175]. In essence, verifying the AnT would involve showing that an AnT workload calculated from an incremental test was, in fact, the maximal lactate steady state (MLSS). Detection of the MLSS is, in turn, influenced by the length of the constant-load exercise and the maximum acceptable increase in blood lactate that is applied [176]. The generally accepted [138,177] standard appears to be no more than 1 mmol·l^{-1} increase in blood lactate after 10 min of an exercise bout at least 30 min in duration [176,178].

Although some studies have reported stable blood lactate concentrations at the workload associated with the individual anaerobic threshold (IAT) [131,179], they did not demonstrate the accumulation of lactate at higher workloads and it is possible that the MLSS was underestimated. In instances where the IAT was indeed verified using the blood lactate response during two to three 30 min exercise bouts, the IAT overestimated the MLSS at a group level [180], and in some individuals [141], respectively. Another study [133] found no significant group mean differences between the workload at the MLSS, at 4 mmol·l^{-1} blood lactate and at the AnT determined according to Cheng's "D_{max}" method [181]. However, the authors concluded that neither of the single effort methods was sufficiently precise to identify the MLSS on an individual level and recommended constant load verification trials if the MLSS was to be considered valid.

3.3.2 Is the measurement reliable?

If a measurement has been verified as accurate on one particular day, would a subsequent verification produce a different result? It is to be expected that each of the anchor measurements will have a

certain amount of day-to-day variation as a result of biological variation, equipment measurement error and reproducibility of the testing protocol. However, the smaller the variation on a day-to-day basis, the more reliable the measurement and the more likely that exercise bouts determined relative to the measurement will have the anticipated physiological effect.

The literature contains a variety of reliability measurements including absolute and relative measures of within-participant variation and measures of between-participant variation by way of test-retest correlations. Absolute within-participant variation may be influenced by the absolute magnitude of the participant's measurements and the between-participant variation is influenced by the degree of heterogeneity within the participant group. Therefore, the most reasonable basis for comparing the reliability of different anchor measurements may be the relative within-participant reliability or "coefficient of variation" (CV). The CV represents the measurement error as a % of the measurement mean and the present discussion includes CV's calculated in the following ways a) CV = ($\sqrt{(\sum d^2/2n)}$) / sample mean where d is the sum of the between-trial differences and n is the number of participants [182] b) CV = (SD of difference/$\sqrt{2}$) / sample mean, referring to the standard deviation of the between-trial differences [183] c) CV = (SD $\sqrt{}$ (1 - r) / sample mean, referring to the average standard deviation of the repeated trials and Pearson's correlation coefficient or intraclass correlation coefficient [184] and finally d) CV = SD of the test-retest differences / sample mean [185].

3.3.2.1 Relative reliability of VO_2max, HRmax and threshold measurements

Variation in the statistical basis of the CV calculation may affect the reported measurement reliability, along with factors such as the training status of the participants [186], exercise mode, equipment used and the time period between repeated trials. Therefore, this review focused on studies reporting the reliability of at least 2 anchor measurements such that their relative reliability could be compared on an equal basis. For example, of 6 studies reporting the reliability of VO_2max, HRmax and 1 or more threshold measurements, 5 studies found HRmax to be the most reliable measurement (HRmax CV = 1.0-3.2%) [185,187–190] and 1 study found it to be approximately equivalent to the most reliable measurement (AnT HR CV = 1.2%, HRmax CV = 1.3%) [191](Table 3.1, page 36). There was a lack of agreement over which was the next most reliable measurement with 3 studies reporting VO_2max as more reliable than AerT [188,190] or AerT and AnT[185], 2 studies reporting threshold measurements as more reliable than VO_2max [189,191] and 1 study reporting very similar CV values for VO_2max and AerT (CV = 3.5% vs. 3.8%)[187].

Those studies that found VO_2max was more reliable than threshold measurements reported VO_2max CV's of 1.9%, 2.0% and 4.0%, which are in keeping with the 2.2-2.7% VO_2max CV's reported

elsewhere [182,192,193]. Studies that found threshold measurements to be more reliable than VO_2max appeared to have somewhat higher VO_2max CV values of 4.7% [191] and 8.5% [189]. The VO_2max CV of 8.5%, reported by Lourenco *et al.* [189], was calculated from 4 maximal efforts ≥ 48 h apart and there was a decrease of 2.9 ml·kg^{-1}·min^{-1} in the group mean VO_2max between the 2nd and 4th trials. This suggests that the abnormally high variation in VO_2max in that study could be attributed to accumulated fatigue and that participants did not complete all 4 trials in an equivalent physiological state. Were the findings of Lourenco *et al.* to be set aside, it can be concluded that VO_2max shows a typical CV of 1.9-4.7% and that VO_2max is more reliable than threshold measurements on most, but not all, occasions.

3.3.2.2 Relative reliability of the AerT and AnT

In comparison to HRmax and VO_2max, the range of CV's reported for threshold measurements is large, spanning 1.5-10.4% for AerT and 1.2-11.9% for AnT (Table 3.1). This may be partly attributed to differences in protocol and study design, including, in some cases, the reliability with which investigators are able to identify threshold measurements by visual inspection [194,195]. It is also apparent that the reliability of threshold measurements varies according to whether the threshold is reported as a workload, a HR, a VO_2 or a blood lactate concentration. Of 6 examples [185,187,188,191,196] of a threshold reported according to the corresponding HR, speed or power output and VO_2, 4 found threshold HR to be the most reliable with CV's of 1.5-3.8% [185,187,188,191]. Furthermore, of 9 examples [182,185,187–191,193] reporting both threshold speed or power output and threshold VO_2, 6 reported speed or power output to be the more reliable threshold measurement with speed or power output CV's of 1.7-5.9% [182,185,187,189,191,193]. When comparing the relative reliability of the AerT or AnT on the basis of the associated workload, Aunola *et al.* [187], Weltman *et al.* [191] and Dickhuth *et al.* [197] all found AnT to be the more reliable threshold measure with AnT vs. AerT CV differences of 3.0 vs. 3.5%, 1.7 vs. 3.0% and 2.6% vs. 5.3-5.6%, respectively.

3.3.2.3 Relative reliability of blood lactate samples

Given that several of the methods of AerT and AnT determination or verification involve measuring blood lactate, it is important to mention the many sources of variation in blood lactate measurements. Blood lactate responses during exercise may be affected by factors such as prior exercise [196], the glycogen status of the participant [198] and ambient temperature [199,200]. Furthermore, the lactate concentration measured may vary depending on the sampling site [201–203], sweat contamination and the accuracy of the lactate analyzer. The portable Accusport® analyzer (Boehringer-Mannheim), for example, has a standard error of measurement of 0.3-0.5 mmol·l^{-1} for duplicate samples during a single trial and a day-to-day standard error of measurement of 0.4 mmol·l^{-1} when measuring the same lactate

solution [204]. "Random" measurement error of this magnitude could have a significant effect on the identification of the MLSS (< 1mmol·l^{-1} change in blood lactate between 10 min and the end of the exercise). As a measure of systematic error, the limits of agreement for measurements made using the Accusport analyzer and a criterion measure of lactate concentration were +1.9 to -2.2 mmol·l^{-1} [204]. The errors associated with measuring blood lactate are discussed in greater detail in the following reviews [205–207].

3.3.2.4 Reliability of VO_2R and HRres

The relative reliability of VO_2R and HRres does not appear to have been directly investigated. Nevertheless, the influence of variation in both resting and maximal measurements on repeated determinations of VO_2R and HRres would be expected to result in higher variation than that of VO_2max or HRmax. In other words, VO_2R reliability would incorporate both VO_2max reliability (CV = 2-5% (Table 3.1)) and resting VO_2 reliability (CV = 10%)[173] whereas HRres would incorporate both HRmax reliability (CV = 1-2% (Table 3.1)) and resting HR reliability (CV = 7-8%)[208].

Another factor to consider is the reliability of the %VO_2R-%HRres relationship given that these methods of exercise intensity prescription could be assumed to be equivalent based on previous findings [108–110]. In a recent review, da Cunha *et al.* raised a number of evidence-based concerns for prescribing exercise intensity based on this relationship, including the influence of the incremental protocol on the %VO_2R-%HRres relationship, the influence of resting VO_2 measurements on the %VO_2R-%HRres relationship, the stability of the %VO_2R-%HRres relationship during prolonged exercise and the consistency of the %VO_2R-%HRres relationship across different exercise modes [113]. The authors clearly demonstrated that the %VO_2R-%HRres is not consistently reliable and researchers should consider verifying the %VO_2R-%HRres relationship within their own context of exercise prescription.

3.3.2.5 Reliably targeting a relative response

In a final word on reliability, it is advantageous to be able to monitor, during a particular exercise bout, whether the measured VO_2 or HR or lactate response is in fact the target response prescribed for that bout. For example, 60% of a 60 ml·kg^{-1}·min^{-1} VO_2max would be a target VO_2 of 36 ml·kg^{-1}·min^{-1}, and exercise at the MLSS workload would be expected to produce a stable blood lactate concentration rather than a blood lactate concentration which increases. With this in mind, both VO_2 and HR can be monitored continuously and non-invasively during single exercise bouts, although only HR measurements would be practical for use in regular training sessions. The effect of small adjustments in workload can be observed within a short period and as a result it is comparatively easy to match the measured exercise intensity to the target exercise intensity. This is not the case when the target

intensity is prescribed relative to a threshold concept. Blood lactate is monitored at discrete time points, rather than continuously, and a longer period is required to observe the blood lactate response at a particular workload. If a threshold measurement has been verified, it might be argued that blood lactate monitoring is not necessary. However, the ability to verify both the anchor measurement and the target exercise intensity are practical points to consider for study design.

3.4 SUMMARY AND CONCLUSION

Based on a theoretical understanding of each anchor measurement, training prescribed relative to threshold measurements would be expected to elicit more comparable metabolic and respiratory responses between individuals than exercise prescribed relative to VO_2max, HRmax, VO_2R or HRres. Possible consequences of comparable metabolic and respiratory responses include less variation in time to exhaustion during constant intensity exercise, a more homogenous exercise stimulus at the molecular level and less individual variation in the adaptive responses following a training program. However, many of these theoretical expectations have yet to be directly demonstrated. For example, there do not appear to be any studies describing individual variation in response to training prescribed relative to a threshold concept. In a similar way, we are not aware of any studies comparing the effect of method of relative exercise intensity prescription on the transcriptional and translation responses to single exercise bouts.

While there is a strong theoretical basis for using threshold-based exercise prescription, the challenges of determining thresholds in practice may partially explain why many researchers continue to favour the use of %VO_2max, %HRmax, %VO_2R or %HRres. For instance, when derived from a blood lactate curve, neither the AerT nor the AnT can be assumed to pinpoint the true thresholds of metabolic response in all individuals without verification. Verification of threshold measures on an individual basis would require 2-3 additional visits to the laboratory and is highly uncommon. Nevertheless, failure to verify threshold measurements may create the same individual variation in blood lactate accumulation for which %VO_2max and %HRmax have been criticized. It follows that VO_2max and HRmax, which can be measured and verified within a single laboratory visit, have a definite practical, if not theoretical, advantage over threshold measurements for prescribing exercise intensity.

It can be concluded that none of the methods of relative exercise intensity prescription under discussion are without limitations and the most appropriate measurement for a research study may differ according to factors such as exercise intensity, number of participants, participant characteristics and laboratory resources. For example, exercise prescribed at a %VO_2max of moderate intensity would be less likely

to elicit individual variation in blood lactate accumulation than exercise prescribed at a high %VO_2max. Therefore, studies involving only moderate exercise intensity (e.g. ≤ 60% VO_2max) might reasonably choose %VO_2max, %HRmax, %VO_2R or %HRres over threshold-based relative exercise intensity prescription. Furthermore, studies involving moderate intensity exercise might favour %VO_2R over %VO_2max in order to place individuals at an equivalent intensity above rest; %VO_2R and %VO_2max go on to converge as exercise intensity increases. At higher exercise intensities, the importance of accounting for threshold measurements becomes more important. Nevertheless, it could be argued that it is more important to control exercise intensity relative to threshold measurements in participants with a range of exercise capacities compared to participants with similar exercise capacities. In terms of threshold verification, it could be argued that exercise intensity prescribed relative to unverified threshold measurements would be more likely to impact results in a study with a small number of participants compared to a study with a large number of participants. Furthermore, it is acknowledged that in some cases the time frame for a research study, the available resources and other practical constraints may not allow for the most appropriate method of exercise intensity prescription. For example, it may not be feasible to verify threshold measurements for threshold measurement-based exercise prescription due to the overall testing burden on the participants. In these cases, the possibility of individuals exercising above and below threshold measurements could at least be acknowledged and considered when interpreting the study results.

As a final comment, even a brief overview of individual responses within a study allows for more balanced data interpretation and provides useful insight for subsequent studies. Therefore, irrespective of the method of relative intensity prescription used, future studies should emphasize the reporting of individual responses, particularly for studies involving small sample sizes.

Table 3.1. Reliability of VO_2max, HRmax, the aerobic threshold and the anaerobic threshold

Anchor measurement	Authors	Participants	Exercise mode	No trials	Period between trials	Calculation	Units	CV
VO_2max	Jensen & Johansen 1998 [185]	7 M	Cycling	2	7 days	n/a	l·min^{-1}	1.9 [d]
	Weston & Gabbett 2001 [188]	16 M	Cycling	2	≤ 14 days	n/a	l·min^{-1}	2.0 [a]
	Lamberts *et al.* 2009 [192]	15 M	Cycling	3	7 days	n/a	ml·kg^{-1}·min^{-1}	2.2 [b]
	Zhou and Weston 1997 [182]	10 M	Cycling	2	4 weeks	n/a	l·min^{-1}	2.5 [c]
	Amann *et al.* 2004 [193]	20 M	Cycling	2	48 h	n/a	ml·kg^{-1}·min^{-1}	2.7 [a]
	Aunola *et al.* [187]	33 M	Cycling	2	7 days	n/a	l·min^{-1}	3.8 [c]
	Wisen & Wohlfart 2004 [190]	19 M	Cycling	2	5 - 6 weeks	n/a	ml·min^{-1}	4.0 [b]
	Weltman *et al.* 1990 [191]	15 M	Running	2	≥ 7 days	n/a	l·min^{-1}	4.7 [c]
	Lourenco *et al.* 2011 [189]	11 M	Running	4	≥ 48 h	n/a	l·min^{-1}	8.5 [b]
HRmax	Lamberts *et al.* 2009 [192]	15 M	Cycling	3	7 days	n/a	bpm	0.9 [b]
	Weston & Gabbett 2001 [188]	16 M	Cycling	2	≤ 14 days	n/a	bpm	1.0 [a]
	Weltman *et al.* 1990 [191]	15 M	Running	2	≥ 7 days	n/a	bpm	1.3 [c]
	Jensen & Johansen 1998 [185]	7 M	Cycling	2	7 days	n/a	bpm	1.3 [d]
	Aunola *et al.* [187]	33 M	Cycling	2	7 days	n/a	bpm	1.9 [c]
	Wisen & Wohlfart 2004 [190]	19 M	Cycling	2	5 - 6 weeks	n/a	bpm	1.9 [b]
	Lourenco *et al.* 2011 [189]	11 M	Running	4	?	n/a	bpm	3.2 [b]
	Weltman *et al.* 1990 [191]	15 M	Running	2	≥ 7 days	LT HR	bpm	1.5 [c]
	Weltman *et al.* 1990 [191]	15 M	Running	2	≥ 7 days	LT speed	m.min^{-1}	3.0 [c]
	Aunola *et al.* [187]	33 M	Cycling	2	7 days	LT HR	bpm	3.2 [c]
	Aunola *et al.* [187]	33 M	Cycling	2	7 days	LT PO	Watts	3.5 [c]

Table continues on next page - 38

Anchor measurement	Authors	Participants	Exercise mode	No trials	Period between trials	Calculation	Units	CV
	Weston & Gabbett 2001 [188]	16 M	Cycling	2	≤ 14 days	V-slope HR	bpm	3.8 [a]
	Amann *et al.* 2004 [193]	20 M	Cycling	2	48 h	V-slope PO	Watts	4.0 [a]
	Aunola *et al.* [187]	33 M	Cycling	2	7 days	LT VO_2	$l \cdot min^{-1}$	4.3 [c]
	Weltman *et al.* 1990 [191]	15 M	Running	2	≥ 7 days	LT VO_2	$l \cdot min^{-1}$	4.3 [c]
	Meyer *et al.* 1996	10 (M & F?)	Cycling	2	24 h	V_E/VO_2 VO_2	$ml \cdot min^{-1}$	4.6 [a]
	Meyer *et al.* 1996	10 (M & F?)	Cycling	2	24 h	V-slope VO_2	$ml \cdot min^{-1}$	4.8 [a]
	Lourenco *et al.* 2011 [189]	11 M	Running	4	≥ 48 h	V-slope speed	$km \cdot h^{-1}$	5.2 [b]
	Amann *et al.* 2004 [193]	20 M	Cycling	2	48 h	V_E/VO_2 PO	Watts	5.3 [a]
AerT	Dickhuth *et al.* 1999 [197]	11 (M & F?)	Running	2	7 days	LT speed	$km \cdot h^{-1}$	5.3 [c]
	Amann *et al.* 2004 [193]	20 M	Cycling	2	48 h	V_E/VO_2 VO_2	$ml \cdot kg^{-1} \cdot min^{-1}$	5.5 [a]
	Dickhuth *et al.* 1999 [197]	11 (M & F?)	Running	2	7 days	V-slope speed	$km \cdot h^{-1}$	5.6 [c]
	Wisen & Wohlfart 2004 [190]	19 M	Cycling	2	5 - 6 weeks	V-slope HR	bpm	5.9 [b]
	Weston & Gabbett 2001 [188]	16 M	Cycling	2	≤ 14 days	V-slope VO_2	$l \cdot min^{-1}$	6.1 [a]
	Meyer *et al.* 1996	10 (M & F?)	Cycling	2	24 h	LT VO_2	$ml \cdot min^{-1}$	6.2 [a]
	Lourenco *et al.* 2011 [189]	11 M	Running	4	≥ 48 h	V-slope VO_2	$l \cdot min^{-1}$	6.2 [b]
	Amann *et al.* 2004 [193]	20 M	Cycling	2	48 h	V-slope VO_2	$ml \cdot kg^{-1} \cdot min^{-1}$	6.8 [a]
	Weston & Gabbett 2001 [188]	16 M	Cycling	2	≤ 14 days	V-slope PO	Watts	6.9 [a]
	Wisen & Wohlfart 2004 [190]	19 M	Cycling	2	5 - 6 weeks	V-slope VO_2	$ml \cdot min^{-1}$	8.6 [b]
	Wisen & Wohlfart 2004 [190]	19 M	Cycling	2	5 - 6 weeks	V-slope PO	Watts	10.4 [b]

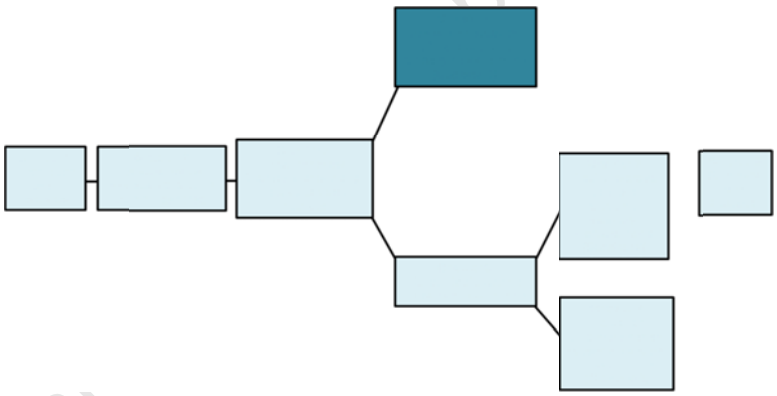
Table continues on next page - 39

Anchor measurement	Authors	Participants	Exercise mode	No trials	Period between trials	Calculation	Units	CV
AnT	Coen *et al.* 2001 [196]	25 (M & F)	Running	2	3-4 days	IAT speed	km·h^{-1}	1.1 [c]
	Weltman *et al.* 1990 [191]	14 M	Running	2	≥ 7 days	OBLA HR	bpm	1.2 [c]
	Weltman *et al.* 1990 [191]	14 M	Running	2	≥ 7 days	OBLA speed	m/min	1.7 [c]
	Coen *et al.* 2001 [196]	25 (M & F)	Running	2	3-4 days	OBLA HR	bpm	1.7 [c]
	Zhou and Weston 1997 [182]	10 M	Cycling	2	4 weeks	OBLA PO	Watts	2.1 [c]
	Coen *et al.* 2001 [196]	25 (M & F)	Running	2	3-4 days	OBLA speed	km·h^{-1}	2.2 [c]
	Jensen & Johansen 1998 [185]	7 M	Cycling	2	7 days	OBLA HR	bpm	2.4 [d]
	Aunola *et al.* [187]	33 M	Cycling	2	7 days	AnT VO_2	l·min^{-1}	2.4 [c]
	Coen *et al.* 2001 [196]	25 (M & F)	Running	2	3-4 days	IAT HR	bpm	2.5 [c]
	Dickhuth *et al.* 1999 [197]	11 (M & F?)	Running	2	7 days	LT+1.5 mmol^{-1}	km·h^{-1}	2.6 [c]
	McLellan & Jacobs 1993 [141]	11 M	Cycling	2	≥ 5 days	IAT PO	Watts	2.5 [c]
	Aunola *et al.* [187]	33 M	Cycling	2	7 days	AnT PO	Watts	3.0 [c]
	Aunola *et al.* [187]	33 M	Cycling	2	7 days	AnT HR	bpm	3.0 [c]
	Zhou and Weston 1997 [182]	10 M	Cycling	2	4 weeks	OBLA VO_2	l·min^{-1}	3.3 [c]
	Weltman *et al.* 1990 [191]	14 M	Running	2	≥ 7 days	OBLA VO_2	l·min-1	3.7 [c]
	Jensen & Johansen 1998 [185]	7 M	Cycling	2	7 days	OBLA PO	Watts	5.9 [d]
	Jensen & Johansen 1998 [185]	7 M	Cycling	2	7 days	OBLA VO_2	l·min^{-1}	7.7 [d]
	Coen *et al.* 2001 [196]	25 (M & F)	Running	2	3-4 days	IAT Lactate	mmol·l^{-1}	11.9 [c]

VO_2max = maximal oxygen uptake, HRmax = maximal heart rate, AerT = aerobic threshold, AnT = anaerobic threshold, M = male, F = female, n/a = not applicable, HR = heart rate, VO_2 = oxygen consumption, V_E = ventilatory equivalent, PO = power output, LT = lactate threshold, OBLA = onset of blood lactate accumulation (4 mmol·l^{-1} blood lactate), ? = number of male vs. female participants or time period between repeated trials not specified. a. CV = ($\sqrt{(\sum d^2/2n)}$) / sample mean where d is the sum of the between-trial differences and n is the number of participants b. CV = (SD of difference/$\sqrt{2}$) / sample mean where SD is standard deviation of the between-trial differences c. CV = (SD $\sqrt{(1 - r)}$ / sample mean where SD is average standard deviation of the repeated trials and r is the Pearson's correlation coefficient or intraclass correlation coefficient d. CV = SD / sample mean where SD is the standard deviation of the test-retest differences.

CHAPTER 4

POST-EXERCISE OXYGEN CONSUMPTION AND HEART RATE RECOVERY: ASSOCIATION WITH RPE



4.1 INTRODUCTION

The homeostatic stress associated with an exercise bout has important implications for the adaptive stimulus incurred, the appropriate timing and load of subsequent exercise bouts and the extent to which the responses of individuals performing an "equivalent" exercise bout can be compared. In the latter example, it is routine to assume that individuals exercising at the same relative intensity for a fixed duration or caloric expenditure experience a similar homeostatic stress. However, there are a number of different methods of prescribing relative exercise intensity and different methods may have different physiological implications [97]. Furthermore, one particular method of relative exercise intensity prescription can generally only standardize one aspect of an exercise response while other aspects of the exercise response are free to vary between individuals. For example, an exercise bout standardized at a certain percentage of maximal oxygen uptake (VO_2max) may produce large inter-individual variation (henceforth simply referred to as "individual variation") in heart rate (HR), blood lactate accumulation and ratings of perceived exertion (RPE) [12,13,209]. It follows that, in standardized training programs, individual variation in the overall homeostatic stress of each exercise bout may contribute to the phenomenon of "high-responders" and "low-responders" for certain training response parameters [76].

Although it is very challenging to prospectively prescribe a standardized exercise bout that produces an equivalent overall homeostatic stress in different individuals, it may be possible to retrospectively detect and/or account for individual variation in the homeostatic stress of a standardized exercise bout through measures of acute recovery. The rationale for this approach is that the homeostatic stress of an exercise bout would be expected to have a large influence on the time taken to reverse the associated exercise responses. This rationale is supported by a number of studies in which measures of dynamic autonomic or metabolic recovery were shown to be sensitive to changes in exercise intensity and/or duration [15,16,20,25,210–212]. However, it is rare for autonomic and metabolic recovery measurements to be compared within the same study and the relative sensitivity of different recovery measurements to variation in the homeostatic stress of the preceding exercise bout is not clear.

Therefore, the broad aim of the current study was to investigate to what extent variation in different metabolic and autonomic recovery measurements was related to variation in exercise responses in the preceding exercise bout. To be specific, we were particularly interested in which recovery measurement was most closely associated with Borg's RPE during submaximal exercise. Although RPE may be influenced by psychological factors [213,214], it has been strongly

correlated with HR and blood lactate measurements in a variety of populations [215] and is widely recognized as an integrated measure of the homeostatic disturbance during exercise [216]. Therefore, it was anticipated that the recovery measurement most closely associated with RPE during submaximal exercise may have the highest relative potential to represent individual variation in the homeostatic stress of the preceding exercise bout. The 4 recovery measurements under investigation were the magnitude of excess post-exercise oxygen consumption ($EPOC_{MAG}$), the time constant of the oxygen consumption recovery curve ($EPOC\tau$), 1 min heart rate recovery (HRR_{60s}) and the time constant of the heart rate recovery curve ($HRR\tau$).

4.2 METHODS

4.2.1 Participants

A heterogeneous group of 46 untrained individuals and trained runners were recruited for the study. Untrained individuals were not engaged in any regular exercise training whereas the trained individuals had accumulated a training distance of ≥ 20 km per week most weeks for the past 3 months, by self-report. All participants were required to be between the ages of 18 and 45 years, non-smokers, able to answer "no" to all the questions in a Physical Activity Readiness Questionnaire (PAR-Q) [217] and have a body mass index (BMI) < 30 $kg \cdot m^2$. The study protocol was approved by the Human Research Ethics Committee of the University of Cape Town and all participants were required to sign an informed consent before laboratory testing.

4.2.2 Experimental overview

Participants visited the laboratory on 2 occasions, approximately 3-7 days apart. Visit 1 was comprised of anthropometric measurements and a maximal treadmill test and visit 2 was comprised of a submaximal treadmill exercise followed by a period of controlled recovery. All participants were asked to refrain from any strenuous exercise the day before each session and not to exercise prior to the laboratory visit on the day of testing.

4.2.3 Visit 1: Anthropometry and maximal treadmill test

Participant's body mass and height were determined using a calibrated scale (Detecto BW-150, Webb City, USA) and stadiometer (Detecto BW-150, Webb City, USA), respectively. In addition, each participant's fat free mass and body fat % was determined using Dual-energy X-ray Absorptiometry (DXA) (Hologic Discovery-W, software version 12.1, Hologic, Bedford, MA, USA). Body mass was re-measured at the start of the 2nd laboratory visit.

The Bruce protocol [218] was used to determine VO_2max, maximal heart rate (HRmax) and total time to exhaustion (Bruce protocol time). Use of the Bruce protocol when testing both trained and untrained individuals follows the example of Dewland *et al.* [219] and was deemed a more appropriate protocol for untrained individuals than a peak running speed protocol. Before the start of the maximal treadmill test, participants were given an opportunity to become familiar with the laboratory treadmill (Quinton Instruments, Seattle, USA) and completed a self-paced warm-up followed by the Bruce protocol. All participants began the Bruce protocol from the 2nd stage (4.7 $km \cdot h^{-1}$, 12% gradient) and continued until volitional exhaustion. All participants were verbally encouraged to produce a maximal effort during the test.

HR (Suunto t6, Suunto Oy, Vantaa, Finland) and breath-by-breath respiratory gases (Jaeger Oxycon Pro, Hoechberg, Germany) were measured continuously during the maximal treadmill test. VO_2max was defined as the highest 15 s average oxygen uptake (VO_2) measured during the test, as recommended by Macfarlane [220], while HRmax was defined as the highest 2 s average HR during the test. The Oxycon Pro, which has been previously validated against the Douglas Bag system [221], was calibrated immediately before each laboratory visit using a 3 L syringe (SensorMedics®, Milan, Italy) and a reference gas of known composition (16% oxygen, 5% carbon dioxide, balance nitrogen).

4.2.4 Visit 2: Submaximal exercise and recovery trial

4.2.4.1 Pre-exercise measurements

Participants were asked to refrain from eating and to drink only water for at least 2 hours before the trial to reduce the influence of digestive processes on the metabolic measurements. Compliance with the 2 hour fast was verbally confirmed with each participant upon arrival at the laboratory. Although several studies have specified that participants be overnight fasted before measuring resting metabolism [18,31,35,222,223], this approach does not necessarily represent "real-world" behavior. The minimum 2 hour fast is in keeping with the approach of Campos *et al.* [34] and investigates the current recovery measurements under more typical free-living circumstances.

For pre-exercise VO_2 measurements, participants lay supine in a darkened room and were asked to remain quiet and still until VO_2 had stabilized and 10-15 min of stable VO_2 data had been collected using a breath-by-breath gas analysis system (Quark CPET, Cosmed, Rome, Italy). This method of obtaining a baseline measurement is similar to those reported elsewhere [35,222,223]. The gas analyzers and flow metre of the gas analysis system were calibrated shortly before the start of each trial according to the manufacturer's instructions.

4.2.4.2 Submaximal treadmill exercise

The submaximal bout consisted of 3 km of treadmill exercise at 70% VO_2max and was intended to be similar to a typical training session in the early stages of a 12 week training program for novice runners on which our laboratory was also conducting research. Treadmill speed for the exercise was inferred based on each participant's performance in the maximal treadmill test. Breath-by-breath respiratory gases (Jaeger Oxycon Pro, Hoechberg, Germany) and HR (Suunto t6, Suunto Oy, Vantaa, Finland) were measured continuously throughout the treadmill exercise. If necessary, the treadmill gradient was adjusted within the first 2-3 min of the exercise bout to elicit a VO_2 as close as possible to the target VO_2 (70% of VO_2max). Shortly before the 3 km exercise was complete, participants were asked to indicate an RPE on Borg's 6-20 RPE scale [224]. This scale had been fully explained to each participant at the start of the trial.

4.2.4.3 Recovery measurements

Immediately upon completing the 3 km exercise, the treadmill was stopped and the participant stood as still as possible for the first 5 min post-exercise to obtain a continuous recording of respiratory gases and HR for the steepest portion of the recovery curve. The Oxycon mask was then removed and the participant sat in a chair and was wheeled to a bed ± 40 m away. The participant lay down and the recovery measurements continued using the Cosmed Quark until a total of 60 min of recovery had been measured.

4.2.5 Data collection and analysis

For the baseline, exercise and recovery components of the trial, respiratory gases were expressed in 15 s averages and HR was expressed in 2 s averages.

4.2.5.1 Submaximal exercise trial baseline and exercise measurements

Pre-exercise VO_2 measurements were obtained by averaging the last ± 10 min of the stable, supine rest data. For the exercise bout, the first 3 min of data were discarded and the remainder averaged to obtain the steady-state VO_2, respiratory exchange ratio (RER) and HR for the treadmill exercise. The energy expenditure associated with the 3 km exercise was calculated according to standard caloric equivalents for oxygen at different RER values [225] and was reported as absolute energy expenditure (EE), EE relative to body mass ($EE.kg^{-1}$) and EE relative to fat free mass ($EE.kgFFM^{-1}$). The first 3 min of exercise were included when calculating EE, although it is acknowledged that RER does not reliably reflect caloric expenditure until a steady-state is acquired. To ensure that participants did indeed complete the exercise at approximately 70% of VO_2max, the average VO_2 during the exercise bout was required to be within 2 $ml \cdot kg^{-1} \cdot min^{-1}$ of

the target absolute VO_2 and/or within 5% of the 70% VO_2max target to avoid exclusion from the subsequent analysis.

4.2.5.2 HRR_{60s} and HRRτ

HRR_{60s} was calculated as the difference between the end of exercise HR (defined as the average of the last 16 s of the exercise period) and the 1 min recovery HR (defined as the average of the last 16 s of the first recovery minute) as described elsewhere [226]. The start of recovery was timed from the point at which the participant was standing upright on the stationery treadmill belt. To calculate HRRτ, a one phase decay curve was fitted to the HR data from immediately after the termination of exercise until the 60th minute of recovery using Graphpad Prism (GraphPad Prism version 5.00 for Windows, GraphPad Software, San Diego California USA). HRRτ was defined as the time constant of the heart rate recovery curve. A one phase decay has been found to be suitable for modeling heart rate recovery from submaximal exercise intensities [227].

4.2.5.3 $EPOC_{MAG}$ and EPOCτ

Recovery VO_2 (ml·min^{-1}) was plotted on the same set of axes for 0-5 min (Oxycon data) and 8-60 min (Cosmed Quark data), respectively. The start of the recovery curve was made equal to the average VO_2 of the last 3 min of exercise and a one phase decay was used to form a continuous recovery curve from the two data sets (GraphPad Prism version 5, GraphPad Software, San Diego California USA). It has previously been shown that recovery VO_2 kinetics are adequately characterized by a mono-exponential function following steady-state exercise at “moderate” and “heavy” exercise intensities [228,229]. EPOCτ was defined as the time constant of the one phase decay. $EPOC_{MAG}$ was calculated as the area under the one phase decay curve with the base of the curve adjusted to each participant’s pre-exercise VO_2.

4.2.6 Statistical analysis

All descriptive, exercise and recovery data were tested for normality using a D’Agostino and Pearson normality test. The normality test was completed for all participants, for the untrained participants only, and for the trained participants only, to ensure that all of these groups met the criteria for parametric statistical analysis. Although most variables passed the initial normality test, some variables (VO_{2max}, 3 km %VO_{2max}, EE, $EPOC_{MAG}$, EPOCτ and HRRτ within the group of all participants) required log-transformation before passing the D’Agostino and Pearson normality test. Parameters that passed the initial normality test were included in subsequent analyses in the raw form. Parameters that were log-transformed before passing the normality test are reported in the raw form but were analyzed in the log-transformed form. The height and

training volume of the untrained participants did not pass the normality test despite log-transformed hence height and training volume in trained vs. untrained participants were compared using non-parametric statistics.

All descriptive, exercise and recovery measurements, with the exception of height and training volume, were compared in the trained vs. untrained participants using an unpaired t-test. Height and training volume in the trained vs. untrained participants were compared using a Mann Whitney test. Coefficients of variation (CV) were calculated as the (standard deviation of the group/group mean)*100. The associations between anthropometric measurements and recovery measurements and exercise measurements and recovery measurements were investigated by calculating Pearson's correlation coefficients. These relationships were investigated in all participants as well as in the trained participants only. The magnitude of correlation coefficients was interpreted as ≤ 0.1 = trivial, >0.1 to ≤0.3 = small, >0.3 to ≤0.5 = moderate, >0.5 to ≤0.7 = large, >0.7 to ≤0.9 = very large and >0.9 = near perfect [230]. All of the afore-mentioned statistical analyses were conducted using Graphpad Prism 5 (GraphPad Prism 5, GraphPad Software, San Diego California USA) with statistical significance was accepted as $p < 0.05$. Data are reported as mean ± standard deviation (SD).

Multiple regression analysis was performed for each of 4 recovery measurements using the recovery measurement as the dependent variable and anthropometric and exercise measurements as possible predictive variables. A forward stepwise method was adopted with the p-value to enter the model set at $p = 0.05$ (Statistica 11, StatSoft Inc, Tulsa, OK, USA).

4.3 RESULTS

4.3.1 Participant characteristics

Although 46 participants completed the laboratory procedures, some were excluded from further analysis for disclosing ill-health during testing (1 participant) and falling outside of the target intensity of 70% VO_{2max} during the submaximal treadmill exercise (9 participants) (*see section 4.2.5.1*). The remaining 36 participants included a mixture of trained (n = 25) and untrained individuals (n = 11) and showed large inter-individual variation in body fat %, training volume, VO_2max and Bruce protocol time. These and other participant characteristics appear in Table 4.1.

Table 4.1 Participant characteristics.

	Untrained participants *n* = 11 (2M, 9F)		Trained participants *n* = 25 (12M, 13F)		All participants *n* = 36 (14M, 22F)	
	Mean ± SD (Range)	CV	Mean ± SD (Range)	CV	Mean ± SD (Range)	CV
Age (years)	31 ± 4 (25-40)	14	31 ± 5 (23-44)	17	31 ± 5 (23-44)	16
Height (cm)	168 ± 6 (159-182)	3	175 ± 10 (161-196)	6	173 ± 9 (159-196)	5
Body Mass (kg)	74.8 ± 6.8 (62.9-85.0)	9	68.8 ± 12.1 (49.1-99.2)	18	70.6 ± 11.0 (49.1-99.2)	16
Body Mass Index ($kg \cdot m^2$)	26.5 ± 2.7 (21.1-29.6)	10	22.5 ± 2.8* (17.9-29.5)	12	23.7 ± 3.3 (17.9-29.6)	14
Body fat (%)	36.5 ± 7.5 (19.0-45.4)	21	20.0 ± 7.4* (9.8-37.4)	37	25.0 ± 10.7 (9.8-45.4)	43
Training volume ($km \cdot wk^{-1}$)	0.0 ± 0.0 (0-0)	0	46 ± 26* (20-120)	55	32 ± 30 (0-120)	93
VO_2max ($ml \cdot kg^{-1} \cdot min^{-1}$)	32.6 ± 6.4 (25.4-45.8)	20	55.9 ± 7.6* (39.3-66.9)	14	48.8 ± 13.0 (25.4-66.9)	27
Bruce protocol time (min)	6.1 ± 1.4 (4.0-9.0)	23	12.1 ± 2.3* (7.8-16.4)	19	10.3 ± 3.4 (4.0-16.4)	34

M = male participants F = female participants CV = coefficient of variation. All coefficient of variation values are reported as a percentage *Significant difference between untrained participants and trained participants ($p < 0.05$)

4.3.2 Submaximal exercise and recovery measurements

The required VO_2 for the 3 km exercise bout was achieved using a combination of speed (8.2±1.2 $km \cdot h^{-1}$) and gradient (4.7±2.6%) and resulted in an average exercise intensity of 70.3±2.3%VO_2max. Although participants completed the exercise bout at the same %VO_2max, there was noticeable individual variation in the duration and energetic cost of the exercise as well as in HR and RPE responses (Table 4.2). In a similar way, there was large individual variation in all 4 recovery measurements (Table 4.2).

Table 4.2 Exercise and recovery measurements associated with the submaximal treadmill protocol

	Untrained participants *n* = 11 (2M, 9F)			Trained participants *n* = 25 (12M, 13F)			All participants *n* = 36 (14M, 22F)		
	Mean ± SD	Range	CV	Mean ± SD	Range	CV	Mean ± SD	Range	CV
%VO_2max (%)	71.7 ± 2.8	*(65.4-74.5)*	4	69.7 ± 1.8*	*(66.4-72.9)*	3	70.3 ± 2.3	*(65.4-74.5)*	*3*
Duration (min)	27.4 ± 2.5	*(22.5-30.0)*	9	20.4 ± 1.5*	*(17.8-24.0)*	8	22.6 ± 3.7	*(17.8-30.0)*	*17*
EE (kcal)	179.9 ± 27.1	*(137.5-228.9)*	15	209.5 ± 49.6	*(139.9-344.3)*	24	200.4 ± 45.7	*(137.5-344.3)*	*23*
EE·kg^{-1} (kcal·kg^{-1})	2.4 ± 0.3	*(1.9-2.9)*	12	3.0 ± 0.3*	*(2.3-3.6)*	12	2.8 ± 0.4	*(1.9-3.6)*	*16*
EE·kgFFM^{-1} (kcal·kgFFM^{-1})	3.8 ± 0.3	*(3.3-4.3)*	9	3.8 ± 0.4	*(3.2-4.4)*	9	3.8 ± 0.3	*(3.2-4.4)*	*9*
RER	0.94 ± 0.01	*(0.79-1.00)*	8	0.92 ± 0.05	*(0.85-1.02)*	6	0.92 ± 0.06	*(0.79-1.02)*	*6*
HR (bpm)	167 ± 18	*(131-188)*	11	150 ± 10	*(131-174)*	7	155 ± 15	*(131-188)*	*10*
%HRmax (%)	86.8 ± 6.4	*(73.3-92.5)*	7	81.5 ± 5.0*	*(72.9-92.3)*	6	83.1 ± 5.9	*(72.9-92.5)*	*7*
RPE (6-20)	14.7 ± 2.0	*(11.0-17.0)*	14	12 ± 1*	*(10-15)*	12	13 ± 2	*(10-17)*	*15*
$EPOC_{MAG}$ (L)	4.0 ± 1.3	*(2.3-6.0)*	33	4.2 ± 1.3	*(2.2-6.5)*	32	4.2 ± 1.3	*(2.2-6.5)*	*32*
$EPOC_{MAG}$ (ml·kg^{-1})	54 ± 16	*(32-77)*	30	61 ± 15	*(33-96)*	25	59 ± 16	*(32-96)*	*26*
EPOCτ (s)	77 ± 12*	*(61-94)*	16	61 ±7*	*(51-74)*	11	66 ± 12	*(51-94)*	*17*
HRR_{60s} (beats)	24 ± 5*	*(17.0-31.0)*	20	42 ± 9*	*(26-65)*	20	37 ± 11	*(17-65)*	*31*
HRRτ (s)	368 ± 120*	*(266-670)*	33	250 ± 59*	*(119-352)*	24	286 ± 98	*(119-670)*	*34*

M = male participants F = female participants CV = coefficient of variation. All coefficient of variation values are reported as a percentage *Significant difference between untrained participants and trained participants ($p<0.05$)

4.3.3 Relationships between anthropometric-, exercise- and recovery-measurements

HRR_{60s}, HRRτ and EPOCτ showed *moderate*-to-*large*, significant associations with the RPE of the 3 km exercise ($p < 0.05$)(Fig 4.1, Table 4.3). Variation in RPE was able to explain ~48% of the variation in HRR_{60s}, 26% of the variation in HRRτ and 23% of the variation in EPOCτ, respectively. In contrast, $EPOC_{MAG}$ showed no significant association with RPE as an absolute measure nor when expressed relative to body mass (Fig 4.1, Table 4.3). $EPOC_{MAG}$ was most closely related to EE of the exercise bout with EE able to explain ~58% of $EPOC_{MAG}$ variation. Conversely, there was no association between EPOCτ, HRR_{60s} and HRRτ and exercise EE. The relationships between each recovery measurement and anthropometric- or exercise-related measurements are shown in Table 4.3.

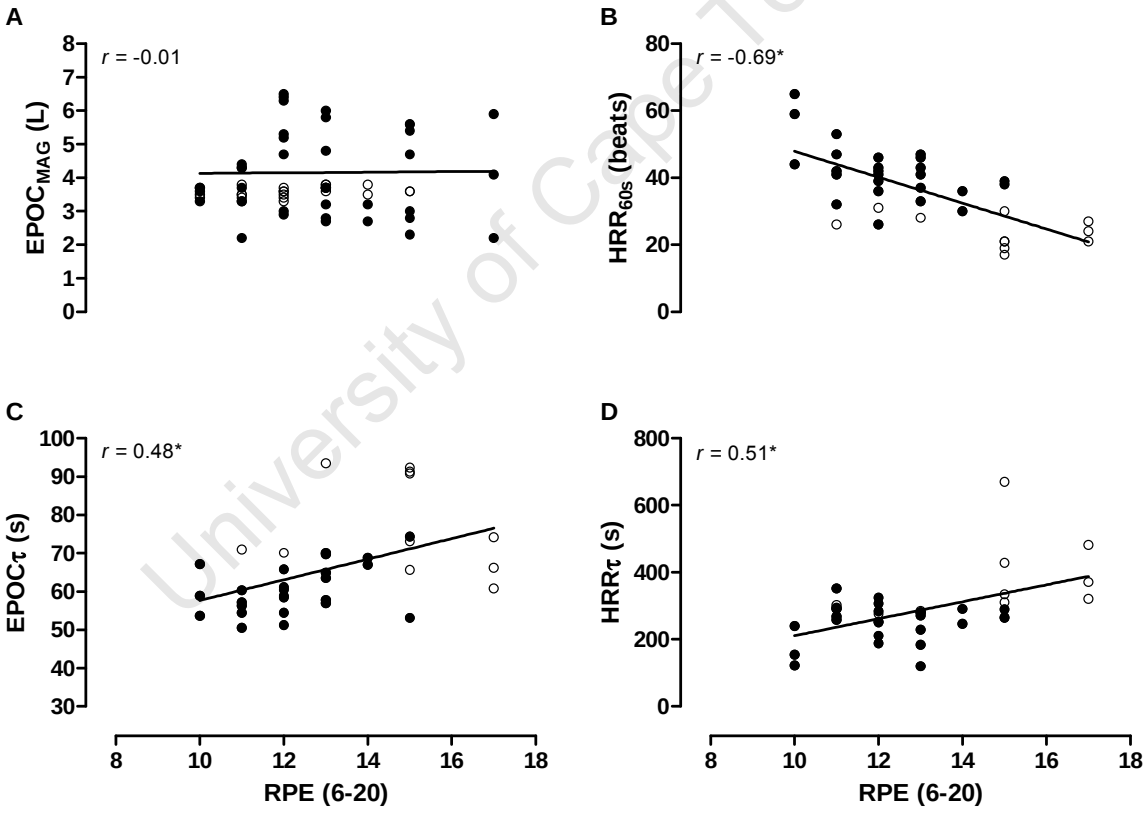


Fig 4.1 Linear regression and Pearson's correlation coefficients (*r*) for RPE and $EPOC_{MAG}$ (A), HRR_{60s} (B); EPOCτ (C) and HRRτ (D). Solid circles indicate trained participants and open circles indicate untrained participants. *Significant at $p < 0.05$.

Table 4.3 Correlations between recovery measurements and anthropometric or exercise measurements.

	Anthropometry & cardiovascular fitness				Submaximal exercise bout							
	Body Mass	Fat free mass	Body fat %†	VO_2max†	Duration†	EE	EE·kg^{-1}†	EE·kgFFM^{-1}	RER	HR	%HRmax	RPE
$EPOC_{MAG}$ (L)	0.55	0.60	-----	-----	0.37	0.76	0.50	0.45	0.45	-----	-----	-----
$EPOC_{MAG}$ (ml·kg^{-1})	-----	-----	-0.35	0.48	-0.39	0.49	0.63	0.54	----	----	----	----
EPOCτ (s)	-----	-----	0.63	0.64	-0.62	-----	-0.44	-----	-----	0.45	0.42	0.48
HRR_{60s} (beats)	-0.33	-----	-0.66	-0.67	0.71	-----	0.63	-----	-----	-0.44	-0.38	-0.69
HRRτ (s)	-----	-0.35	0.49	0.47	-0.56	-0.41	-0.60	-----	-----	0.39	-----	0.51

Only significant correlations are shown ($p < 0.05$), "-----" indicates that the correlation was not significant. †Variables collinear ($r \geq \pm 0.80$)

4.3.4 Multiple regression analysis

Although it had originally been intended to include all anthropometric and exercise measurements as possible predictors of each recovery measurement, body fat %, VO_2max, exercise duration and EE·kg^{-1} were considered collinear based on the associated Pearson's correlation coefficients (r = + or - 0.80-0.95, data not shown). As the main aim of the study was to relate recovery measurements to relative exercise responses, the decision was taken to include only EE·kg^{-1} as a predictive variable from among these closely related measurements. Therefore, the predictive variables that were selected for possible inclusion in the multiple regression analysis were body mass, fat free mass, EE, EE·kg^{-1}, EE·kgFFM^{-1}, RER, HR, %HRmax and RPE. The results of the multiple regression analysis for each recovery variable appear in Table 4.4. A combination of exercise measurements explained more variation in EPOCτ, HRR_{60s} and HRRτ than could be explained by RPE alone. The difference between the total variation explained by multiple regression analysis and the variation explained by RPE alone corresponded to ~31%, ~14% and ~14% for, HRR_{60s} and HRRτ, respectively.

Table 4.4 Multiple regression analysis for $EPOC_{MAG}$, EPOCτ, HRR_{60s} and HRRτ.

Recovery measure	Predictive variables	Adjusted R^2
$EPOC_{MAG}$ (L)	EE (0.78***); HR (0.24*)	0.61***
EPOCτ (s)	EE·kg^{-1} (-0.73***); EE·kgFFM^{-1} (0.60***); RER (0.37**)	0.54***
HRR_{60s} (beats)	EE·kg^{-1} (0.52***); RPE (-0.47***); EE·kgFFM^{-1} (-0.27*)	0.62***
HRRτ (s)	EE·kg^{-1} (-0.61***); RER (0.27*)	0.40**

Significant at *p <0.05 **p < 0.01 ***p < 0.001

4.4 DISCUSSION

The main finding of the study was that, of the 4 recovery measurements under investigation, HRR_{60s} was most closely associated with RPE following a 3 km exercise bout at 70% VO_2max. Variation in RPE was able to explain 48% of the variation in HRR_{60s}, 26% of the variation in HRRτ and 23% of the variation in EPOCτ but was not significantly associated with $EPOC_{MAG}$. Significant correlations between RPE and HRR_{60s}, HRRτ and EPOCτ do not necessarily represent cause-and-effect relationships between the homeostatic stress of an exercise bout and recovery measurements. However, they do suggest that these recovery measurements may have potential to represent inter-individual differences in the homeostatic stress of an exercise bout among individuals with a wide range of fitness levels.

4.4.1 HRR_{60s} and the homeostatic stress of an exercise bout

To the best of our knowledge, the current study was the first to investigate the association between RPE and measures of autonomic recovery and RPE and measures of metabolic recovery within the same study. However, the current findings are in keeping with previous reports of a significant association between HRR_{60s} and measures of homeostatic stress. For example, Buchheit *et al.* reported significant correlations between blood pH and HRR_{60s} (r = 0.62) and blood lactate and HRR_{60s} (r = -0.67) during repeated sprint exercise [231]. These correlations were observed in a heterogeneous group of children, adolescents and adults [231]. In a different study, Buchheit *et al.* reported a significant association between RPE and HRR_{60s} (r = -0.33) in moderately trained men after 5 min of running at 60±6 %VO_2max [232]. When considered together, the current findings and those of Buchheit *et al.* [231,232] suggest that the association between RPE and HRR is stronger amongst individuals with a range of fitness levels than among individuals with similar fitness levels. However, it is also likely that the association between RPE and HRR increases with increased exercise intensity and the relative contribution of these influences is not clear.

The current finding of significant associations between RPE and HRR could also be regarded as compatible with significant associations between HRR and physical activity levels reported previously [38,233]. For example, Lee *et al.* found a significant association between HRR_{60s} and a questionnaire-based physical activity in a (relatively heterogeneous) group of well-trained athletes (r = -067) [233] and Buchheit and Gindre found a significant association between HRRτ and questionnaire-based physical activity levels among individuals with a range of fitness levels (r = 0.55) [38]. In a heterogeneous participant group, physical activity levels may serve as a proxy

for an individual's level of training adaptation. Furthermore, increased training adaptation would be expected to result in lower homeostatic stress during a standardized exercise bout. Therefore, it could be speculated that individual variation in HRR_{60s} may represent individual variation in both levels of training adaptation and homeostatic stress during an exercise bout, amongst individuals with a range of fitness levels.

On a practical level, future studies could investigate whether the HRR_{60s} responses associated with an initial training session show potential to predict relatively high- or low- training responses to further training of a similar kind.

4.4.2 Unexplained variation in recovery measurements

In the current study, we chose Borg's RPE as a "standard" of homeostatic stress against which to compare each recovery measurement. However, there is in effect no gold standard of the homeostatic stress or (internal) training load of an exercise bout to which the recovery measurements could be compared [234]. For example, RPE could not be considered a gold standard of homeostatic stress because of the subjective nature of the ratings [213,214].

Given that RPE is an imperfect measure of homeostatic stress, inclusion of further exercise measurements would be expected to account for more variation in the recovery measurement than RPE alone. For example, multiple regression analysis showed that the inclusion of $EE{\cdot}kg^{-1}$, $EE{\cdot}kgFFM^{-1}$ and RER with or without RPE was able to explain a further ~14-31% in $EPOC\tau$, HRR_{60s} and $HRR\tau$ than RPE alone. In total, exercise measurements were able to explain 40-62% of variation in each of these recovery measurements.

Inclusion of other measures of exercise response such as blood lactate accumulation and change in body temperature may have been able to explain more of the variation in each recovery measurement that could be accounted for by the current exercise response parameters. However, some of the unexplained variation in recovery responses may also be related to the efficiency of metabolic recovery and genetic factors. In the case of the efficiency of metabolic recovery, increased training adaptation would be expected to decrease the homeostatic stress of exercise at 70% VO_2max, however training adaptations would also be expected to enhance recovery from homeostatic stress that did occur [235]. The design of the current study did not allow us to distinguish these effects and variation in the "efficiency" of recovery may have contributed to some of the variation in each recovery measurement that could not be explained by exercise responses. Finally, it is possible that some of the variation in recovery measurements may arise from genetic factors. For example, Hautala *et al.* found that a genetic polymorphism in the

acetylcholine receptor M2 DNA sequence appears to modify the HRR_{60s} response following maximal exercise both in the sedentary state and following short-term training [236]. It is likely that genetic polymorphisms contribute to variation in all 4 recovery measurements although the relative influence of genetic factors has yet to be determined.

4.4.3 Limitations

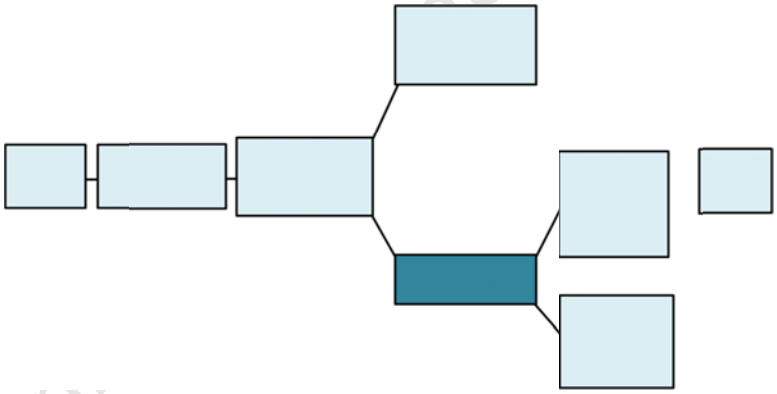
As mentioned previously, the submaximal exercise bout in the current study was intended to be similar to a training session from a 12 week training program for novice runners on which our laboratory was also conducting research. However, prescribing the exercise bout according to distance produced inter-individual variation in both exercise duration and exercise EE. In retrospect, it would have been preferable to standardize one of these exercise parameters to aid interpretation of the current findings.

4.5 CONCLUSION

In the current study, HRR_{60s}, $HRR\tau$ and $EPOC\tau$ were significantly associated with the RPE of the preceding exercise bout whereas there was no significant association between RPE and $EPOC_{MAG}$. Of these 4 recovery measurements, HRR_{60s} showed the highest relative potential to represent individual variation in the homeostatic stress of a standardized exercise bout in a group with a wide range of fitness levels.

CHAPTER 5

DAY-TO-DAY VARIATION IN POST-EXERCISE OXYGEN CONSUMPTION AND HEART RATE RECOVERY AFTER A SUBMAXIMAL TREADMILL PROTOCOL



5.1 INTRODUCTION

There is at present no "gold standard" measure of the overall homeostatic stress or internal "training load" of an exercise bout [234,237]. For example, training load based on Session Rating of Perceived Exertion (S-RPE) [238] may be influenced by subjective factors and training load calculated as a Training Impulse [239] involves population-based averages that may not be accurate for some individuals. It follows that alternative measures of training load warrant investigation. For example, a recent novel approach has been to investigate measures of the acute recovery towards resting homeostasis at the end of an exercise bout as possible measures of training load [15,16].

It is intuitive that the overall homoeostatic stress of an exercise bout would be related to the post-exercise recovery towards resting homeostasis. For example, heart rate recovery (HRR) is determined by the time course of parasympathetic reactivation and sympathetic withdrawal after the cessation of exercise [40] and Excess Post Exercise Oxygen Consumption (EPOC) has been attributed to factors such as the restoration of phosphocreatine reserves, increased body temperature, increased circulating catecholamines and lactate removal in the post-exercise period [25,35,240,241]. As possible measures of training load, these measures of acute autonomic- or metabolic- recovery have the advantage of being objective and individualized. However, it is not yet clear whether recovery measurements are sufficiently sensitive to changes in the training load of the preceding exercise bout. To be specific, for a recovery measurement to have practical value as a measure of training load, the minimal detectable change in the recovery measurement should be lower than the smallest worthwhile change in training load [242].

In previous studies, some exercise scientists have justified their own estimation of the smallest worthwhile change in a measurement [243,244] and others have calculated the smallest worthwhile change in a measurement as 0.2 x the between-subject standard deviation of the measurement [245–249] based on the principle of Cohen's effect sizes [250]. However, it is not always easy to estimate the smallest worthwhile change in a measurement, particularly when there is no gold standard of the measurement, as in the case of training load. With this in mind, an alternative, preliminary approach is to interpret experimental changes using the minimal detectable change or reliability of the measurement [10,226,251,252].

Although measurement reliability is not a substitute for the smallest worthwhile change, it can be used as an indication of whether experimental changes are "real" and/or detectable within a typical amount of biological and technical variation. While some methods of calculating measurement reliability represent between-subject reliability (e.g. intraclass correlation

coefficients), interpreting meaningful differences in a measurement requires a measure of within-subject reliability such as the typical error or typical error as a coefficient of variation. As measurement reliability can be affected by factors such as the testing protocol, equipment, participant characteristics and the period between repeated trials [184,253], it is optimal for laboratories to collect their own reliability data under similar conditions to those of subsequent studies.

It is not yet clear which measures of recovery show relatively greater or smaller potential to represent training load and in the current study, we chose to include both autonomic recovery, in the form of HRR, and metabolic recovery, in the form of EPOC. To be specific, the aim of the current study was to determine the within-subject reliability of 2 variations of HRR and 2 variations of metabolic recovery as follows: the magnitude of EPOC ($EPOC_{MAG}$), the time constant of the oxygen consumption recovery curve ($EPOC_{\tau}$), HRR within the first minute post-exercise (HRR_{60s}) and the time constant of the heart rate recovery curve (HRR_{τ}). It was envisaged that determining the reliability of these recovery measurements would allow for subsequent changes in each recovery measurement to be interpreted in the context of typical day-to-day variation and assist in the further investigation of dynamic recovery measurements as possible measures of training load. While the reliability of heart rate recovery has been reported previously [41,254–257], to the best of our knowledge this was the first study to report the reliability of EPOC magnitude and the associated recovery curve.

5.2 METHODS

Thirteen male and female runners were recruited for the study, according to the following criteria a) non-smoker b) typically running at least 3 times per week and c) able to answer “no” to all the questions in a Physical Activity Readiness Questionnaire [217]. This study was approved by the Human Research Ethics Committee of the University of Cape Town. All individuals signed an informed consent document prior to participation and agreed to visit the laboratory on 4 occasions. In contrast to Chapter 4, this study and subsequent studies used a peak treadmill running speed (PTRS) protocol rather than a Bruce protocol and adopted a modified form of the submaximal treadmill exercise and recovery protocol. For further explanation of these changes in protocol, please see the Appendix.

5.2.1 Temperature, humidity and equipment calibration

Temperature and humidity in the laboratory were kept as constant as possible during all trials and the Oxycon Pro (Jaeger Oxycon Pro®, Hoechberg, Germany), which has been previously validated against the Douglas Bag system [221], was calibrated immediately before each trial using a 3 L syringe (SensorMedics®, Milan, Italy) and a reference gas of known composition (16% oxygen, 5% carbon dioxide, balance nitrogen).

5.2.2 Visit 1: Maximal incremental treadmill test

During the first visit, participants completed a brief, self-paced warm-up on the treadmill followed by a continuous, incremental protocol for the determination of maximal oxygen uptake (VO_2max) and PTRS. The protocol started with participants running at 10 km·h^{-1} for 1 minute after which the treadmill speed was increased by 0.5 km·h^{-1} every 30 s until volitional exhaustion [258]. Participants were verbally encouraged during the test for maximal exertion and a subsequent "verification" run was included as follows: 8-10 min after the incremental protocol, participants completed a run to exhaustion at one stage higher than the highest stage completed during the incremental test to ensure that a "true" VO_2max was attained. Breath-by-breath respiratory gases (Jaeger Oxycon Pro®, Hoechberg, Germany) and heart rate (HR) (Suunto Oy®, Vantaa, Finland) were measured throughout the incremental test and verification run. VO_2max was determined as the highest 15 s average value during the incremental test or verification run [220], although the differences between the two values were small (within 2±2%). Maximal heart rate (HRmax) was defined as the highest 2 s average HR value during the incremental test and PTRS was recorded as the highest stage completed in the incremental test.

5.2.3 Visits 2, 3 and 4: Submaximal exercise and recovery trials

5.2.3.1 Overview

The 3 subsequent trials for reliability testing were performed at least 48 h after the first visit and took place on consecutive days. The start time for each trial varied by no more than 1 hour to avoid variation as a result of circadian rhythm [259]. Participants were asked to abstain from food and all drink except water for at least 2 h before each trial (this was verbally confirmed at the start of each trial) and to abstain from any other exercise training over the 3 days of the trials. The submaximal protocol consisted of a 5 min treadmill warm-up, a 20 min treadmill exercise and a 15 min passive recovery period.

5.2.3.2 Warm-up

The treadmill speed was adjusted to 70% of the participant's PTRS, measured during the first laboratory visit, and a 4 min run at 70% PTRS was timed from the point at which the participant had made a comfortable transition onto the moving belt. After 4 min, the treadmill speed was increased to 90% of PTRS and the participant ran a further 1 min at this speed. This warm-up was intended as a "priming" exercise to accelerate the matching of oxygen delivery to oxygen utilization at the onset of the subsequent exercise [260–262].

5.2.3.3 Submaximal exercise bout

Participants were given a 10 min break after the warm-up before starting the 20 min treadmill run at 70% VO_2max. For the first submaximal protocol, the initial speed was estimated based on the relationship between VO_2 and speed determined during the maximal test. Respiratory gases (Jaeger Oxycon Pro®, Hoechberg, Germany) and HR (Suunto Oy®, Vantaa, Finland) were measured continuously throughout each submaximal exercise bout and recovery period. Small adjustments were made to the speed as necessary during the early part of the run to elicit the target VO_2. These adjustments were made on the basis of an average real-time VO_2 vs. target VO_2 discrepancy of greater than 2 $ml \cdot kg^{-1} \cdot min^{-1}$. For data analysis, only trials where the average measured VO_2 within 2 $ml \cdot kg^{-1} \cdot min^{-1}$ of the target VO_2 were included in the reliability calculations.

With 1 min of the submaximal run remaining, participants were asked to indicate their rating of perceived exertion (RPE) on a Borg 6-20 RPE scale [224] and with 10 s of the 20 min run remaining, participants were given a countdown to dismount the treadmill and begin the recovery phase. After 20 min of running, the participant held the rails of the treadmill and stepped to the side of the treadmill belt. The treadmill belt was stopped immediately and the participant stepped back onto the treadmill belt and stood upright on the treadmill belt without moving or speaking- a transition of 3-5 s.

5.2.3.4 Recovery

The first part of the recovery period consisted of 1 min 30 s standing phase, during which the participants remained motionless and upright on the treadmill. The purpose of this phase was to minimize movement over the time period from which HRR_{60s} would be calculated. The 1 min 30 s of standing recovery was immediately followed by a seated phase whereby participants were given a cue to be seated upright on the chair placed directly behind them on the treadmill. The combination of a short period of standing recovery followed by seated recovery is similar to the protocol used by Bosquet *et al.* [263]. The duration of the seated phase was 13 min 30 s,

completing a total of 15 min of controlled recovery. Participants were requested to remain silent and to minimize movement during both the standing and seated phases of recovery.

5.2.4 Data analysis

All respiratory data was averaged over 15 s intervals and all HR data was averaged over 2 s intervals. Due to the slow half-life responses of both HR and VO_2 kinetics, the first 3 minutes of the 20 min submaximal run was excluded from analyses. Therefore, VO_2, minute ventilation (VE), the respiratory exchange ratio (RER) and HR during the submaximal run were calculated as the average of the final 17 min of the 20 min exercise. Energy expenditure (EE) during the 20 min submaximal run was calculated based on standard caloric equivalents for oxygen at different respiratory exchange ratio values [225].

The start of recovery was timed from the point at which the participant was standing upright on the stationery treadmill belt. HRR_{60s} was calculated as the difference between the end of exercise HR (taken as the average of the last 16 s of the submaximal run) and the 1 min recovery HR (taken as the average of the last 16 seconds of the first recovery minute) as described previously [226,256]. The start of recovery was timed from the point at which the participant was standing upright on the stationery treadmill belt. HR data over the 15 min recovery period was modeled as a one-phase decay and HRRτ was reported as the time constant of the curve (Graphpad Software, SanDiego, California, USA)[37].

Recovery VO_2 was also modeled using a one-phase decay and the time-constant of the curve was taken as EPOCτ (Graphpad Software, SanDiego, California, USA) [34]. It has previously been shown that recovery VO_2 kinetics are adequately characterized by a mono-exponential function following steady-state exercise at “moderate” and “heavy” exercise intensities [228,229]. The starting point of the curve was made to equal the average VO_2 over the final 3 min of exercise in order to minimize the influence of spurious breaths during the transition from exercise to recovery. $EPOC_{MAG}$ was calculated as the total area under the recovery curve (using Graphpad Prism 5 (Graphpad Software, SanDiego, California, USA)) and was reported in $ml \cdot kg^{-1}$.

5.2.5 Statistical analysis

Exercise and recovery measurements were normally distributed according to the D’Agostino and Pearson normality test (GraphPad Software, SanDiego, California, USA). Between-trial differences were examined for statistical significance using a repeated measures ANOVA and

significant differences were further investigated using a Tukey's post-hoc test (Statistica version 10, Stat-soft Inc., Tulsa, OK, USA). Significance was accepted at $p < 0.05$.

The effect sizes associated with between-trial differences were calculated as the difference between the means divided by pooled standard deviation and were interpreted as <0.2 = trivial, ≥0.2 to <0.5 = small, ≥0.5 to <0.8 = moderate and ≥0.8 = large [250]. Effect sizes have been used previously to interpret test reliability [247].

Measurement reliability in the form of typical error of measurement (TEM) and typical error as a coefficient of variation (CV_{TEM}) [264] and all measures of reliability were expressed with 90% confidence limits (90% CL).

5.3 RESULTS

All 13 participants completed 3 repetitions of the submaximal protocol on consecutive days, at the same time of day (within 1 h). One participant completed an additional 2 consecutive repetitions of the protocol due to problems with an Oxycon mask and HR belt respectively. All trials, except one, met the defined maximum acceptable deviation of 2 $ml \cdot kg^{-1} \cdot min^{-1}$ above or below the target VO_2 during the 20 min run. This exception was a trial 1 deviation of 2.6 $ml \cdot kg^{-1} \cdot min^{-1}$ from the target VO_2 and resulted in the participant's data set being excluded from further analysis. Participant characteristics, training habits and performance in the VO_2max test for the remaining 12 participants are shown in Table 5.1. Laboratory conditions were stable at 21.4±1.1°C and 48±5 % relative humidity over the course of the testing period.

Table 5.1 Participant characteristics, habitual training and VO_2max test performance. Data expressed as mean ± SD with range in brackets.

Variable	Men (n = 4)	Women (n = 8)	Total (n = 12)
Age (years)	30 ± 7 (22-39)	26 ± 6 (20-35)	27 ± 6 (20-39)
Body Mass Index ($kg \cdot m^2$)	22.5 ± 3.6 (18.6-26.6)	21.0 ± 1.9 (18.7-23.9)	21.5 ± 2.5 (18.6-26.6)
Training runs per week ($runs \cdot wk^{-1}$)	5 ± 1 (4-6)	4 ± 1 (3-5)	4 ± 1 (3-6)
Average distance per training run (km)	11.3 ± 3.0 (8.0-15.0)	7.3 ± 1.4 (5.0-9.0)	8.6 ± 2.7 (5.0-15.0)
VO_2max ($ml \cdot kg^{-1} \cdot min^{-1}$)	67.6 ± 5.8 (59.8-72.2)	53.2 ± 5.3 (45.7-61.0)	58.0 ± 8.8 (45.7-72.2)
PTRS ($km \cdot h^{-1}$)	19.0 ± 1.0 (18.0-20.0)	16.0 ± 1.0 (13.5-17.0)	17.0 ± 2.0 (13.5-20.0)

5.3.1 Between-trial differences in submaximal exercise and recovery measurements

The treadmill exercises were completed at 71.1±1.5 % VO_2max with an RPE of 12±1. There were no significant differences in treadmill speed, VE, RER or EE across the 3 trials and between-trial differences were associated with *trivial* effect sizes (Table 5.2). There were, however, significant differences in HR between the trials with HR significantly lower in the 2nd and 3rd trials compared to the 1st trial ($p < 0.05$). These differences were associated with *small* effect sizes. There were no significant differences in $EPOC_{MAG}$, EPOCτ, HRR_{60s} or HRRτ between trials and the associated effect sizes were for the most part *small*-to-*trivial*. One exception was a difference in EPOCτ between the 1st and 3rd trials, which was associated with a *moderate* effect size (Table 5.2).

Table 5.2 Exercise and recovery measurements from 3 repetitions of the submaximal treadmill protocol including the effect size of the between-trial changes

Phase	Variable	Trial 1	Trial 2	Trial 3	Mean	Trial 1 vs. 2		Trial 1 vs. 3		Trial 2 vs. 3	
						Effect size	Descriptor	Effect size	Descriptor	Effect size	Descriptor
Exercise	Speed ($km \cdot h^{-1}$)	10.8 ± 1.5	10.7 ± 1.5	10.6 ± 1.6	10.6 ± 1.4	0.1	*Trivial*	0.1	*Trivial*	0.0	*Trivial*
	%VO_2max (%)	71.8 ± 1.4	70.7 ± 1.5	71.0 ± 1.5	71.1 ± 1.5	0.7	*Moderate*	0.5	*Moderate*	0.2	*Small*
	VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	41.6 ± 5.9	40.9 ± 5.5	41.1 ± 5.8	41.2 ± 5.8	0.1	*Trivial*	0.1	*Trivial*	0.0	*Trivial*
	VE ($l \cdot min^{-1}$)	68.0 ± 19.9	65.6 ± 19.2	66.4 ± 18.8	66.7 ± 19.3	0.1	*Trivial*	0.1	*Trivial*	0.0	*Trivial*
	RER	0.88 ± 0.03	0.88 ± 0.03	0.88 ± 0.03	0.88 ± 0.03	0.0	*Trivial*	0.0	*Trivial*	0.0	*Trivial*
	EE (kcal)	245 ± 74	240 ± 69	243 ± 69	243 ± 69	0.1	*Trivial*	0.0	*Trivial*	0.0	*Trivial*
	HR (bpm)	161 ± 10	156 ± 11*	155 ± 11**	157 ± 11	0.4	*Small*	0.5	*Moderate*	0.1	*Trivial*
	%HRmax (%)	86 ± 5	84 ± 5*	83 ± 5**	85 ± 5	0.5	*Moderate*	0.6	*Moderate*	0.1	*Trivial*
	RPE (6-20)	12 ± 1	12 ± 2	12 ± 1	12 ± 1	0.2	*Small*	0.1	*Trivial*	0.1	*Trivial*
Recovery	$EPOC_{MAG}$ ($ml \cdot kg^{-1}$)	92 ± 14	96 ± 16	96 ± 18	95 ± 16	0.3	*Small*	0.3	*Small*	0.0	*Trivial*
	EPOCτ (s)	52 ± 10	49 ± 11	48 ± 7	50 ± 9	0.3	*Small*	0.6	*Moderate*	0.2	*Small*
	HRRτ (s)	81 ± 12	83 ± 18	83 ± 13	82 ± 14	0.2	*Small*	0.2	*Small*	0.0	*Trivial*
	HRR_{60s} (bpm)	39 ± 7	39 ± 6	40 ± 4	39 ± 5	0.1	*Trivial*	0.3	*Small*	0.4	*Small*
	VO_2 plateau ($ml \cdot kg^{-1} \cdot min^{-1}$)	4.0 ± 0.6	4.4 ± 0.9	4.5 ± 1.1	4.3 ± 0.9	0.6	*Moderate*	0.6	*Moderate*	0.0	*Trivial*
	HR plateau (bpm)	83 ± 12	80 ± 12	79 ± 10	81 ± 12	0.2	*Small*	0.3	*Small*	0.1	*Trivial*

Values are mean ±SD. Significantly different to Trial 1 *$p < 0.01$ and **$p < 0.001$. Effect size <0.2 = trivial, ≥0.2 to <0.5 = small, ≥0.5 to <0.8 = moderate and ≥0.8 = large

5.3.2 Within-subject reliability of exercise and recovery measurements

The CV_{TEM} values of VO_2, RER, HR, EE, VE and speed were similar, ranging from 1.8% (90% C.L. 1.5-2.3%) to 4.0% (90% C.L. 3.4-5.1%) (Table 5.3). The RPE rating at the end of the 20 min treadmill exercise were somewhat more variable with a CV_{TEM} value of 7.3% (90% C.L. 6.1-9.3%). However, the highest CV_{TEM} values of the current study were those of recovery measurements (Fig 5.1). For example, the day-to-day variation of EPOCτ and HRRτ was particularly high with CV_{TEM} = 12.9% (90% C.L. 10.6-16.4%) and CV_{TEM} = 10.0 (90% C.L. 8.2-12.8%), respectively.

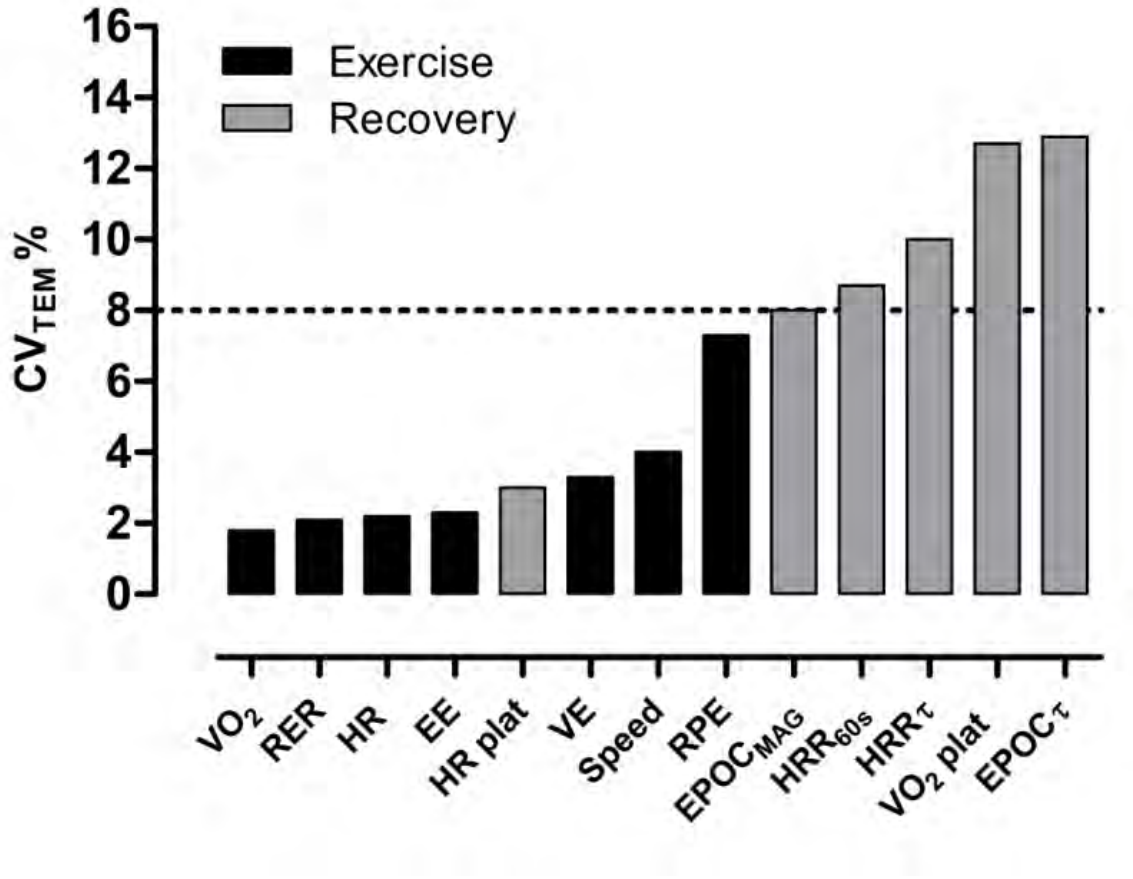


Fig 5.1 Typical error as a coefficient of variation (CV_{TEM}) for submaximal exercise and recovery measurements.

HR plat = plateau value of the heart rate recovery curve, VO_2 plat = plateau of the oxygen consumption recovery curve

Table 5.3 Day-to-day reliability of exercise and recovery measurements associated with the submaximal treadmill protocol. Data expressed with 90% confidence limits.

Phase	Variable	TEM (Measurement Units)				CV_{TEM} (%)			
		Trial 1 vs. 2	Trial 2 vs. 3	Trial 1 vs. 3	Mean	Trial 1 vs. 2	Trial 2 vs. 3	Trial 1 vs. 3	Mean
Exercise	Speed ($km \cdot h^{-1}$)	0.4 (0.3-0.7)	0.3 (0.2-0.4)	0.5 (0.2-0.8)	0.4 (0.3-0.5)	4.3 (3.2-6.8)	2.6 (1.9-4.1)	4.8 (3.6-7.6)	4.0 (3.4-5.1)
	$\%VO_2max$ (%)	1.3 (1.0-2.0)	1.2 (0.9-1.9)	1.4 (1.0-2.1)	1.3 (1.1-1.6)	1.9 (1.4-2.9)	1.7 (1.3-2.7)	1.9 (1.5-3.0)	1.8 (1.5-2.3)
	VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	0.8 (0.6-1.2)	0.7 (0.5-1.1)	0.7 (0.56-1.12)	0.7 (0.6-0.9)	1.9 (1.4-2.9)	1.7 (1.3-2.7)	1.9 (1.5-3.0)	1.8 (1.5-2.3)
	VE ($l \cdot min^{-1}$)	2.0 (1.5-3.0)	1.6 (1.2-2.5)	2.6 (2.0-4.0)	2.1 (1.8-2.6)	3.0 (2.2-4.6)	2.7 (2.0-4.1)	4.1 (3.0-6.4)	3.3 (2.7-4.1)
	RER	0.02 (0.02-0.03)	0.01 (0.01-0.02)	0.02 (0.02-0.03)	0.02 (0.02-0.02)	2.3 (1.7-3.6)	1.3 (1.0-2.0)	2.5 (1.9-3.9)	2.1 (1.7-2.6)
	EE (kcal)	5.5 (4.1-8.6)	5.3 (3.9-8.2)	6.2 (4.6-9.6)	5.7 (4.7-7.1)	2.4 (1.8-3.8)	2.0 (1.5-3.2)	2.4 (1.8-3.8)	2.3 (1.9-2.9)
	HR (bpm)	3.4 (2.5-5.3)	2.6 (2.0-4.1)	3.7 (2.8-5.8)	3.3 (2.7-4.1)	2.2 (1.6-3.4)	1.7 (1.3-2.7)	2.5 (1.9-3.9)	2.2 (1.8-2.7)
	%HRmax (%)	1.8 (1.4-2.8)	1.4 (1.1-2.2)	2.0 (1.5-3.1)	1.8 (1.5-2.2)	2.2 (1.6-3.4)	1.7 (1.3-2.7)	2.5 (1.9-3.9)	2.2 (1.8-2.7)
	RPE (6-20)	1.0 (0.8-1.6)	0.7 (0.5-1.0)	0.6 (0.5-1.0)	0.8 (0.7-1.0)	9.6 (7.1-15.2)	6.3 (4.7-10.0)	5.5 (4.1-8.7)	7.3 (6.1-9.3)

Table continues on next page - 68

Phase	Variable	TEM (Measurement Units)				CV_{TEM} (%)			
		Trial 1 vs. 2	Trial 2 vs. 3	Trial 1 vs. 3	Mean	Trial 1 vs. 2	Trial 2 vs. 3	Trial 1 vs. 3	Mean
Recovery	$EPOC_{MAG}$ ($ml \cdot kg^{-1}$)	7.2 (5.4-11.2)	7.6 (5.7-11.7)	6.7 (5.0-10.4)	7.2 (6.0-9.0)	8.3 (6.1-13.2)	8.2 (6.0-12.9)	7.6 (5.7-12.1)	8.0 (6.7-10.3)
	$EPOC\tau$ (s)	7.4 (5.5-11.4)	4.8 (3.6-7.4)	6.1 (4.5-9.4)	6.2 (5.1-7.7)	15.5 (11.4-25.1)	10.5 (7.8-16.8)	12.1 (8.9-19.3)	12.9 (10.6-16.4)
	$HRR\tau$ (s)	8.9 (6.5-14.1)	8.0 (5.9-12.7)	5.6 (4.2-8.7)	7.6 (6.3-9.6)	11.9 (8.7-19.7)	10.1 (7.4-16.6)	7.6 (5.7-12.1)	10.0 (8.2-12.8)
	HRR_{60s} (bpm)	3.8 (2.8-6.0)	3.3 (2.4-5.2)	2.8 (2.1-4.4)	3.3 (2.6-4.4)	9.7 (7.1-15.9)	8.7 (6.4-14.3)	7.8 (5.8-12.3)	8.7 (7.2-11.2)
	VO_2 plateau ($ml \cdot kg^{-1} \cdot min^{-1}$)	0.5 (0.4-0.8)	0.4 (0.3-0.6)	0.6 (0.4-0.9)	0.5 (0.4-0.6)	13.1 (9.6-21.0)	9.7 (7.2-15.4)	14.9 (11.0-24.1)	12.7 (10.5-16.2)
	HR plateau (bpm)	2.2 (1.7-3.5)	2.4 (1.8-3.7)	2.9 (2.2-4.6)	2.6 (2.1-3.2)	2.5 (1.8-3.8)	3.0 (2.2-4.6)	3.4 (2.5-5.4)	3.0 (2.5-3.8)

n = 11 rather than 12 for $HRR\tau$ and HRR_{60s} in trials 1 vs. 2 and 2 vs. 3. TEM = Typical Error of Measurement, CV_{TEM} = TEM as a % of the mean

5.4 DISCUSSION

A recent novel approach to quantifying training load has been to investigate measures of acute post-exercise recovery for their potential in this role [15,16]. However, an important practical consideration for this novel approach is whether changes in training load (i.e. changes in the intensity and/or duration of an exercise bout) can be detected above the day-to-day technical and biological variation associated with a particular recovery measurement. Before this consideration can be addressed, typical day-to-day variation or within-subject reliability must be established. Therefore, the aim of the current study was to determine the within-subject reliability of 2 variations of HRR and 2 variations of EPOC.

The main finding of the study was that in moderately-trained individuals completing the current laboratory protocol, the minimal detectable change or within-subject reliability was 8% for $EPOC_{MAG}$, 13% for EPOCτ, 9% for HRR_{60s} or 10% for HRRτ. Changes in training load (i.e. changes in exercise intensity and/or duration) that produce changes in excess of these CV_{TEM} values are likely to be of practical significance. However, the smallest worthwhile difference in training load is currently unknown.

In the case of EPOCτ and HRRτ, CV_{TEM} values were 3-5% smaller in trials 2 vs. 3 when compared to trials 1 vs. 2 and incorporating a familiarization trial would allow for smaller changes in these variables to be considered practically significant. The increased reliability of these recovery measurements in trials 2 and 3 may have been linked to the small decrease in HR between trials 1 and 2 that was not present between trials 2 and 3. The higher HR during trial 1 is most likely to reflect a degree of nervous tension on the part of participants when completing the protocol for the first time that was no longer present in subsequent trials. In contrast to EPOCτ and HRRτ, $EPOC_{MAG}$ and HRR_{60s} showed ≤ 1% change in CV_{TEM} in trials 1 vs. 2 and 2 vs. 3. A brief discussion of the current findings in the context of previous reliability studies follows.

5.4.1 Reliability of HRR_{60s} and HRRτ

The TEM for HRR_{60s} of 3 bpm is in the present study is equivalent to the 3 bpm reported by Lamberts *et al.* and somewhat smaller than the TEM or standard error of measurement (SEM) values of 8-10 bpm reported elsewhere [41,254,256,263]. Conversely, the HRRτ TEM of ~8 s is similar to what was reported by Al Haddad *et al.* (HRRτ TEM = 7 s)[254] and Buchheit *et al.* [41] (HRRτ SEM = 7 s) but somewhat smaller than the HRRτ SEM reported by Bosquet *et al.* [263] (SEM = 13 s). Despite some similarities in the TEM values for HRR_{60s} and HRRτ between the current study and

previous studies, there has been little consensus as to which of these measurements is more reliable. In the current study and in the study by Bosquet *et al.*, HRR_{60s} was more reliable than HRRτ whereas Buchheit *et al.* found HRRτ to be more reliable than HRR_{60s} and Al Haddad reported very similar reliability for HRR_{60s} and HRRτ.

Variation in the reported reliability of HRR_{60s} and HRRτ is likely to result from differences in methodology and study design. For example, the current study differed from previous studies in factors such as the training status of the participants, exercise mode, exercise intensity, exercise duration, the number of trial repetitions, the time period between trial repetitions and the equipment used [41,254,256,257,263]. These factors may have important implications for the reliability of a measurement [184,253] and allude to the need for protocol-specific, laboratory-specific reliability analyses where possible. As an example, Lamberts *et al.* demonstrated that the TEM of HRR_{60s} was smaller following shuttle runs that elicited 86-93% of HRmax when compared to shuttle runs that elicited %HRmax values above or below this range [256].

5.4.2 Reliability of $EPOC_{MAG}$ and EPOCτ

Although some previous studies have reported the reliability of oxygen consumption measurements following submaximal exercise [228,265,266], these studies used the aggregate of multiple square-wave bouts to model transition-phases in oxygen kinetics, rather than the reliability of the oxygen consumption recovery curve associated with EPOC. Furthermore, although Jacobsen *et al.* [30] reported the mean intra-individual coefficient of variation for EPOC magnitude, the EPOC trials were repeated before and after a 9 month training period and could not be compared with the outcomes of the current study in a meaningful way. Therefore, to the best of our knowledge, this was the first study to report the reliability of $EPOC_{MAG}$ and EPOCτ. $EPOC_{MAG}$ was noticeably more reliable than EPOCτ and these measurements were the most- and least- reliable of 4 main recovery outcomes in the study, respectively.

5.4.3 Reliability of submaximal exercise measurements

Although the main focus of this study was to determine the reliability of the recovery measurements, it is briefly noted that the CV's of the submaximal exercise measurements were similar to- or smaller than- those reported previously. For example, the current VE CV_{TEM} of 3.3% was slightly smaller than the VE CV's of 4-5% reported previously [267–269], the current CV_{TEM} of VO_2 of 1.8% was similar to the VO_2 CV's of 2-4% reported previously [247,267,268,270] and the RER CV_{TEM} of 2.1% was slightly smaller than the RER CV's of 3-4% reported previously [247,268]. Finally,

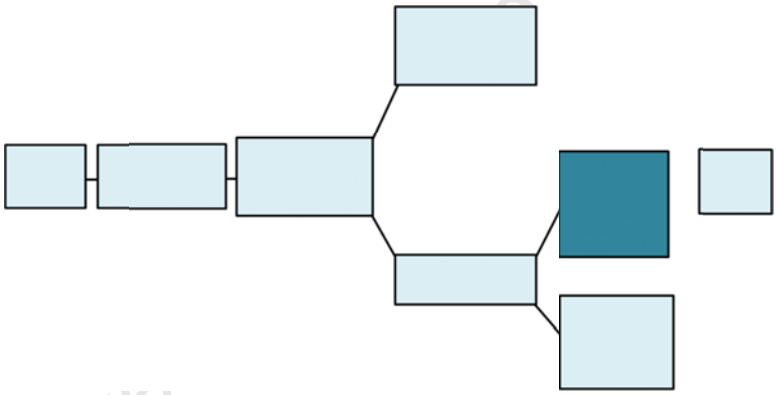
the HR CV_{TEM} of 2.2% was in the range of submaximal HR CV's of 1-4% reported previously [247,256,267,268].

5.5 CONCLUSION

The current study determined the minimal detectable change or within-subject reliability (CV_{TEM}) of $EPOC_{MAG}$, $EPOC\tau$, HRR_{60s} and $HRR\tau$ in moderately-trained individuals who completed a submaximal treadmill protocol on 3 occasions. Future studies could investigate whether changes in training load (i.e. changes in exercise intensity and/or duration) produce changes in recovery measurements that are in excess of this day-to-day variation and so provide insight into whether acute recovery measurements have practical value as markers of training load.

CHAPTER 6

EFFECT OF EXERCISE INTENSITY ON POST-EXERICSE OXYGEN CONSUMPTION AND HEART RATE RECOVERY



6.1 INTRODUCTION

During a bout of exercise, factors such as the exercise intensity and duration interact to produce the overall homeostatic stress or "training load" of the session. The specific nature of the homeostatic stress determines which adaptive signaling pathways will be activated [75,156]. Furthermore, the magnitude of the homeostatic stress has important implications for balancing the distribution of training and recovery in order to optimize adaptation and performance [82]. Existing approaches for quantifying the homeostatic stress of a training session have limitations [234]. For example, Session RPE [238] may be over- or under-reported based on subjective factors and the Training Impulse (TRIMP) method [239] uses population-based averages in the training load calculation. It follows that alternative methods for quantifying the homeostatic stress of an exercise bout warrant further investigation.

One approach that shows potential as a means of quantifying the homeostatic stress of an exercise bout is to measure the dynamic recovery towards resting homeostasis following the end of the exercise bout [15,16]. The rationale for this approach is that the severity of the homeostatic stress would be expected to have a large influence on the characteristics of the recovery curve and/or the area under the recovery curve. For example, the depletion of phosphocreatine reserves, increase in body temperature and increase in circulating catecholamines during an exercise bout are among the factors thought to contribute to the magnitude of excess post exercise oxygen consumption (EPOC) [25,35,241]. In a similar way, the extent of the parasympathetic withdrawal and increase in sympathetic activity during an exercise bout may influence the time-course of parasympathetic reactivation and sympathetic withdrawal after the exercise bout has been terminated. This post-exercise recovery towards resting autonomic tone can be measured indirectly through heart rate recovery (HRR) or changes in post-exercise heart rate variability [29,39,271,272].

For the most part, the magnitude and time course of EPOC have been investigated in the context of weight loss [17–24] and post-exercise changes in autonomic function, such as HRR, have been related to training status [28] and the risk of mortality [26,27]. However, there are examples of post-exercise changes in autonomic balance or metabolism being linked to the homeostatic stress or "training load" of the preceding exercise bout [15,16,212]. For example, Seiler *et al.* [212] hypothesized that the time course of the return to resting levels of heart rate variability was indicative of the overall magnitude of the stress response induced by the preceding exercise bout and Kaikkonen *et al.* [16] described EPOC as a "physiological reference" for the training load of the preceding exercise bout.

In addition to these direct references, a number of studies have supported the association between the homeostatic stress of exercise and measurements of dynamic recovery by reporting recovery measurements following exercise of different exercise intensities and durations. Exercise intensity and duration are two key determinants of the homeostatic stress of an exercise bout and the associated adaptive stimulus, with exercise intensity established as the more dominant influence [273,274]. It follows that a potential measure of the homeostatic stress of exercise should be sensitive to these exercise parameters. For example, EPOC has a curvilinear increase with increased exercise intensity and a linear increase with increased exercise duration at exercise intensities ≥ 50% of maximal oxygen uptake (VO_2max) [20,25]. In a similar way, several studies have demonstrated slower heart rate recovery [29,33,37,254,275] or a delay in the return to resting heart rate variability [15,210–212,276,277] at higher vs. lower exercise intensities, although these observations were not always analyzed statistically [33,37,254].

While both metabolic- and autonomic-type recovery measurements appear to be influenced by the homeostatic stress of the preceding exercise bout, differences in the physiological determinants and/or day-to-day variation of each measurement may result in some measurements showing greater sensitivity to detect changes in homeostatic stress than others [15,16]. With this in mind, the aim of the current study was to compare 4 different recovery measures of metabolic or autonomic recovery at 3 different exercise intensities. It was anticipated that increased exercise intensity would increase the homeostatic stress of the exercise bout which, in turn, would result in slower recovery towards resting homeostasis. Therefore, a recovery measurement suitable to represent the homeostatic stress of an exercise bout would be expected to show (i) slower recovery with increased exercise intensity, (ii) sensitivity to both smaller- and larger- changes in exercise intensity, and (iii) consistent responses on an individual level as well as on a group mean level. The 4 recovery measurements under investigation were EPOC magnitude ($EPOC_{MAG}$), the time constant of the VO_2 recovery curve ($EPOC\tau$), heart rate recovery within the first minute post exercise (HRR_{60s}) and the time constant of the heart rate recovery curve ($HRR\tau$).

6.2 METHODS

6.2.1 Participants

Thirty-eight male and female runners between the ages of 20 and 40 years were recruited for the study. All participants had been training regularly for at least 3 months prior to participation, indicated that they were able to complete a 10 km run in 60 min or less and were able to answer "no" to all the questions in a Physical Activity Readiness Questionnaire (PAR-Q)[217]. Prior to participation, all participants signed an informed consent document and provided a 3 month training history including the typical number of training sessions per week, typical total weekly training distance and current 10 km time. The study was approved by the University of Cape Town's Human Research Ethics Committee prior the recruitment of participants.

6.2.2 Visits 1 and 2: Maximal incremental treadmill tests

During the first visit to the laboratory, participants completed a self-paced warm-up and treadmill familiarization followed by a continuous incremental treadmill protocol to determine VO_2max, maximal heart rate (HRmax) and peak treadmill running speed (PTRS). As described in Chapter 5, the protocol started with the participants running at a speed 10 km·h^{-1} for 1 min followed by an increase of 0.5 km·h^{-1} every 30 s until exhaustion [278]. The second visit to the laboratory involved a repeat of the maximal incremental running test. However, on this occasion participants also completed a verification bout performed 8-10 min after the termination of the incremental test. The verification bout consisted of a run to exhaustion at a workload 0.5 km·h^{-1} higher than the highest completed workload from the incremental test and was included to ensure that a "true" VO_2max was attained. Breath-by-breath respiratory gases (Jaeger Oxycon Pro®, Hoechberg, Germany) and heart rate (HR) (Suunto Oy®, Vantaa, Finland) were measured throughout each incremental test and during the verification run. Although the differences in peak VO_2 from the incremental test and verification bout were generally small (within 4±3%) and similar to what has been reported elsewhere [166], VO_2max was defined as the highest 15 s average VO_2[220] value when comparing the 2nd incremental protocol and verification bout. Maximal heart rate (HRmax) was defined as the highest 2 s average HR value during the incremental protocol and PTRS was recorded as the highest stage completed in an incremental test.

6.2.3 Visits 3, 4 and 5: Submaximal exercise and recovery trials

6.2.3.1 Overview

The 3rd, 4th and 5th visits to the laboratory were designed to compare recovery measurements following 20 min of treadmill exercise at 60%, 70% and 80% of VO_2max. Apart from the differences in exercise intensity, these trials followed the same submaximal exercise and recovery protocol described in Chapter 5.

The 3 exercise intensities were assigned in random order and the 3 trials were completed within a period of approximately 7 days. The time of day the trial started for a particular participant (3rd visit) was kept consistent to within ± 1 h on subsequent visits (4th and 5th) to avoid variation as a result of circadian rhythm [259]. Participants were asked to refrain from hard training over this period and to restrict themselves to no more than light exercise on the day preceding each trial. Participants were also asked to abstain from training on the day of each trial and to abstain from food and all drink except water for a minimum of 2 h before reporting to the laboratory.

6.2.3.2 Warm-up

Participants completed a 5 min warm-up on the treadmill, which was comprised of 4 min at 70% of PTRS and 1 min at 90% PTRS. This warm-up also functioned as a "priming" exercise to accelerate the matching of oxygen delivery to utilization at the onset of the subsequent exercise [260–262]. The warm-up was followed by a 10 min break during which participants could relax or perform light stretching exercises prior to the submaximal run.

6.2.3.3 Submaximal exercise bout

The treadmill speed predicted to elicit 60%, 70% or 80% of a participant's VO_2max was estimated based on the relationship between VO_2 and treadmill speed during the incremental tests. Respiratory gases (Jaeger Oxycon Pro®, Hoechberg, Germany) and HR (Suunto Oy®, Vantaa, Finland) were measured continuously throughout each submaximal exercise bout and recovery period. Small adjustments were made to the treadmill speed as necessary during the early part of the run to elicit the target VO_2. These adjustments were made on the basis of an average real-time VO_2 vs. target VO_2 discrepancy of greater than 2 $ml \cdot kg^{-1} \cdot min^{-1}$.

With 1 min of the submaximal exercise remaining, participants were asked to indicate their rating of perceived exertion (RPE) on a Borg 6-20 RPE scale [224]. This scale was fully explained to each participant before the start of the trial. With 10 s of the 20 min run remaining, participants were given a countdown to dismount the treadmill and begin the recovery phase. After 20 min of

running, the participant held the handrails of the treadmill and stepped to the side of the treadmill belt. The treadmill belt was stopped immediately and the participant stepped back onto the treadmill belt and stood upright on the stationary treadmill belt without moving or speaking- a transition of approximately 3-5 s.

6.2.3.4 Recovery

The first part of the recovery period consisted of a 1 min 30 s standing phase, during which the participant remained motionless and upright on the treadmill. The purpose of this phase was to minimize movement over the time period from which HRR_{60s} would be calculated. The 1 min 30 s of standing recovery was immediately followed by a seated phase whereby participants were given a cue to be seated upright on the chair placed directly behind them on the treadmill. The combination of a short period of standing recovery followed by seated recovery is similar to the protocol used by Bosquet *et al.* [263]. The duration of the seated phase was 13 min 30 s, completing a total of 15 min of controlled recovery. Participants were requested to remain silent and to minimize movement during both the standing and seated phases of recovery. VO_2 and HR kinetics were measured continuously in the transition from exercise to recovery and throughout the recovery period.

6.2.4 Data analysis

All respiratory data was averaged over 15 s intervals and all HR data was averaged over 2 s intervals. Due to the slow half-life responses of both HR and VO_2 kinetics, the first 3 minutes of the 20 min submaximal run was excluded from analyses. Therefore, VO_2, the respiratory exchange ratio (RER) and HR during the submaximal run were calculated as the average of the final 17 min of the 20 min exercise. Energy expenditure (EE) during the submaximal run was calculated based on standard caloric equivalents for oxygen at different respiratory exchange ratio values [225]. EE was calculated based on the full 20 min of exercise although it is acknowledged that RER may not accurately reflect caloric expenditure during the transition from rest to steady state.

HRR_{60s} was calculated as the difference between the end of exercise HR (taken as the average of the last 16 s of the submaximal run) and the 1 min recovery HR (taken as the average of the last 16 seconds of the first recovery minute) as described previously [226,256]. In addition, HR data over the 15 min recovery period was modeled using a one-phase decay and $HRR\tau$ was reported as the time constant of the HR recovery curve (GraphPad Software, SanDiego, California, USA)[37].

Recovery VO_2, composed of 60 data points, was visually inspected for spurious values or values which were not "physiological" (e.g. VO_2 < 1.0 ml·kg^{-1}·min^{-1}). For the majority of trials, ≤ 3 data points were removed. However, 4 participants had 4-6 data points removed from a trial and 2 participants had 9-10 data points removed from a trial. Recovery VO_2 data was then modelled using a one-phase decay and EPOCτ was taken as the time-constant of the VO_2 recovery curve (GraphPad Software, SanDiego, California, USA)[34]. The starting point of the curve was made to equal the average VO_2 over the final 3 min of exercise in order to minimize the influence of spurious breaths at the end of exercise on the span of the recovery curve. It has previously been shown that recovery VO_2 kinetics are adequately characterized by a mono-exponential function following steady-state exercise at "moderate" and "heavy" exercise intensities [228,229].

$EPOC_{MAG}$ was calculated as the total area under the curve using Graphpad Prism 5 (GraphPad Software, SanDiego, California, USA). The area under the curve was not corrected for a baseline measurement as total area under the curve was found to be a more precise method of comparing individuals on different occasions according to the work of Jacobsen *et al.* [30].

6.2.5 Statistical analysis

Exercise and recovery measurements at each exercise intensity were tested for normality using the D'Agostino and Pearson normality test. Data that passed the initial normality test were analyzed in the raw form for intensity-related differences using a Repeated Measures ANOVA and Tukey post-hoc test. However, EE at 70% VO_{2max}, EE at 80% VO_{2max} and %HRmax at 60% VO_2max did not pass the initial normality test. To make these measurements suitable for parametric statistics, all EE and %HRmax data were then log transformed and subsequently passed the D'Agostino and Pearson normality test. For these measurements, a Repeated Measures ANOVA and Tukey post-hoc test were performed on the log transformed values. All statistical analyses were conducted using Graphpad Prism 5 (GraphPad Software, SanDiego, California, USA) and significance was accepted at $p < 0.05$. Data are presented as mean ± standard deviation (SD).

Intensity-related differences were also investigated by calculating Cohen's effect sizes [250]. Effect sizes (d) were calculated as the difference between the group means divided by the pooled standard deviation of both groups and were interpreted using Cohen's original descriptors as thresholds i.e. small (0.5 > d ≥ 0.2), moderate (0.8 > d ≥ 0.5) or large (d ≥0.8). Effect sizes of less than 0.2 were described as "trivial". As effect sizes assume normally distributed data, the

between-trial effect sizes for exercise EE and %HRmax were calculated using the log transformed values.

On an individual level, changes in each recovery measurement were compared to the day-to-day reliability of the measurement as determined in Chapter 5. The current study involved the same equipment and laboratory setting as the study in Chapter 5 and the current participants, although different, were of a comparable physical fitness to the participants in Chapter 5. Between-trial changes in individual recovery measurements were interpreted as having practical significance if they exceeded the following typical error values (expressed as a coefficient of variation (CV_{TEM}) with 90% confidence limits): 8.0% (6.7% to 10.3%) for $EPOC_{MAG}$, 12.9% (10.6% to 16.4%) for EPOCτ, 10.0% (8.2% to 12.8%) for HRRτ and 8.7% (7.2 to 11.2%) for HRR_{60s}.

6.3 RESULTS

Although 38 runners completed all 5 laboratory testing sessions, some participants were excluded from statistical analysis on the basis of a > 2 ml·kg^{-1}·min^{-1} deviation from the target VO_2 during a submaximal exercise bout (2 participants) or loss of critical VO_2 or HR data during a recovery period (4 participants). The characteristics of the remaining 32 participants appear in Table 6.1.

Table 6.1 Participant characteristics. Data expressed as mean ± SD with range in brackets.

Variable	Men (n = 20)	Women (n = 12)	Total (n = 32)
Age (years)	30 ± 6 (20–40)	28 ± 4 (21–37)	29 ± 5 (20–40)
Height (cm)	179 ± 7 (168–196)	166 ± 8 (149–175)	174 ± 10 (149–196)
Body mass (kg)	77.5 ± 7.8 (63.2–94.5)	59.7 ± 7.2 (48.2–73.3)	70.8 ± 11.5 (48.2–94.5)
Body Mass Index (kg·m^2)	24.4 ± 2.1 (19.3–27.7)	21.7 ± 2.6 (18.0–26.0)	23.4 ± 2.6 (18.0–27.7)
Training frequency (runs·wk^{-1})	4 ± 1 (2–6)	4 ± 1 (3–6)	4 ± 1 (2–6)
Total training distance (km·wk^{-1})	39 ± 16 (20-90)	39 ± 17 (18–70)	39 ± 16 (18–90)
Self-reported 10 km time (min)	46 ± 6 (34–55)	53 ± 6 (43–60)	48 ± 7 (34–60)
VO_2max (ml·kg^{-1}·min^{-1})	60.2 ± 4.8 (51.9–66.8)	51.9 ± 5.9 45.1–62.3	57.1 ± 6.6 (45.1–66.8)
PTRS (km·h^{-1})	19.0 ± 1.0 (16.0–22.0)	16.0 ± 1.0 (13.5–18.0)	17.8 ± 2.0 (13.5–22.0)

VO_2max = maximum oxygen uptake, *PTRS* = Peak Treadmill Running Speed

6.3.1 Submaximal exercise parameters

Exercise at 60%, 70% and 80% of VO_{2max} was associated with significant differences in treadmill speed, HR, EE and RPE ($p < 0.0001$)(Table 6.2). Furthermore, there were *large* between-trial effects for speed, HR and RPE ($d = 0.9$-1.6) whereas between-trial differences in EE and RER were associated with *moderate*-to-*large* effect sizes ($d = 0.5$-1.3) (Table 6.2).

6.3.2 Recovery parameters: group level responses

Each of the 4 recovery measurements showed a different pattern of response following exercise at 60%, 70% and 80% of VO_2max. $EPOC_{MAG}$ was the only measurement to differ significantly across all 3 exercise intensities ($p \leq 0.003$), showing slower recovery with each increase in exercise intensity. EPOCτ also demonstrated slower recovery with increased exercise intensity, however between-trial differences were only significant when comparing the 70% vs. 60% and 80% vs. 60% VO_2max trials ($p \leq 0.004$). Between trial-changes in HRRτ were only significantly slower when comparing the 80% vs. 60% VO_2max trials ($p = 0.01$). In contrast to $EPOC_{MAG}$, EPOCτ and HRRτ responses, HRR_{60s} showed faster recovery when comparing the 70% vs. 60% and 80% vs. 60% VO_2max trials ($p \leq 0.0002$) but did not differ significantly between the 80% and 70% VO_2max trials.

EPOCτ and HRRτ responses were associated with differences in the value at which recovery VO_2 or HR plateaued during the 15 min recovery period. The plateau in recovery VO_2 was significantly higher following exercise at 80% VO_2max vs. 60% or 70% VO_2max ($p \leq 0.0003$) whereas the plateau in recovery HR was significantly different across all 3 exercise intensities ($p \leq 0.03$). A representative example of one individual's recovery responses is shown in Figure 6.1.

Although all 4 recovery measurements were significantly different when comparing the 80% vs. 60% VO_2max trials, differences in $EPOC_{MAG}$ ($d = 1.2$) and EPOCτ ($d = 0.9$) were associated with larger effect sizes than differences in HRR_{60s} ($d = 0.6$) or HRRτ ($d = 0.5$)(Table 6.2). However, significant differences in $EPOC_{MAG}$, EPOCτ and HRR_{60s} when comparing the 70% vs. 60% VO_2max trials were associated with similar, *moderate* effect sizes ($d = 0.5$-0.6)(Table 6.2).

Table 6.2 Exercise and recovery parameters at 3 submaximal exercise intensities

	Parameter	60% VO_{2max}	70% VO_{2max}	80% VO_{2max}	Effect sizes					
					70% vs. 60%		80% vs. 70%		80% vs. 60%	
Exercise	%VO_{2max} (%)	60.8 ± 1.4	70.5 ± 1.2*	81.0 ± 1.6*†	1.9	*Large*	1.9	*Large*	2.0	*Large*
	Speed (km·h^{-1})	9.4 ± 1.4	11.1 ± 1.4*	12.7 ± 1.7*†	1.1	*Large*	0.9	*Large*	1.5	*Large*
	EE (kcal)	234 ± 51	273 ± 60*	318 ± 68*†	0.6	*Moderate*	0.6	*Moderate*	1.1	*Large*
	RER	0.86 ± 0.03	0.88 ± 0.03	0.92 ± 0.03*†	0.5	*Moderate*	1.1	*Large*	1.3	*Large*
	HR (bpm)	144 ± 10	157 ± 9*	169 ± 10*†	1.1	*Large*	1.1	*Large*	1.6	*Large*
	%HRmax (%)	77 ± 5	84 ± 4*	90 ± 4*†	1.2	*Large*	1.2	*Large*	1.6	*Large*
	RPE (6-20)	10 ± 1	12 ± 1*	14 ± 2*†	1.1	*Large*	1.2	*Large*	1.6	*Large*
Recovery-outcomes	$EPOC_{MAG}$ (ml·kg^{-1})	86 ± 18	95 ± 15*	111 ± 18*†	0.5	*Moderate*	0.9	*Large*	1.2	*Large*
	EPOCτ (s)	44 ± 9	50 ± 9*	53 ± 8*	0.6	*Moderate*	0.3	*Small*	0.9	*Large*
	HRR_{60s} (bpm)	30 ± 7	35 ± 8*	35 ± 9*	0.7	*Moderate*	0.0	*Trivial*	0.6	*Moderate*
	HRRτ (s)	91 ±23	94 ±22	103 ±27*	0.2	*Small*	0.4	*Small*	0.5	*Moderate*
Recovery curve plateaus	VO_2 plateau (ml·kg^{-1})	4.2 ± 1.2	4.3 ± 0.9	5.1 ± 1.1*†	0.1	*Trivial*	0.7	*Moderate*	0.7	*Moderate*
	HR plateau (bpm)	79 ± 11	82 ± 10*	92 ± 10*†	0.3	*Small*	0.9	*Large*	1.1	*Large*

Values are mean ±SD *VO_{2max}* = maximal oxygen uptake, *EE* = energy expenditure, *RER* = respiratory exchange ratio, *HR* = heart rate, *HR_{max}* = maximal heart rate, *RPE* = rating of perceived exertion, $EPOC_{MAG}$ = magnitude of excess post-exercise oxygen consumption, EPOCτ = time constant of the oxygen consumption recovery curve, HRR_{60s} = 1 min heart rate recovery, HRRτ = time constant of the heart rate recovery curve, *VO_2* = oxygen uptake *Significantly different to 60% trial ($p < 0.05$), †Significantly different to 70% trial ($p < 0.05$). Effect size descriptions <0.2 = trivial, ≥ 0.2 to < 0.5 = small, ≥ 0.5 to < 0.8 = moderate and ≥ 0.8 = large

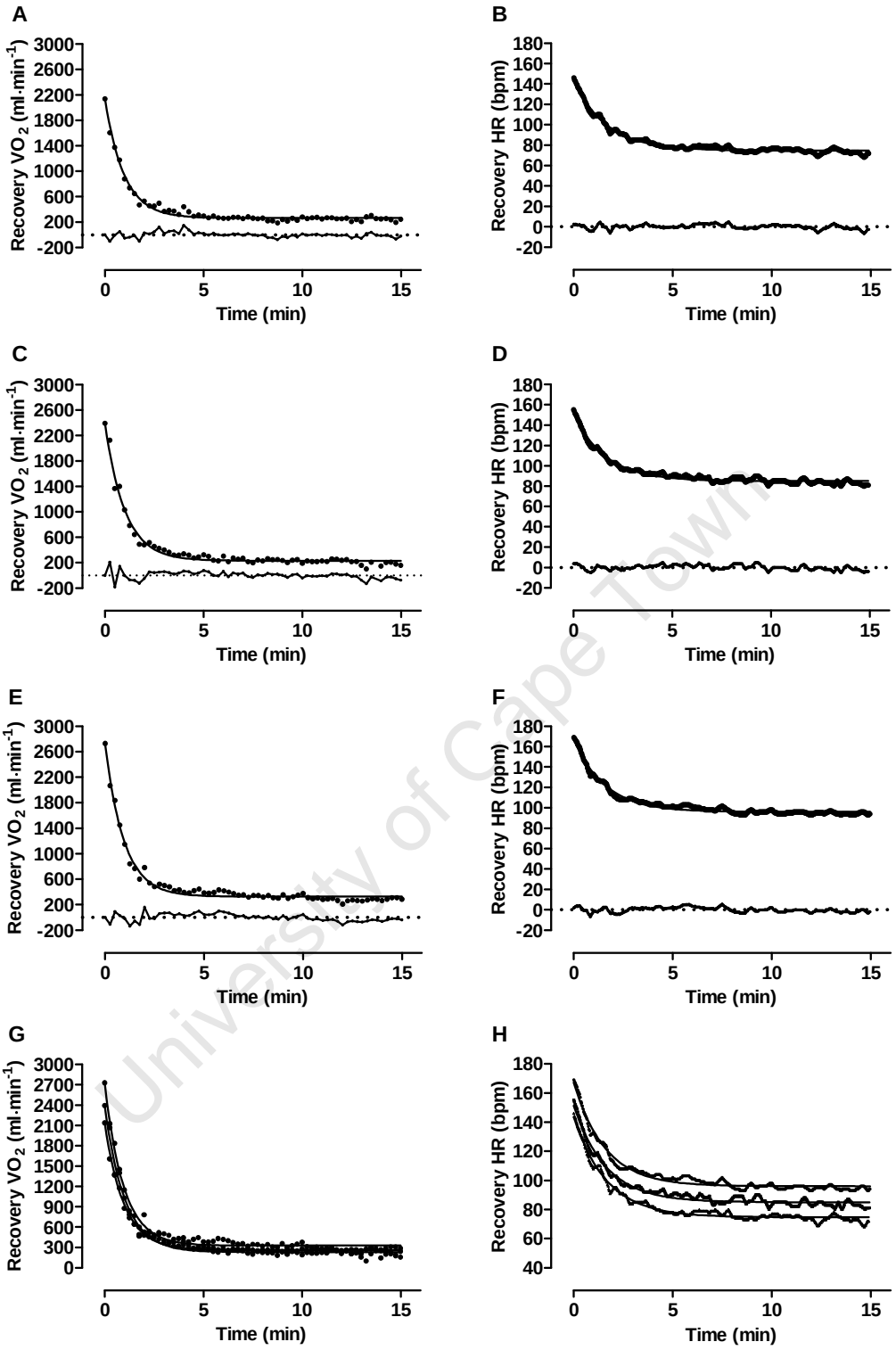


Fig 6.1 Example of one individual's recovery oxygen consumption (VO_2) following 20 min of treadmill exercise at (A) 60% VO_2max, (C) 70% VO_2max, (E) 80% VO_2max and (G) all exercise intensities as well as the same individual's recovery heart rate (HR) responses at (B) 60% VO_2max, (D) 70% VO_2max, (F) 80% VO_2max and (H) all exercise intensities. Data presented include single data points, one phase exponential decay curves and the associated residuals.

6.3.3 Recovery parameters: individual responses

Individual changes in $EPOC_{MAG}$, EPOCτ, HRR_{60s} and HRRτ relative to the day-to-day variation in each of these recovery measurements are shown in Fig 6.2A-D. The proportion of individual responses that showed a decrease, no change or an increase in each recovery measurement is shown in Fig 6.2E-H. For $EPOC_{MAG}$, EPOCτ and HRRτ, an increase in the measurement value indicated an increased time to recover i.e. slower recovery. In contrast, an increase in the value for HRR_{60s} was indicative of more rapid recovery.

For $EPOC_{MAG}$, 53%, 75% and 88% of participants showed a slower recovery when comparing the 70% vs. 60%, 80% vs. 70% and 80% vs. 60% VO_{2max} trials, respectively (Fig 6.2E). A noticeably smaller proportion of individuals showed no change in $EPOC_{MAG}$ following an increase in exercise intensity and only a few individuals showed a meaningful decrease in $EPOC_{MAG}$ response with an increase in exercise intensity (Fig 6.2E).

In contrast, there was greater similarity between the proportion of individuals who showed no change in EPOCτ or HRRτ and the proportion of individuals who showed a meaningful increase in EPOCτ or HRRτ (Fig 6.2F and 6.2H). For example, the proportion of individuals showing no change- or a meaningful increase- in EPOCτ in the 70% vs. 60% VO_2max trials was 38% and 50%, respectively (Fig 6.2F). When comparing HRRτ in the 70% vs. 60% VO_2max trials, the proportion of individuals showing no change- or an increase- in HRRτ was the same (both 38%)(Fig 6.2H).

While the majority of $EPOC_{MAG}$, EPOCτ and HRRτ responses showed slower recovery with increased exercise intensity, 75% and 66% of individuals showed more rapid recovery when comparing HRR_{60s} in the 70% vs. 60% and 80% vs. 60% VO_2max trials, respectively (Fig 6.2G). However, when comparing HRR_{60s} in the 80% vs. 70% VO_2max trials, participant responses were fairly evenly divided between those who showed a decrease (~34%), no change (~34%) or an increase (~32%) in HRR_{60s}.

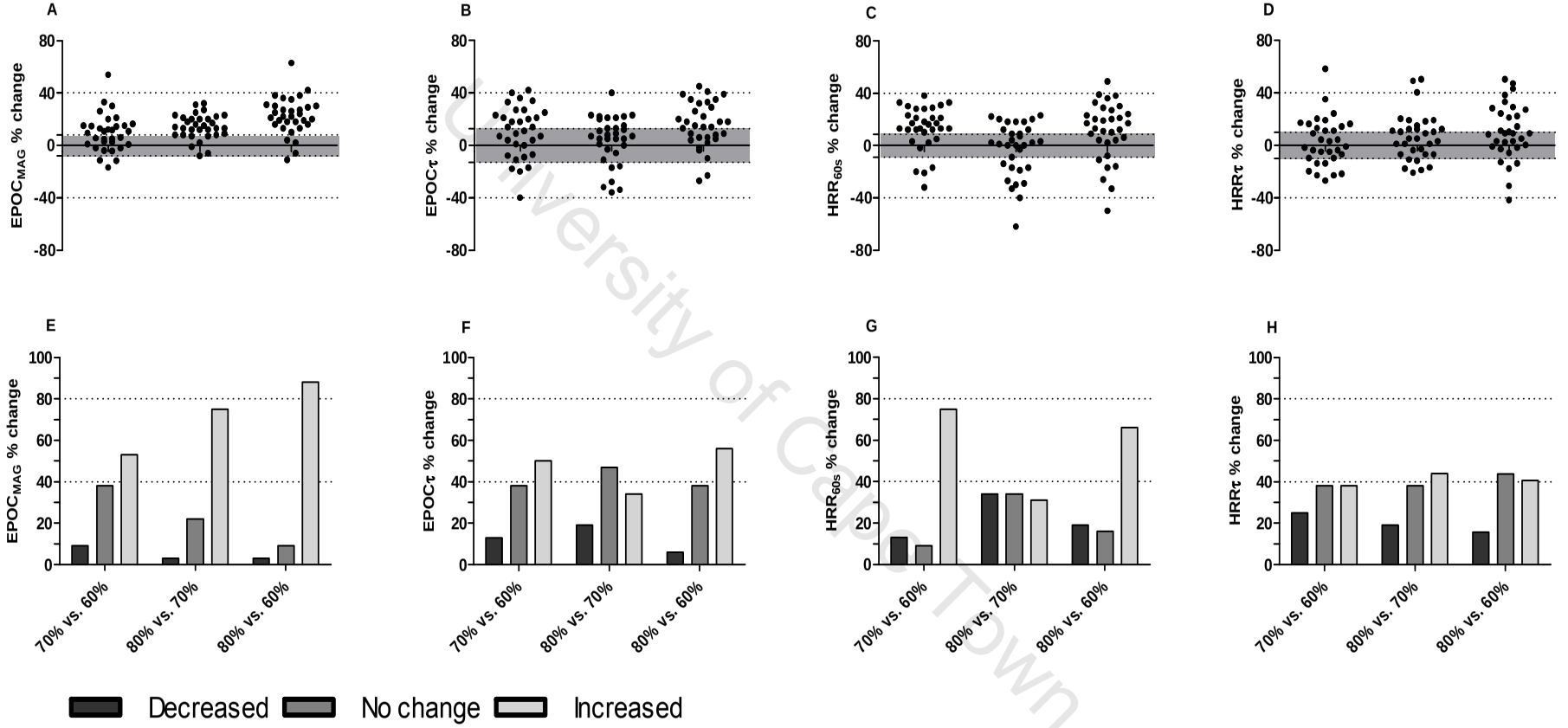


Fig 6.2 Individual responses for % change in (A) $EPOC_{MAG}$, (B) EPOCτ, (C) HRR_{60s} and (D) HRRτ when comparing 70% vs. 60% VO_2max trials, 80% vs. 70% VO_2max trials and 80% vs. 60% VO_2max trials. Proportion of individuals who showed a decrease, no change or an increase in (E) $EPOC_{MAG}$, (F) EPOCτ, (G) HRR_{60s} and (H) HRRτ when comparing 70% vs. 60% VO_2max trials, 80% vs. 70% VO_2max trials and 80% vs. 60% VO_2max trials.

Grey bars indicate % change values that fall within the typical error of the measurement as a coefficient of variation (CV_{TEM}). CV_{TEM} values are ± 8.0% for $EPOC_{MAG}$, ±12.9% for EPOCτ, ±8.7% for HRR_{60s} and ±10.0% for HRRτ

6.4 DISCUSSION

This study showed that exercise at 60%, 70% and 80% of VO_2max was associated with significant differences and, for the most part, large effect sizes for treadmill speed, %HRmax, EE and RPE. Therefore it may be concluded that there were meaningful differences in the homeostatic stress of each exercise bout. However, $EPOC_{MAG}$, EPOCτ, HRR_{60s} and HRRτ showed different patterns of response to these changes in exercise intensity. The main finding of the study was that $EPOC_{MAG}$ was the only recovery measurement to reflect significantly slower recovery with each increase in exercise intensity at the group level and meaningful and consistent changes in the majority of participants at an individual level. While it is well-established that $EPOC_{MAG}$ increases significantly with increases in exercise intensity > 50% VO_2max [20,25], it is uncommon for measurements representing the return to resting homeostasis to be compared within the same study, as was the focus of the current investigation. Our findings suggest that $EPOC_{MAG}$ would be more suitable than EPOCτ, HRR_{60s} or HRRτ to represent intra-individual variation in the homeostatic stress of the preceding exercise bout, as discussed in further detail as follows.

6.4.1 $EPOC_{MAG}$ and EPOCτ

It would be expected that an increase in exercise intensity would increase the homeostatic stress of an exercise bout and result in an increased time to recover towards resting homeostasis. While both $EPOC_{MAG}$ and EPOCτ reflected slower recovery responses with increased exercise intensity, $EPOC_{MAG}$ was significantly different across all 3 exercise intensities whereas EPOCτ was only significantly different when comparing the 70% vs. 60% and 80% vs. 60% VO_2max trials. This suggests that $EPOC_{MAG}$ is more sensitive to changes in exercise intensity than EPOCτ when compared over a short period of recovery and hence shows greater potential to reflect intra-individual variation in the homeostatic stress of an exercise bout.

It is likely that the sensitivity of measures of VO_2 recovery rate (such as EPOCτ) to exercise intensity is influenced by the time period over which recovery is measured. For example, previous studies focusing on the kinetics of VO_2 during the transition from exercise to recovery found no difference in the time constant of VO_2 "off-kinetics" at moderate vs. high exercise intensities [228,229,279] whereas studies that measured EPOC magnitude and duration to the point at which resting metabolism was restored found that both EPOC magnitude and duration showed significant differences with changes in exercise intensity [31,280].

In the present study, the similar EPOCτ responses in the 70% and 80% VO_2max trials could be partially explained by the significant increase in the level at which recovery VO_2 plateaued during the 15 min recovery period following the 80% VO_2max exercise. The higher plateau in recovery VO_2 may be explained by factors such as increased body temperature and increased catecholamine- and metabolite- levels following the 80% VO_2max exercise when compared to the lower exercise intensities [25,35,241]. Had recovery been measured until these changes had been reversed, a lower overall plateau in recovery VO_2 following the 80% VO_2max exercise may have resulted in significantly different EPOCτ values in the 70% and 80% VO_2max trials. Nevertheless, to measure the full time-course of recovery can be too time-consuming to be of practical value (up to several hours) and involves methodological challenges such as obtaining an accurate resting measurement and identifying the point at which recovery measurements can be considered to have regained resting levels [31,281].

Although the absence of a significant difference in EPOCτ between the 70% and 80% VO_{2max} trials may be explained by the duration over which recovery was measured, another factor to consider is the relatively high EPOCτ CV_{TEM} of 12.9%. In other words, day-to-day "noise" in the EPOCτ measurement may have contributed to this measurement being less sensitive to exercise intensity than $EPOC_{MAG}$, a measurement which had lower day-to-day variation (CV_{TEM} = 8.0%). This effect was particularly evident in individual responses. For example, the most common individual response for $EPOC_{MAG}$ when comparing the 70% and 80% VO_2max trials was a meaningful increase (75% of individuals) whereas the most common individual response for EPOCτ for the same change in exercise intensity was no meaningful difference (47% of individuals). Furthermore, even when both $EPOC_{MAG}$ and EPOCτ showed significant differences at the group level (e.g. in the 80% vs. 60% VO_{2max} trials), the proportion of individuals showing a meaningful change in $EPOC_{MAG}$ was higher than that of EPOCτ (88% vs. 56%). Increased sensitivity to changes in exercise intensity at an individual level as well as at a group level confirm that in the current study, $EPOC_{MAG}$ showed more potential than EPOCτ as a possible measure of the homeostatic stress of the preceding exercise bout.

6.4.2 HRR_{60s} and HRRτ

As noted previously, an increase in exercise intensity was expected to result in slower recovery responses in a recovery measurement sensitive to the homeostatic stress of the preceding exercise. While HRRτ showed significantly slower recovery at 80% vs. 70% and 60% VO_2max,

HRR_{60s} responses showed an opposite trend with significantly faster recovery at 70% and 80% vs. 60% VO_2max.

HRR measured within the first minute of recovery is determined primarily by parasympathetic reactivation [29,37] and some previous studies have reported that this form of HRR is delayed at maximal vs. submaximal intensities [29,275] but independent of exercise intensity at moderate levels of submaximal exertion [29]. In contrast, Buchheit *et al.* [231] found significant correlations between HRR_{60s} and blood acidosis and HRR_{60s} and blood lactate concentration and the authors suggested that HRR_{60s} may vary with the contribution of anaerobic metabolism at high exercise intensities. However, this study involved a cross-sectional design and is not clear whether HRR_{60s} would vary with changes in blood acidosis or blood lactate concentration within the same individual.

To the best of our knowledge, the current study is the first to show an increase in HRR_{60s} with an initial increase in exercise intensity followed by a "plateau" in HRR_{60s} following a subsequent increase in exercise intensity. This observation suggests a "threshold" in HRR_{60s} responses whereby exercise intensities in excess of a certain threshold involve a sufficiently large elevation in HR to elicit a "steep" exponential decay towards resting levels at the cessation of exercise. In contrast, lower exercise intensities that involve smaller changes in HR may result in a more "shallow" exponential decay towards resting levels at the termination of exercise. In the current study, this "threshold" appears to occur in the vicinity of 80% HRmax.

The premise of a threshold effect in HRR_{60s} responses is supported by the findings of Perini *et al.* [37]. These authors measured HRR following exercise at 50 W, 100 W and 150 W and reported HRR_{60s} values of 36±7 beats and 35±4 beats following the 100 W and 150 W exercises, respectively. These values are almost identical to those observed in the current study for the 70% and 80% VO_2max trials. Although the authors did not report HRR_{60s} for 50 W, they noted that resting HR was restored within 50 s following the 50 W exercise [37]. With this in mind, the mean end-of-exercise HR and resting HR data provided suggest a slower HRR_{60s} of ~25 beats for the lowest exercise intensity. This creates the same "threshold" pattern of responses in the HRR_{60s} values of Perini *et al.* [37] as was observed in the current study. However, this threshold appeared to occur at a noticeably lower intensity than in the current study given that the 50 W, 100 W and 150 W exercises were associated with 22±3 %VO_2max, 43±2 %VO_2max and 65±2 %VO_2max, respectively [37]. The associated %HRmax values were not provided. This discrepancy in the "threshold" intensity for faster HRR_{60s} responses may be explained by the use of untrained participants in the study by Perini *et al.* and moderately trained participants in the current study.

The "plateau" in HRR_{60s} at submaximal intensities above a certain threshold level is also supported by the findings of Lamberts *et al.* [256]. These authors showed that group mean HRR_{60s} responses remained relatively stable following shuttle runs at somewhat higher- or lower-absolute speeds. For example, one group showed mean HRR_{60s} responses of 58 ±14 bpm and 56±13 bpm following exercise corresponding to 82±2 %HRmax and 88±2 %HRmax, respectively.

On an individual level, 75% of individuals (when comparing the 70% vs. 60% VO_2max trials) and 66% of individuals (when comparing the 80% vs. 60% VO_2max trials) showed an increase in HRR_{60s} that was greater than day-to-day variation. This suggests that the afore-mentioned "threshold" for HRR_{60s} responses might be present in the majority of individuals. In contrast, there was little consistency when comparing the 80% vs. 70% VO_2max trials because the individual HRR_{60s} were approximately evenly divided between those who showed no change, a meaningful increase or a meaningful decrease in HRR_{60s}. These varied responses suggest that, within a certain range of submaximal intensities, meaningful changes in HRR_{60s} are not determined by changes in exercise intensity but rather by other factors e.g. changes in training status [28]. Limited sensitivity of HRR_{60s} to exercise intensity may in fact be an advantage when using HRR_{60s} as a monitoring tool as it suggests that it may not be imperative to elicit precisely the same intensity on each occasion in order to interpret changes in HRR_{60s}. For the purposes of the current investigation, however, our data suggest that HRR_{60s} is likely to be a poor measure of the homeostatic stress of the preceding exercise bout, particularly at submaximal intensities.

In contrast to HRR_{60s}, HRR measurements that extend beyond the first minute of recovery are determined by a combination of continued parasympathetic activation and progressive sympathetic withdrawal [29,37]. Although previous studies have reported this form of HRR at different exercise intensities [29,33,37,254,275], in some cases the associated changes in HRR were beyond the main scope of the study and were not analyzed statistically, making comparison with the current findings difficult [33,37,254]. Nevertheless, Imai *et al.* [29] reported significant increases in HRR measured over the first 2 min of recovery following exercise at 50% of the anaerobic threshold, at the anaerobic threshold and at maximal exertion and Buchheit *et al.* [275] reported a significant increase in $HRR\tau$ following repeated sprint exercise vs. moderate intensity exercise. The current findings were in keeping with these previous studies in that $HRR\tau$ was sensitive to large changes in exercise intensity (60% vs. 80% VO_2max). However, the current study showed that this measurement was not sensitive to smaller changes in exercise intensity. As was the case with $EPOC\tau$, similar $HRR\tau$ values with changes in exercise intensity may be partially

explained by higher recovery curve plateau values with increases in exercise intensity when modelled over a 15 min recovery period. Nevertheless, the results of our own reliability analyses as well as other studies investigating the reliability of HRRτ suggest that this measurement shows relatively high variability [254,263] and even HRRτ modelled over a longer period of recovery may show limited sensitivity to changes in exercise intensity. The limited sensitivity of HRRτ to detect changes in exercise intensity was also apparent on an individual level given that, across all trials, the proportion of individuals who showed no change in HRRτ with increased exercise intensity was similar to the proportion of individuals who showed an increase in HRRτ with an increase in exercise intensity. These observations would largely preclude the practical application of HRRτ to representing changes in exercise intensity.

6.4.3 Limitations

A limitation of the current study is that the aerobic and anaerobic thresholds were not determined. It would have been of interest to examine whether there were threshold-related differences between those individuals who showed a meaningful change in a recovery measurement with an increase in exercise intensity and those that did not. A second limitation of the study was that CV_{TEM} values based on the 70% VO_2max trial were used to interpret individual responses at all 3 exercise intensities. It would have been preferable to use intensity-specific CV_{TEM} values when interpreting individual responses to the 60% and 80% VO_2max trials.

6.5 CONCLUSION

This study adds to only a small number of previous studies that have compared the relative sensitivity of different recovery measurements to changes in exercise intensity [15,16]. When comparing the novel combination of $EPOC_{MAG}$, EPOCτ, HRR_{60s} and HRRτ responses at 3 different exercise intensities, it was clear that HRR_{60s} was least suitable as a measure of the homeostatic stress of the preceding exercise. This measurement showed faster recovery- or no change in recovery with increases in exercise intensity and, based on the existing literature, appears to be more suited to predicting mortality [26,27] or monitoring changes in training status [28]. In fact, limited sensitivity of HRR_{60s} to small changes in submaximal exercise intensity may be advantageous when using this measurement as a monitoring tool.

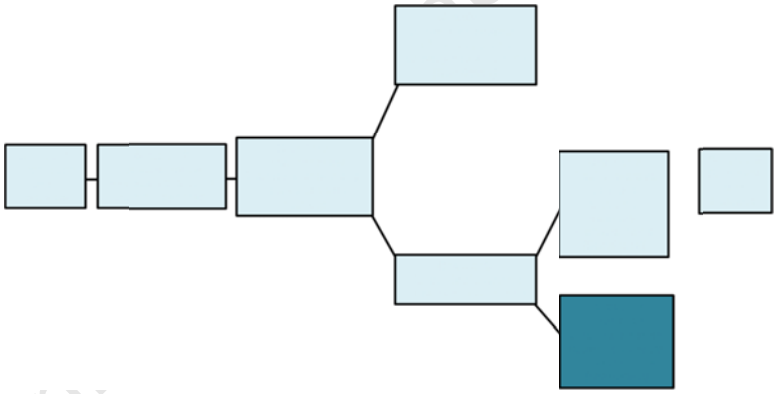
In contrast, $EPOC_{MAG}$, EPOCτ and HRRτ reflected slower recovery with increased exercise intensity, albeit with different levels of sensitivity. EPOCτ and HRRτ measured over 15 min of

recovery showed relatively high measurement "noise" and did not always detect differences between exercise intensities at the group level. Furthermore, individual changes in EPOCτ and HRRτ with changes in exercise intensity were not always detectable above day-to-day variation in the measurement or in a consistent direction of change. This suggests that these measurements would have limited practical value for interpreting intra-individual variation in the homeostatic stress of an exercise bout.

In comparison to HRR_{60s}, EPOCτ and HRRτ, $EPOC_{MAG}$ was clearly the most suitable as a potential measure of intra-individual changes in homeostatic stress, showing significant differences across all 3 exercise intensities and meaningful and consistent changes in the majority of individuals. Unfortunately, the day-to-day use of $EPOC_{MAG}$ to help interpret the homeostatic stress of training sessions is precluded by the need for gas analysis equipment. Nevertheless, this application of $EPOC_{MAG}$ may add valuable insight in certain laboratory-based training sessions or research studies.

CHAPTER 7

EFFECT OF AN ULTRA-ENDURANCE ROAD RACE ON POST-EXERCISE OXYGEN CONSUMPTION AND HEART RATE RECOVERY



7.1 INTRODUCTION

Adaptation to exercise training occurs in response to the stimulus of an increased training load. However, high training loads must be accompanied by appropriate recovery periods if an individual is to avoid becoming overreached and, in time, overtrained [82,282,283]. The deleterious effects of overtraining on performance, psychological and physiological parameters have been well-described in the literature [282–286] and have prompted ongoing interest into measurements that could be used to detect fatigue or overreaching before progression into overtraining has occurred [81,90,287–291]. Ideally, measurements of this kind would not only provide an indication of when to reduce training load but also provide an indication of when an individual was adapting well and could tolerate an increase in training load. In other words, a sensitive measurement that was objective, easy to administer and inexpensive could potentially serve as a valuable tool for athlete monitoring- fine-tuning day-to-day training prescription and avoiding unanticipated decrements in performance.

As yet, no single measurement can be relied upon to reflect an individual's current response to training and/or "readiness" to adapt to further training [292]. However, one measurement that shows potential in this role is heart rate recovery (HRR) [28]. Longitudinal studies have demonstrated good relationships between changes in HRR and changes in performance measured before and after a standardized training program of 4 or 8-9 weeks [36,41,73,81] and a recent systematic review concluded that faster HRR was indicative of an improvement in training status and slower HRR was related to a decrease in training status [28]. In contrast, the relationship between HRR and "readiness to train" measured at more frequent intervals does not appear to be as well-established. For example, Dupuy *et al.* reported faster HRR responses after 2 weeks of overload training even though "readiness to train" would be expected to have decreased [42]. In a similar way, Lamberts *et al.* reported faster HRR despite an increased RPE following weeks of high training load in a case study of a an elite cyclo-cross athlete [293]. In contrast, Borresen and Lambert found that 1 week of increased training load was associated with slower HRR responses [292]. It is likely that differences in study design including the training status of the participants, the relative load of the training, the characteristics of the exercise bout preceding the HRR measurement, the time period between HRR measurements and the form of HRR may to a large extent explain these contrasting findings. Nevertheless, it would seem that HRR responses following acute increases in training load may require further investigation before short-term changes in HRR can be interpreted with confidence.

Another aspect of HRR that is currently unclear is to what extent changes in HRR are associated with changes in metabolic recovery measurements such as the magnitude of excess post-exercise oxygen consumption ($EPOC_{MAG}$) or oxygen consumption (VO_2) recovery rate. Longitudinal studies have shown smaller $EPOC_{MAG}$ values [35] or faster VO_2 recovery rates [33] at the same absolute intensity after training. However, we are not aware of any studies that have related longitudinal changes in $EPOC_{MAG}$ or VO_2 recovery rate to changes in performance or monitored these measurements over the course of acute changes in training load. There are, however, 2 studies that have reported increased $EPOC_{MAG}$ responses following a single bout of strenuous exercise [294,295], suggesting a possible link between metabolic recovery and fatigue or readiness to train. In both of these studies, change in $EPOC_{MAG}$ was associated with a change in VO_2 during the exercise bout and this may imply that changes in $EPOC_{MAG}$ are related to changes in the homeostatic disturbance of the preceding exercise bout. In contrast, it would seem that changes in HRR are not necessarily linked to changes in the homeostatic disturbance of the preceding exercise as significant changes in HRR can occur without any change in exercise HR [36,292].

To the best of our knowledge, the relative sensitivity of HRR and metabolic recovery to changes in training load has not previously been investigated. We speculated that an ultra endurance event would represent an acute training overload and serve as an experimental model that would help to clarify the acute effect of increased training load on HRR and provide insight into the possible potential of VO_2 recovery as a monitoring tool. Therefore, the aim of the current study was to compare HRR and VO_2 recovery responses before and (3 days) after the Comrades ultra-marathon, an 87 km road race between Durban and Pietermaritzburg in South Africa. HRR was defined as the heart rate recovery 1 min post-exercise (HRR_{60s}) and the time constant of the HR recovery curve whereas VO_2 recovery responses were defined as $EPOC_{MAG}$ and the time constant of the VO_2 recovery curve ($EPOC\tau$).

The start and finish of the Comrades race are alternated annually and in 2011 the race was an “up-run”, starting at sea level in Durban and finishing in Pietermaritzburg at an elevation of 650 m. The Comrades ultra-marathon is a highly strenuous event and post-race vs. control measurements have shown significant disturbances in metabolic, hematological and hormonal parameters along with significant muscle damage [296–300]. Nevertheless, Burgess found no difference in steady state oxygen consumption (VO_2), respiratory exchange ratio (RER), heart rate (HR) or blood lactate measurements between race participants and controls when compared 4 days after a Comrades ultra-marathon “up run” [301]. As previous studies have shown that

changes in EPOC are linked to changes in exercise VO_2 [294,295], the first hypothesis of the current study was that there would be no significant difference in the VO_2 or $EPOC_{MAG}$ of a submaximal exercise bout performed before the race and 3 days after the race. Nevertheless, it was anticipated that the "readiness to train" of Comrades participants would remain altered and that they would be in a poor state to adapt to training so soon after an ultra-endurance, overload event. Therefore, the second hypothesis of the current study was that HRR would be significantly slower 3 days after the race compared to pre-race measurements

7.2 METHODS

7.2.1 Participants and study design

Ten men and 3 women who were registered to take part in the 2011 Comrades ultra-marathon "up-run" (87 km) volunteered to participate in this prospective cohort study. The study was approved by the Human Research Ethics Committee of the University of Cape Town and all participants signed an informed consent document after receiving a full explanation of the laboratory tests involved. Furthermore, all participants were able to answer "no" to all the questions in a Physical Activity Readiness Questionnaire (PAR-Q) [217]. Although all 13 individuals completed the pre-race laboratory testing as well as the Comrades ultra-marathon "up-run", one participant was subsequently diagnosed with a severe viral infection and 2 other participants chose to withdraw from the study as they were no longer willing to complete post-race testing. As a result, the final participant group consisted of 10 runners, including 8 men and 2 women.

Participants were asked to visit the laboratory on 3 occasions as follows: ± 14 days before the race, ± 7 days before the race and 3 days after the race. During the first visit, participants completed a short, 3 month training history questionnaire including the typical number of training sessions per week, typical total training distance per week and a recent marathon time. This was followed by anthropometric measurements and a maximal incremental treadmill test. The second and third laboratory visits were identical and included muscle pain ratings, a 5 bound test and 20 min of treadmill exercise at 70% of maximal oxygen consumption (VO_2max) followed by 15 min of controlled recovery. An overview of the laboratory visits is shown in Figure 7.1.

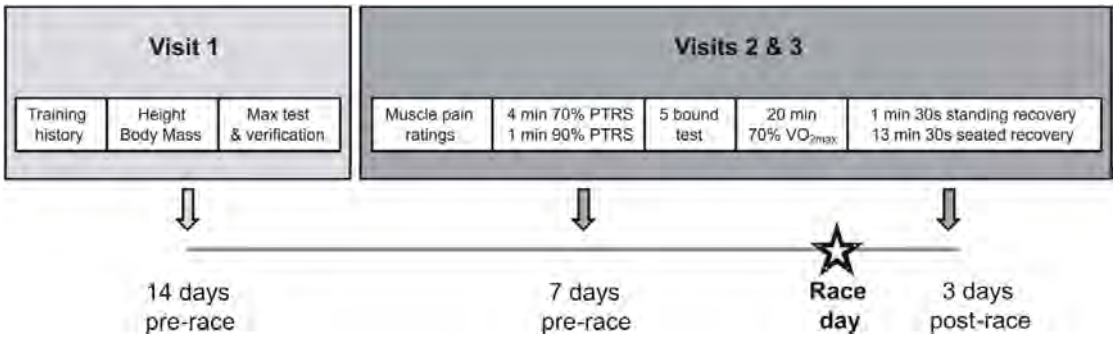


Fig 7.1 Overview of laboratory visits.

7.2.2 Visit 1: Anthropometry and maximal incremental test

7.2.2.1 Anthropometric measurements

Height and body mass were determined using a stadiometer (Seca model 708, Germany) and calibrated scale (Seca model 708, Germany), respectively. Body mass was re-measured on each subsequent visit to the laboratory.

7.2.2.2 Maximal incremental test and verification bout

Participants completed a self-paced warm-up on the treadmill followed by a maximal incremental treadmill test to determine VO_2max, maximal heart rate (HRmax) and peak treadmill running speed (PTRS). The test started with participants running at 10 km·h^{-1} for 1 min after which the treadmill speed was increased by 0.5 km·h^{-1} every 30 s until volitional exhaustion [258,278]. Participants were verbally encouraged to produce a maximal effort. After an 8-10 min rest period, participants ran to exhaustion at one stage higher than the highest stage completed in the incremental test. The purpose of this "verification" run was to ensure that a "true" VO_{2max} had been obtained [163].

Respiratory gases were measured breath-by-breath during both the incremental test and the verification bout (Jaeger Oxycon Pro®, Hoechberg, Germany) and averaged over 15 s intervals [220]. The highest 15 s average VO_2 from the incremental test and the verification bout were compared and VO_{2max} was taken as the higher of these 2 values. Nevertheless, these differences were generally small (within 4±4 %) and consistent with the differences reported elsewhere [166]. Beat-by-beat heart rate data were also collected (Suunto Oy®, Vantaa, Finland) and HR_{max} was defined as the highest 2 s average during the incremental test. PTRS was defined as the highest completed stage during the incremental test.

7.2.3 Visits 2 and 3

7.2.3.1 Perceived muscle pain, 5 bound test and the submaximal treadmill exercise

Participants were asked to refrain from hard training the day before visits 2 and 3 and not to exercise prior to the laboratory visit on the day of the trial (though these conditions applied primarily to visit 2). Furthermore, participants were asked to abstain from all food and drink other than water for at least 2 hours prior to visits 2 and 3. The compliance with these requests was verbally confirmed with each participant upon their arrival at the laboratory. Laboratory visits took place at the same time of day (within 2 hours) for a particular participant to avoid variation as result of circadian rhythm [259].

At the start of visits 2 and 3, participants were asked to rate their perceived muscle pain during light stretching by placing a mark on a 10 cm visual analog scale, ranging from "no pain" to "unbearable pain". Participants provided separate ratings for the quadriceps, hamstrings and calf muscles.

Participants then completed a standardized warm-up consisting of 4 min at 70% of PTRS and 1 min at 90% PTRS. This was followed a 10 min break during which participants performed the 5 bound test on an indoor, synthetic surface. The objective of the 5 bound test was to cover the furthest possible horizontal distance in 5 alternating strides from a standing start [302]. In the current study, post vs. pre-race change in the distance covered over 5 bounds was used as a proxy for change in neuromuscular power [302]. Participants were allowed several practice attempts at the 5 bound test followed by 3 official attempts. Horizontal distance covered was measured from the start line to the point of heel strike after the 5th stride and 5 bound distance was taken as the best of the 3 official attempts.

The 5 bound test was followed by a 20 min treadmill exercise at 70% VO_{2max} and 15 min of controlled recovery. Breath-by-breath respiratory data (Jaeger Oxycon Pro®, Hoechberg, Germany) and HR (Suunto Oy®, Vantaa, Finland) were recorded continuously throughout the exercise and recovery period. For the visit 2, the treadmill speed to elicit 70% VO_{2max} was estimated based on the relationship between VO_2 and running speed during the incremental treadmill test in Visit 1. If necessary, small adjustments to the treadmill speed were made within the first minutes of exercise to ensure that the target intensity was attained. These adjustments were based on an exercise VO_2 vs. target VO_2 discrepancy of > 2 $ml \cdot kg^{-1} \cdot min^{-1}$. For visit 3, treadmill speed was matched to the speed that had elicited 70% VO_{2max} steady state in the pre-race trial. With 1 min of the 20 min treadmill exercise remaining, participants were asked to

provide a rating of perceived exertion (RPE) on Borg's 6-20 scale [224]. This scale was thoroughly explained to each participant at the start of the laboratory visit.

At the end of the 20 min exercise bout, participants were given a countdown to take hold of the treadmill handrails and step to the side of the moving treadmill belt. The treadmill belt was stopped immediately and participants stepped on to the stationary belt and stood upright for the first 1 min 30 s of recovery. After 1 min 30 s had elapsed, participants were asked to be seated on a chair placed directly behind them on the treadmill belt and completed a further 13 min 30 s of seated recovery. The combination of short period of standing recovery followed by seated recovery is similar to an approach that has been used elsewhere [263]. Participants were asked to refrain from speaking and to remain as still as possible during the recovery measurements.

7.2.4 Data analysis

Breath-by-breath respiratory data were analyzed as 15 s averages and beat-to-beat HR data as 2 s averages. Steady state exercise VO_2, minute ventilation (VE), RER, breathing frequency (BF) and HR were taken as the average of the final 17 min of the 20 min exercise bout. Energy expenditure (EE) for the 20 min exercise bout was calculated based on the standard caloric equivalents for oxygen at different RER values [225].

Recovery VO_2 data was modeled as a one phase exponential decay curve with the starting point of the curve made to equal the average VO_2 over the last 3 min of exercise (GraphPad Software, SanDiego, California, USA). It has previously been shown that recovery VO_2 kinetics are adequately characterized by a mono-exponential function following steady-state exercise at "moderate" and "heavy" exercise intensities [228,229]. EPOC$_T$ was defined as the time constant of the recovery curve and VO_2 plateau as the plateau value of the recovery curve, in keeping with the approach of Campos *et al.* [34]. The span of the recovery curve, referring to the change in VO_2 from the start or peak of the curve to the plateau of the curve, was also recorded. EPOC$_{MAG}$ was taken as the total area under the recovery curve, rather than adjusting the area under the curve for baseline VO_2. This approach avoided the methodological challenges of obtaining a meaningful resting VO_2 measurement [173] and has a lower intra-individual variation than methods that incorporate a resting measurement [30]. To investigate changes in post-exercise substrate oxidation, average RER over the 15 min recovery period was also reported.

To calculate HRR$_{60s}$, the average of the last 16 s of the first minute of recovery was subtracted from the average of the last 16 s of the last minute of exercise, as described previously [226,256]. In addition, HR data over the recovery period was modeled as a one phase exponential decay with

HRRτ taken as the time constant of the curve and HR plateau taken as the plateau value of the curve (GraphPad Software, SanDiego, California, USA)[37]. The span of the HR recovery curve has also recorded.

Lastly, each participant's finishing time for the race was used to calculate average speed over the 87 km course. Average race pace was reported as a % of PTRS.

7.2.5 Statistical analysis

Muscle pain ratings included a number of zero values in the pre-race measurements and were not normally distributed based on a D'Agnostino and Pearson Omnibus Normality test (GraphPad Software, SanDiego, California, USA). As a result, muscle pain ratings are reported as median and range and post vs. pre-race changes in muscle pain were assessed using a Wilcoxon t-test for paired observations that are not necessarily normally distributed (GraphPad Software, SanDiego, California, USA). In contrast, 5 bound distance and all submaximal exercise and recovery parameters were normally distributed according to a D'Agnostino and Pearson Omnibus Normality test and are reported as mean ± standard deviation (SD). Post vs. pre-race changes in these measurements were assessed using paired t-tests and Pearson's correlation coefficients for changes in exercise measurements vs. changes in recovery measurements and for PTRS vs. race time were calculated (GraphPad Software, SanDiego, California, USA). Statistical significance was accepted at $p < 0.05$.

Post vs. pre-race changes in 5 bound distance, exercise and recovery measurements were also assessed using Cohen's effect sizes [250]. Effect sizes were calculated as the difference in the post vs. pre-race group means divided by the pooled standard deviation of the pre- and post-race measurements and were described as trivial = <0.2, small = ≥0.2 to < 0.5, moderate = ≥0.5 to < 0.8 or large = ≥ 0.8.

Finally, post vs. pre-race measurement changes in individual participants were compared to the day-to-day variation of each measurements as calculated in Chapter 5. Taken as the typical error of measurement as a coefficient of variation (CV_{TEM}), day-to-day variation for each exercise and recovery measurement was defined as follows: VO_2 CV_{TEM} = 1.8%, VE CV_{TEM} = 3.3.%, RER CV_{TEM} = 2.1%, EE CV_{TEM} = 2.3%, HR CV_{TEM} = 2.2%, RPE CV_{TEM} = 7.3%, $EPOC_{MAG}$ CV_{TEM} = 8.0%, EPOCτ CV_{TEM} = 12.9%, VO_2 plateau CV_{TEM} = 12.7%, HRR_{60s} CV_{TEM} = 8.7%, HRRτ CV_{TEM} = 10.0% and HR plateau CV_{TEM} = 3.0%. A post vs. pre-race change that exceeded the CV_{TEM} of the measurement was regarded as a meaningful increase or decrease for that individual, depending on the direction of change. Conversely, a post vs. pre-race change that did not

exceed the CV_{TEM} of the measurement was not considered meaningful. Individual responses were rank ordered for each measurement to show the proportion of individuals who had a decrease larger than the day-to-day variation of the measurement, a change that fell within the day-to-day variation of the measurement or an increase which exceeded the day-to-day variation of the measurement.

We did not establish the day-to-day variation of perceived muscle soreness in our previous study. However, Burgess and Lambert showed that differences in perceived muscle pain of ~3 units are significantly different at the group level [300]. With this in mind, we elected to compare individual changes in perceived muscle soreness against a “threshold” change of 3 units.

7.3 RESULTS

7.3.1 Participants

The characteristics of the study participants are shown in Table 7.1. All participants completed the 87 km race within the 12 h cut-off with finishing times of between 8 h 11 min and 11 h 39 min. There was a strong correlation between PTRS and race finishing time (r = -0.89, p = 0.0005) and average speed for the race corresponded to 47-56% of each individual's PTRS. Although we had hoped to schedule the post-race trial 3 days after the race, in practice it was necessary to conduct these trials 2 days (2 participants), 3 days (4 participants) or 4 days (4 participants) after the race due to the logistical constraints of testing.

Table 7.1 Participant characteristics. Data expressed as mean ± SD (range).

Variable	Men (n = 8)	Women (n = 2)	Total (n = 10)
Age (years)	36 ± 4 (31–41)	32 ± 15 (21–42)	35 ± 6 (21–42)
Body Mass (kg)	78 ± 7 (70–92)	63 ± 4 (60–66)	75 ± 9 (60–92)
Height (cm)	180 ± 6 (170–190)	159 ± 11 (151–166)	175 ± 11 (151–190)
BMI ($kg \cdot m^2$)	24 ± 2 (22–28)	25 ± 2 (24–26)	24 ± 2 (22–28)
Training frequency ($runs \cdot wk^{-1}$)*	5 ± 1 (3–6)	6 ± 1 (5–6)	5 ± 1 (3–6)
Total training distance ($km \cdot wk^{-1}$)*	71 ± 19 (35–90)	78 ± 4 (75–80)	72 ± 16 (35–90)
Self-reported 42.2 km time (h:min)*	3:31 ± 0:11 (3:20–3:50)	4:20 ± 0:20 (4:06–4:35)	3:42 ± 0:25 (3:20–4:35)
VO_2max ($ml \cdot kg^{-1} \cdot min^{-1}$)	57.9 ± 5.1 (51.4–63.0)	49.9 ± 5.0 (46.3–53.40	56.3 ± 5.7 (46.3–63.0)
PTRS ($km \cdot h^{-1}$)	18.7 ± 1.3 (17.0–21.0)	14.5 ± 0.0 (14.5–14.5)	17.6 ± 2.1 (14.5–21.0)

*n = 9, one participant did not provide the requested training information.

7.3.2 Muscle pain and muscle power

There was a significant increase in perceived pain in the quadriceps muscles (p < 0.05) and hamstring muscles (p < 0.01) after the race with a non-significant increase in perceived pain in

the calf muscles (Table 7.2). However, the typical overall magnitude of perceived muscle pain 3-4 days after the race was relatively low at ratings of 2 to 3 out of a possible 10. There was no difference in 5 bound distance measured before and after the race (Table 7.3).

Table 7.2 Perceived muscle pain

Muscle pain	Pre-race		Post-race	
	Median	Range	Median	Range
Quadriceps pain (0-10)	0.0	0.0-3.1	3.0**	0.0-7.2
Hamstring pain (0-10)	0.4	0.0-1.8	2.1*	0.0-6.4
Calf pain (0-10)	0.7	0.0-6.2	2.7	0.0-8.3

Significantly different to pre-race at **p<0.01 or *p<0.05

7.3.3 Submaximal exercise parameters

There were no significant differences in exercise VO_2, VE, EE, RER or HR during the submaximal exercise bout before and after the race and post vs. pre-race changes showed only *small*-to-*trivial* effect sizes (d = 0.0 to 0.3) (Table 7.3). In contrast, there was a *large*, significant increase in the RPE from 11±1 in the pre-race trial to 13±2 in the post-race trial (d = 1.2)(p < 0.01).

7.3.4 VO_2 and HR recovery parameters

There was no significant difference in $EPOC_{MAG}$, EPOCτ, the plateau of the VO_2 recovery curve or the average RER over the recovery period in the post vs. pre-race trials (Table 7.3). Nevertheless, changes in $EPOC_{MAG}$ and VO_2 plateau were associated with *moderate* effect sizes (d = 0.5) and there was a *small*, significant increase in the span of the VO_2 recovery curve (p < 0.01). HRR_{60s} and HRRτ reflected significantly faster recovery in the post-race trial compared to the pre-race trial with *large* associated effect sizes (d = 0.9 to 1.0)(p < 0.05). There were, however, no significant changes in the span or plateau of the HR recovery curve.

Table 7.3 Exercise and Recovery parameters

Measurement type	Parameter	Pre-race	Post-race	Pre vs. post	
				Effect size	Descriptor
Neuromuscular power	5 jump distance (m)	9.88 ± 1.60	9.73 ± 1.53	0.1	*Trivial*
Submaximal exercise response	VO_2 (ml·kg^{-1}·min^{-1})	39.7 ± 4.1	40.5 ± 4.2	0.2	*Small*
	%VO_2max (%)	70.6 ± 1.4	72 ± 2.9	0.2	*Small*
	VE (l·min^{-1})	73.5 ± 12.4	76.3 ± 12.8	0.2	*Small*
	BF (breath·min^{-1})	38 ± 6	38 ± 6	0.0	*Trivial*
	RER	0.87 ± 0.04	0.86 ± 0.03	0.3	*Small*
	EE (kcal)	285 ± 57	286 ± 52	0.0	*Trivial*
	HR (bpm)	152 ± 11	148 ± 12	0.3	*Small*
	%HRmax (%)	83 ± 4	82 ± 5	0.5	*Moderate*
	RPE (6-20)	11 ± 1	13 ± 2**	1.2	*Large*
VO_2 recovery parameters	$EPOC_{MAG}$ (ml·kg^{-1})	90 ± 15	82 ± 17	0.5	*Moderate*
	EPOCτ (s)	47 ± 6	48 ± 8	0.1	*Trivial*
	VO_2 span (ml·kg^{-1})	35.4 ± 3.1	36.9 ± 3.5**	0.4	*Small*
	VO_2 plateau (ml·kg^{-1})	4.2 ± 1.0	3.6 ± 1.2	0.5	*Moderate*
	RER	0.88 ± 0.06	0.88 ± 0.06	0.0	*Trivial*
HR recovery parameters	HRR_{60s} (beats)	29 ± 4	35 ± 5**	1.0	*Large*
	HRRτ (s)†	106 ± 15	90 ± 16*	0.9	*Large*
	HR span(bpm)	71 ± 11	67 ± 12	0.3	*Small*
	HR plateau (bpm)	84 ± 9	82 ± 15	0.2	*Small*

Values are mean ±SD. †n = 9. Significantly different to pre-race at **p<0.01 or *p<0.05

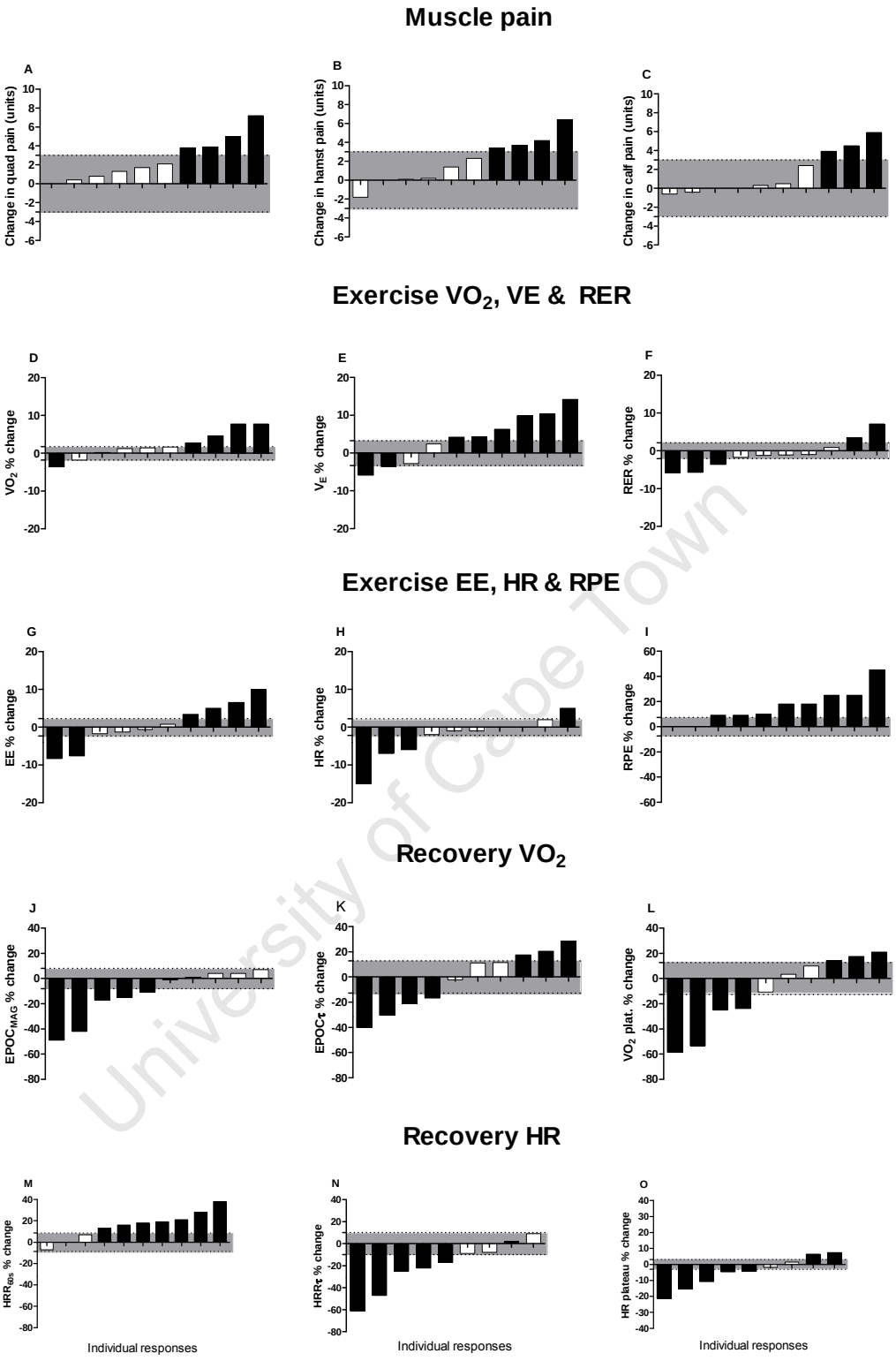


Fig 7.2 Post-race vs. pre-race change in quadriceps pain (A), hamstring pain (B), calf pain (C), VO_2 (D), VE (E), RER (F), EE (G), HR (H), RPE (I), $EPOC_{MAG}$ (J), EPOCτ (K), VO_2 recovery curve plateau (L), HRR_{60s} (M), HRRτ (N) and HR recovery curve plateau (O) for individual participants, ranked by magnitude of change.

Horizontal grey bars indicate day-to-day variation in each measurement. Black columns indicate individual participant responses that exceeded the day-to-day of the measurement and white columns indicate individual participant responses that did not exceed the day-to-day variation of the measurement. n = 9 for HRRτ and HR recovery curve plateau.

7.3.5 Individual responses

Individual responses for post vs. pre race changes in muscle pain, exercise and recovery parameters are shown in Fig 7.2. Exercise and recovery parameters that showed significant post- vs. pre-race changes at the group level generally showed the same direction of change in the majority of individuals. For example, 8 of the 10 participants had a meaningful increase in RPE, with the remaining 2 participants showing no change in RPE (Fig 7.2I). Seven of the 10 participants had meaningfully faster HRR_{60s} responses (Fig 7.2M) and 5 of the 10 participants showed meaningfully faster $HRR\tau$ responses (Fig 7.2N). Participants who did not show faster HRR_{60s} and $HRR\tau$ after the race showed no meaningful change in these responses.

Individual responses were also compared in the form of a "profile" of post vs. pre-race changes for each participant (Fig 7.3). Although there was large inter-individual variation in the pattern and magnitude of responses, we made a few general observations as follows. Firstly, the relative change in the plateau of the VO_2 recovery curve was of a similar magnitude and direction to the relative change in $EPOC_{MAG}$ for all participants except perhaps participant 7 (Fig 7.3G). Secondly, the relative change in RPE did not appear to be related to the relative change in recovery measurements. For example, both participants 4 and 5 showed no change in RPE but a noticeable change in $EPOC_{MAG}$ and HRR_{60s}, respectively (Fig 7.3D, 7.3F). Finally, there did not appear to be any relationship between the pattern and magnitude of post vs. pre-race changes and the finishing time of the participant. Correlations between post vs. pre-race changes in exercise measurements and post vs. pre-race changes in recovery measurements showed no significant relationships (data not shown).

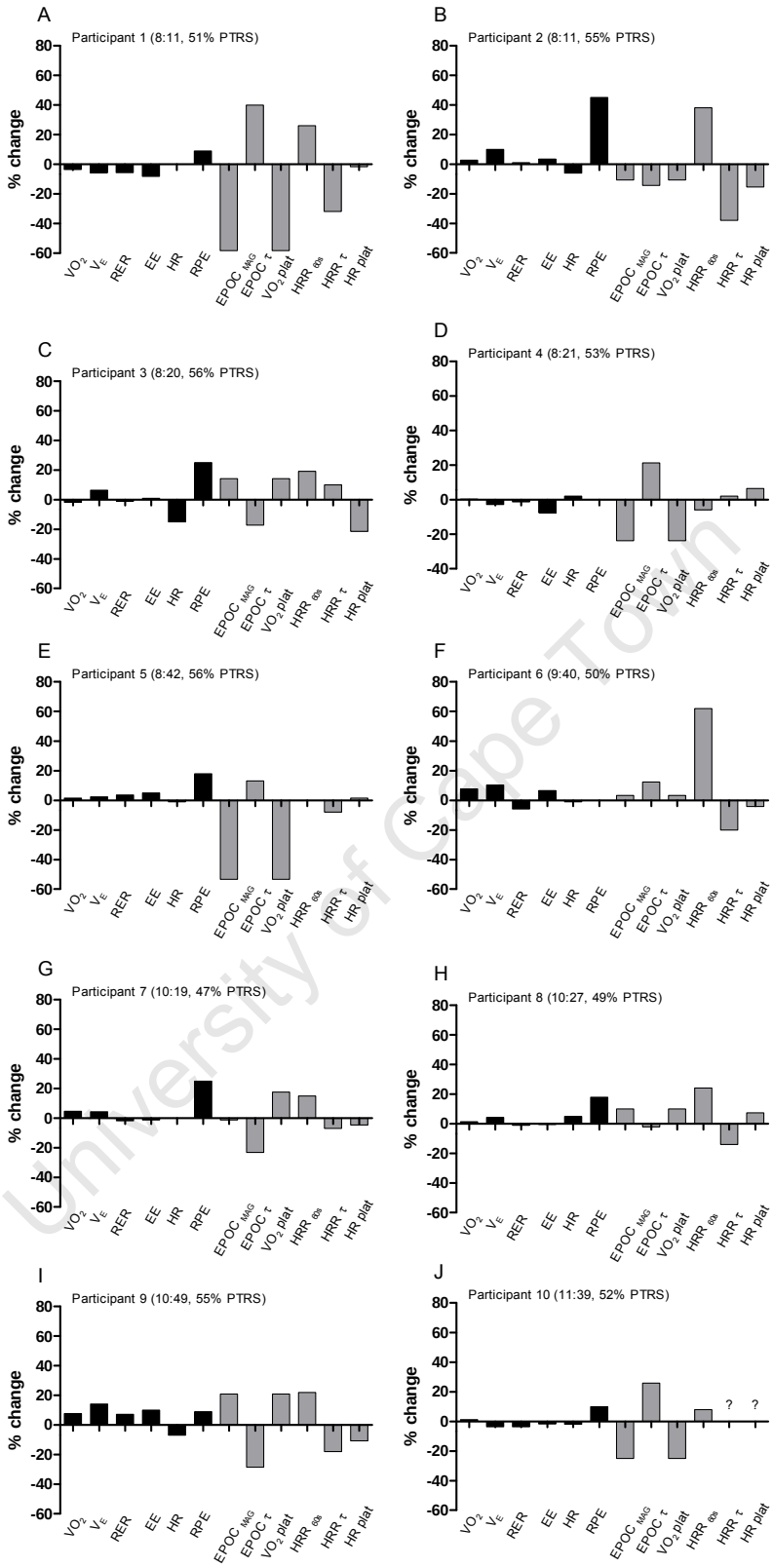


Fig 7.3A-J Profile of post vs. pre-race changes in exercise and recovery measurements for participants 1 to 10. Finishing time for the 87 km Comrades ultra-marathon is shown in brackets as hours:min and average race pace is shown as a percentage of Peak Treadmill Running Speed (PTRS)

Black bars indicate exercise measurements and grey bars indicate recovery measurements. ? indicates lost data.

7.4 DISCUSSION

By self-report, participants had accumulated a typical weekly training distance of 72±16 km, completed over 5±1 training sessions per week, in the 3 months leading up to the race. Although an objective measure of typical training load would have been preferable, it is reasonable to assume that covering the 87 km race distance in a single session would have been a considerable training "overload" for all of the participants. Furthermore, the strong correlation between PTRS and race time would suggest that participants attempted to complete the course in the fastest possible time. The first finding of this study was the HRR_{60s} and $HRR\tau$ reflected significantly faster recovery ~3 days after an ultra-marathon when compared to pre-race measurements, an observation that was contrary to the slower HRR responses we had hypothesized. However, the second finding of the study was that $EPOC_{MAG}$ and $EPOC\tau$ measured ~3 days after the ultra-marathon were not significantly different to pre-race measurements, an observation that was in keeping with what we had hypothesized. Possible mechanisms for these observations are discussed as follows.

7.4.1 HRR_{60s} and $HRR\tau$

Previous studies have reported both faster [42,293] and slower [292] HRR responses following 1-2 weeks of increased training load and the relationship between short-term changes in HRR and "readiness to train" is not yet well-established. It has previously been suggested that slower HRR responses may predict decreased endurance capacity and the accumulation of fatigue [81] and we had expected that participants in the current study would be somewhat fatigued and show slower HRR responses in the post-race trial. Although the large, significant increase in RPE could be interpreted as the anticipated increase in fatigue, HRR responses were faster in the post-race trial- a change typically interpreted as an improvement in training status [28] or what we have described as "readiness to train".

One explanation for faster HRR responses could be decreased sympathetic activity in the days after the race. For example, Sagnol *et al.* monitored catecholamine levels in the days after a 10 h triathlon and reported that plasma free adrenalin levels were below pre-race levels after 3 days of recovery [303]. The authors speculated that this may reflect depleted adrenalin reserves or a suppressed response from the adrenal medulla after high levels of catecholamine release during the triathlon itself. In contrast, Fry *et al.* reported a non-significant increase in epinephrine excretion after 2 weeks of overload training [304]. However, this observation was accompanied by significant decrease in β2 adrenergic receptor density in the muscle, implying decreased

sensitivity of the β adrenergic receptors in the overloaded state. Either of these mechanisms might explain a faster HRRτ response after the race as this form of HRR is associated with a combination of parasympathetic reactivation and sympathetic withdrawal [37,40]. However, the current findings show a very similar effect size in both HRRτ and HRR_{60s}, which is somewhat contrary to this premise. HRR_{60s} has been attributed primarily to parasympathetic reactivation [29,39,40] and reduced sympathetic activity would be expected to improve HRRτ to a greater extent than HRR_{60s}. For example, Dupuy *et al.* [42] reported faster HRRτ responses but no change in HRR_{60s} responses associated with maximal exercise after 2 weeks of overload training in endurance athletes. The authors discussed reduced sympathetic activity or reduced β-adrenergic receptor sensitivity as possible mechanisms for this effect.

Another mechanism which might help to explain the faster HRR responses observed is plasma volume expansion. Plasma volume has been reported to be significantly increased in the days following a 42 km [305] and 56 km marathon [306], an effect attributed to an influx of serum albumin and electrolytes into the intravascular space over this period. In these examples, plasma volume expansion peaked on the 2nd day of recovery but remained significantly elevated on the 3rd day [305,306] and it is reasonable to speculate that plasma volume may have been increased above pre-race levels 3-4 days after the 87 km Comrades ultra-marathon. Buchheit *et al.* investigated the effect of plasma volume on post-exercise heart rate variability and HRR by comparing these measurements before and 2 days after a strenuous, supramaximal exercise bout designed to stimulate plasma volume expansion [307]. The authors reported that while heart rate variability measurements were sensitive to changes in plasma volume, there was no association between plasma volume and HRRτ. This would suggest that increased plasma volume did not contribute to the faster HRRτ observed in the current study. Conversely, Buchheit *et al.* reported a significant improvement in HRR_{60s} with increased plasma volume. However, this finding was attributed to a decrease in exercise HR rather than a true alteration in HRR_{60s} response. In the current study, there was no change in exercise HR hence this effect could not easily account for the faster HRR_{60s} response. Furthermore, a similar exercise HR may argue against significant plasma volume expansion [307,308], despite what has previously been observed following 42 km and 56 km races [305,306].

One further possible explanation for faster HRR responses 3-4 days after the ultra-marathon is faster parasympathetic reactivation following the post-race submaximal exercise bout, an effect that would account for improvements in both HRR_{60s} and HRRτ responses at a similar exercise HR. A “rebound” shift towards increased parasympathetic activity during a period of rest or lighter

training following a period of heavier training is supported by previous studies that monitored resting heart rate variability [73,309–311] or HRR [73] over the course of several weeks. Furthermore, previous studies have also demonstrated that a shift towards increased parasympathetic activity was related to improved performance at an individual level [311–313]. We did not measure performance in the current study and hence cannot be sure whether a similar relationship would have been observed. However, we could speculate that the significant increase in muscle soreness and RPE values in the post vs. pre-race trials would make a concomitant improvement in performance less likely.

The current observation of faster HRR in the presence of an increase in RPE is in keeping with a monitoring-based case-study by Lamberts *et al.* [293]. These authors measured weekly training load and HRR_{60s} responses in a world-class cyclo-cross athlete over 10 weeks and found that peaks in weekly training load were associated with increased RPE's, faster HRR_{60s} responses from 90% HRmax and increased scores on the Daily Analysis of Life Demands for Athletes (DALDA) questionnaire (indicative of increased lifestyle stress) [293]. Although increased RPE and DALDA scores might be expected to negatively affect performance, no maximal performance tests were conducted during this period [293]. Increased HRR_{60s} and increased RPE could suggest a shift towards increased parasympathetic activity accompanied by increased fatigue. However, this observation would be in contrast to the findings of Atlaoui *et al.*, who reported that increased parasympathetic activity (as assessed by resting heart rate variability) and was accompanied by reduced total fatigue [313]. Furthermore, these changes were associated with improved performance [313].

These apparently conflicting findings may suggest that increased parasympathetic activity accompanied by an increase in stress, fatigue or perceived exertion vs. increased parasympathetic activity accompanied by reduced stress, fatigue or perceived exertion represent different physiological states. While further investigation of these relationships is required, it seems that changes in HRR should be interpreted in combination with changes in other parameters when assessing an individual's readiness to train [314].

7.4.2 $EPOC_{MAG}$ and $EPOC\tau$

Although we are not aware of any studies to which the current $EPOC\tau$ responses can be compared, the current $EPOC_{MAG}$ responses can be partially compared to the findings of Bahr *et al.* [294] and Burt *et al.* [295]. In both of these studies, a submaximal exercise protocol, including an $EPOC_{MAG}$ measurement, was performed in a baseline or control condition and after a strenuous

exercise intervention. However, the nature of the exercise bout and the timing of the post-intervention were distinctly different. To be specific, Bahr *et al.* reported a significant, 24% increase in $EPOC_{MAG}$ immediately after 3-4 days of continuous, simulated combat exercises vs. a control condition whereas Burt *et al.* reported a significantly higher $EPOC_{MAG}$ 24 h and 48 h after a muscle-damaging protocol vs. baseline measurements. In both of these studies, the increase in $EPOC_{MAG}$ was associated with an increase in the metabolic demand of the preceding exercise bout. For example, Bahr *et al.* reported increased exercise VO_2, HR and EE and Burt *et al.* reported increased exercise VO_2, HR and VE. Both authors attributed the increase in $EPOC_{MAG}$ to the increase in exercise VO_2 and the implied reduction in mechanical efficiency. However Bahr *et al.* also cited an increased reliance on fat oxidation after the prolonged exercise as a possible mechanism for the increase in $EPOC_{MAG}$ and Burt *et al.* cited increased blood lactate concentration and VE as further possible contributors to the increased $EPOC_{MAG}$ after the muscle-damaging exercise.

In contrast, the current study found no differences in VO_2, VE, HR, RER and EE responses and no differences in $EPOC_{MAG}$ before and ~3 days after the ultra-marathon. This absence of an effect can probably be explained by the numerous methodological differences between the current study and the studies of Bahr *et al.* [294] and Burt *et al.* [295] including differences in the nature of the strenuous exercise intervention and the timing of the post-intervention test. Furthermore, the current findings are in keeping with the findings of Burgess *et al.*, who reported no significant difference in submaximal VO_2, HR or RER responses in race participants vs. controls 4 days after a Comrades ultra-marathon "up run" [301]. Taken together, these findings suggest that changes in EPOC are determined by changes in the homeostatic stress of the preceding exercise and, conversely, that similar exercise responses may produce a similar $EPOC_{MAG}$ despite an increase in RPE and muscle pain scores.

Although the change in $EPOC_{MAG}$ before vs. 3 days after the ultra-marathon was not of statistical significance, this change was associated with a *moderate* effect size. As there was no difference in exercise VO_2 measurements, the moderate decrease in $EPOC_{MAG}$ is likely to be explained by the moderate decrease in the plateau of the VO_2 recovery curve in the post-race trial. A decrease in VO_2 could be indicative of decreased fat oxidation in the post-exercise period, however this was not supported by recovery RER values, which remained constant in the pre- and post-race trials. Furthermore, the change in plateau VO_2 was not related to a change in the plateau of the HR recovery curve. It would seem that the moderate decrease in the recovery VO_2 plateau

cannot be explained from the current data and may be related to a possible shift towards increased parasympathetic activity, as discussed previously.

7.4.3 Individual variation in response

When the data of the current study were analysed at the group-level, there were changes that showed significantly faster HRR_{60s} and $HRR\tau$ responses and no change in $EPOC_{MAG}$ or $EPOC\tau$ 3 days after an ultra-marathon when compared to pre-race measurements. However, these group-level changes were not observed in all individuals. The individual variation in the physiological stress of the event and the time taken to recover can be attributed to the level of training going into the race and the relative intensity at which the race was completed. As a result, it is likely that individual participants were at different stages of the return towards full recovery at the time of the post-race trial. Nevertheless, it is still possible to compare HRR_{60s}, $HRR\tau$, $EPOC_{MAG}$ and $EPOC\tau$ as potential measures of training status based on which of these measurements showed changes in the highest number of participants and whether those changes were in a consistent direction. For example, HRR_{60s} appeared to be the most sensitive of the 4 measurements, showing (faster) HRR responses above day-to-day variation in 7 out of 10 participants and no change in the remaining 3 participants. $HRR\tau$ and $EPOC_{MAG}$ were somewhat less sensitive with a decrease greater than day-to-day variation in 5 of the participants and no change in another 5 participants. Conversely, individual $EPOC\tau$ responses were fairly evenly distributed between those who showed a meaningful decrease in $EPOC\tau$, no change in $EPOC\tau$ or an increase in $EPOC\tau$. It could be argued that inconsistency in individual $EPOC\tau$ response to an ultra-marathon race indicates that, of the 4 measurements under investigation $EPOC\tau$ response shows the least potential as a marker of "readiness to train".

7.4.4 Limitations

A significant limitation of the current study is that we did not measure performance before and after the race. Inclusion of a performance measurement would have allowed us to specify whether or not participants were overreached at the time of the post-race trial and generally aided interpretation of the findings. However, many of the participants found even the moderate-intensity of the submaximal trial aversive in the days after the race and the inclusion of, for example, a running time trial may have led to increased drop-outs and/or lower initial participant recruitment. A further limitation is that we could not standardize the relative physiological stress of the race between individuals and, for practical reasons, had to allow some individual variation in recovery time prior to the post-race trial. The current findings are also somewhat limited by the

relatively small sample size. It is possible, for example, that post vs. pre-race differences in $EPOC_{MAG}$ may have reached statistical significance in a larger sample of participants. Finally, it must also be acknowledged that the CV_{TEM} values by which individual responses were assessed were based on day-to-day variation and that typical CV_{TEM} values between the pre- and post-race trials may be somewhat larger. Conversely, the current participants were more highly trained than the participants group from which the CV_{TEM} values were calculated, a factor that would have contributed to lower typical measurement variation.

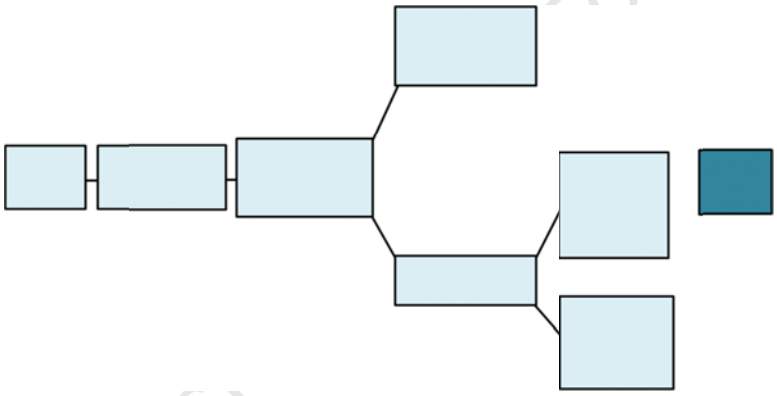
7.5 SUMMARY AND CONCLUSION

This study compared changes in HRR_{60s}, HRRτ, $EPOC_{MAG}$ and EPOCτ in response to the training "overload" of an ultra-endurance race and found that HRR_{60s} and HRRτ showed significantly faster response at the group level whereas $EPOC_{MAG}$ and EPOCτ showed no significant changes. The dissociation of HRR_{60s} and HRRτ vs. $EPOC_{MAG}$ and EPOCτ responses in the current study suggests either that these recovery measurements represent different aspects of exercise response or that HRR responses show greater sensitivity to changes in training load or "readiness to train" than measures of metabolic recovery. Many previous studies have supported a positive association between faster HRR and adaptation to training [36,41,81], however in the current study, HRR was improved just 3 days after an ultra-endurance event, despite an increase in RPE during submaximal exercise. This finding supports previous observations of a possible association between faster HRR and acute fatigue [42,293].

It follows that changes in HRR should be interpreted in combination with other parameters (e.g. RPE) when assessing an individual's fatigue or readiness to train [314], in keeping with a multi-factorial approach to monitoring [82,288,290]. For example, faster HRR in the presence of decreased RPE, stress or fatigue scores may be indicative of favourable adaptation to recent training loads and increased "readiness to train" whereas faster HRR in the presence of increased RPE, stress or fatigue scores may be indicative of a poor response to recent training loads and decreased "readiness to train".

CHAPTER 8

SUMMARY AND DISCUSSION



8.1 SUMMARY AND DISCUSSION

Several methods have been used to quantify the internal training load of a bout of exercise. However, a recent novel approach to quantifying internal training load has been to investigate the dynamic return towards resting homeostasis following the end of an exercise bout [15,16]. Objective and non-invasive methods of monitoring the return towards resting homeostasis include measures of heart rate recovery (HRR) and post-exercise oxygen consumption (EPOC). However, the relative potential of autonomic vs. metabolic recovery measurements to represent the internal training load or homeostatic stress of the preceding exercise bout is not clear. Therefore, the broad aim of this thesis was to investigate the magnitude of EPOC ($EPOC_{MAG}$), the time constant of the EPOC recovery curve ($EPOC\tau$), HRR within the first minute post-exercise (HRR_{60s}) and the time constant of the HRR curve ($HRR\tau$) as measures which might reflect the homeostatic stress of an exercise bout. It was hypothesized that a measure representing homeostatic stress could have the following possible applications; (i) to identify inter-individual variation in the homeostatic stress of a standardized exercise bout, (ii) to detect intra-individual variation in the homeostatic stress of different exercise bouts, and (iii) to detect intra-individual variation in "readiness to train", based on the response to a standardized exercise bout. Therefore, the investigations of this thesis aimed to assess the relative potential of $EPOC_{MAG}$, $EPOC\tau$, HRR_{60s} and $HRR\tau$ in these different roles. The main findings of the thesis, the practical application of these findings and possibilities for future research can be summarized as follows.

8.2 WHICH OUTCOME MEASUREMENT IS MOST CLOSELY RELATED TO INTER-INDIVIDUAL VARIATION IN THE HOMEOSTATIC STRESS OF AN EXERCISE BOUT?

8.2.1 Main finding

Although there is no gold standard measure of the homeostatic stress of an exercise bout, one measurement that is well-established as an integrated measure of homeostatic disturbance is Borg's RPE. Therefore, it was anticipated that a recovery measurement that showed potential to represent inter-individual differences in the homeostatic stress of an exercise bout would be related to RPE at a fixed relative intensity in a heterogeneous group of trained and untrained individuals. This premise was investigated in Chapter 4 and the main finding of the study was that

RPE had 48% of variation in common with HRR_{60s}, 23-26% of variation in common with $EPOC\tau$ or $HRR\tau$ and no significant association with $EPOC_{MAG}$ during a standardized exercise bout. This suggests that, of the 4 recovery measurements under investigation, HRR_{60s} showed the most potential to represent inter-individual variation in the homeostatic stress of an exercise bout in a group with a wide range of fitness levels.

8.2.2 Practical applications and future research

A possible practical application of this finding would be to predict high-responders and low-responders to a particular training intervention based on pre-training HRR_{60s}. Previous studies have found significant associations between HRR and physical activity levels amongst individuals with a relatively wide range of fitness levels [38,233], whereas the current study showed a significant association between HRR_{60s} and RPE. Taken together, these findings could imply that faster HRR_{60s} is associated with greater existing training adaptation and lower homeostatic stress during a standardized exercise bout. It follows that individuals with relatively greater initial levels of training adaptation would be expected to experience a relatively lower homeostatic stress (stimulus) during standardized training sessions and would, therefore, be more likely to show a relatively smaller improvement after a period of training. Conversely, a relatively slower HRR_{60s} response could indicate relatively lower initial levels of training adaptation, a relatively greater homeostatic stress during standardized training sessions and an increased likelihood of relatively larger improvement after a period of training. It is important to note, however, that preliminary evidence of these relationships was observed among individuals with a range of fitness levels and it is not clear whether comparable associations would be present among individuals with similar fitness levels.

Another consideration is that it is not clear whether HRR_{60s} would be more or less effective for predicting training response than pre-training RPE at a fixed relative intensity. Measuring HRR_{60s} requires that the individual wear a heart rate monitor whereas the measurement of RPE requires only a visual scale. HRR_{60s} has the advantage of being an objective measurement whereas RPE may be influenced by psychological factors [213,214]. As both of these measurements are relatively easy to obtain, it might be beneficial to use both measurements to interpret inter-individual variation in the homeostatic stress of an exercise bout and for the prediction of subsequent training responses. Alternatively, it is possible that pre-training HRR_{60s} may be more closely related to one training response phenotype (e.g. change in VO_2max) and RPE more closely related to another training response phenotype (e.g. change in lactate threshold values). To investigate these research questions, future studies could relate variation in pre-training RPE and

HRR_{60s} to variation in the phenotypes of different training responses in previously untrained participants or participants with a range of fitness levels.

8.3 WHICH OUTCOME MEASUREMENT IS MOST SENSITIVE TO INTRA-INDIVIDUAL VARIATION IN THE HOMEOSTATIC STRESS OF DIFFERENT EXERCISE BOUTS?

8.3.1 Main finding

In Chapter 6, the association between recovery measurements and changes in the homeostatic stress of an exercise bout was investigated by comparing the effect of exercise intensity on $EPOC_{MAG}$, $EPOC\tau$, HRR_{60s} and $HRR\tau$. It was anticipated that a recovery measurement sensitive to the homeostatic stress of the preceding exercise bout would show slower recovery with increased exercise intensity. When recovery responses were compared at 60%, 70% and 80% of VO_2max, $EPOC_{MAG}$, $EPOC\tau$ and $HRR\tau$ reflected slower recovery with increased exercise intensity, however only $EPOC_{MAG}$ was significantly different across all 3 exercise intensities. In contrast, HRR_{60s} reflected faster recovery in the 70% vs. 60% VO_2max and 80% vs. 60% VO_2max trials but was not different between the 70% and 80% VO_2max trials. The main finding of the study was that only $EPOC_{MAG}$ was sensitive to both larger- and smaller-changes in exercise intensity and showed the slower recovery responses with increased exercise intensity that were anticipated from a possible measure of intra-individual variation in the homeostatic stress of different exercise bouts.

8.3.2 Practical applications and future research

A practical application of this finding could be to use $EPOC_{MAG}$ as a tool to quantify the relative internal load of an individual's training sessions and to model the relationship between training load and training response. The possible use of $EPOC_{MAG}$ to measure (internal) training load has the disadvantage of being limited to laboratory conditions. However, this method would have the advantage of being objective and specific to the individual whereas existing methods of quantifying training load can have a subjective component (e.g. Session RPE [238]) or involve population-based averages as part of the training load calculation (e.g. TRIMPS [239]). Furthermore, it might be feasible to assess the $EPOC_{MAG}$ associated with an athlete's habitual training sessions in the laboratory and then assign these $EPOC_{MAG}$ values to subsequent field-based training sessions. This approach is supported by the findings of Chapter 7, which showed

that $EPOC_{MAG}$ appears to be related to the absolute demand of the preceding exercise bout rather than changes in "readiness to train".

At present, there is no gold standard measure of internal training load to which $EPOC_{MAG}$, or indeed other measures of training load such as Session RPE or TRIMPS, can be compared. As a result, it is difficult to assess the relative accuracy of these different methods for quantifying training load. While many authors have only compared different methods of quantifying training load against one another [315–317], other authors have related methods of quantifying training load to changes in exercise response parameters (e.g. lactate threshold concepts) [318–320]. Although there are a number of factors which could affect the relationship between a training load or "stimulus" and the subsequent training response (as reviewed in Chapter 2), training response is likely to represent one of the more meaningful standards of comparison available at present. It follows that a comparison of training load as assessed by $EPOC_{MAG}$, Session RPE and TRIMPs and subsequent adaptation to different types of training (e.g. endurance vs. resistance or interval vs. constant- intensity) and in different populations (e.g. trained vs. untrained) could provide a large scope for future study.

8.4 WHICH OUTCOME MEASUREMENT IS MOST SENSITIVE TO INTRA-INDIVIDUAL CHANGES IN "READINESS TO TRAIN"?

8.4.1 Main finding

To investigate the relationship between recovery measurements and "readiness to train", recovery responses were compared before and after an acute training "overload" in the form of an ultra-marathon road race, as described in Chapter 7. It was anticipated that the capacity to respond to training or "readiness to train" would be impaired in the days after the race and that a recovery measure with potential to monitor changes in readiness to train would be sensitive to this change. Although there was an increase in perceived muscle soreness and RPE after the race, other submaximal exercise measurements such as VO_2, HR, VE and RER did not change significantly, and there was no significant change in either $EPOC_{MAG}$ or $EPOC\tau$. The main finding of the study was that both HRR_{60s} and $HRR\tau$ reflected significantly faster HRR in the post-race trial when compared to the pre-race trial. Although both forms of HRR showed large and significant changes at the group level, HRR_{60s} could be considered the more sensitive of the two outcome measures based on individual responses.

8.4.2 Practical application and future research

The findings in Chapter 7 support previous findings that HRR is sensitive to acute changes in training load and may be valuable as a monitoring tool [292,293,321]. Nevertheless, there is some evidence that faster HRR can be associated with both adaptation to recent training loads [36,41] and acute fatigue (e.g. Chapter 7)[42,293]. Under most circumstances, the appropriate response to training adaptation would be an increased training load and the appropriate response to a fatigued state would be a reduced training load. It follows that the prescription of relatively higher or lower training loads based on faster HRR alone could result in suboptimal training adaptation or increased risk of non-functional overreaching or even overtraining syndrome. Therefore, changes in HRR should be interpreted along with changes in other parameters before adjusting training load.

In Chapter 7, the faster post-race HRR responses were associated with increased RPE's and the combination of HRR and RPE responses for interpreting readiness to train would be an interesting area for further research. For example, one potential study design could be to induce a state of overreaching in participants and then monitor day-to-day changes in HRR and RPE over the course of a recovery period. Based on the existing literature, it could be hypothesized that the first 1-2 days of rest or lighter training after an overload period would be characterized by increased RPE and slower HRR responses (possible interpretation: *little recovery, poor readiness to train*), the following 3-4 days by increased RPE and faster HRR responses (possible interpretation: *somewhat recovered but still relatively poor readiness to train*) and the subsequent days by a return to pre-overload RPE or decrease in RPE while retaining faster HRR responses (possible interpretation: *fully recovered, optimal readiness to train*). Testing this hypothesis would help to establish whether RPE and HRR alone might be sufficient to interpret an individual's physiological status or whether additional measurements are required.

As a further practical consideration, it is important to note that whether or not an individual is "ready" for the next training session is dependent on the nature of the training session. For example, a fatigued individual may be "ready" for a light training session but not for a heavy training session. Therefore, the concept described in this thesis as "readiness to train" is likely to be a spectrum ranging from "ready for rest" and "ready for light training" to "ready for hard training" and "ready for very hard training". In the event that a particular monitoring system has been shown to detect large changes in an individual's physiological status, subsequent studies could aim to increase the precision of detecting where an individual might fall on the "readiness to train" spectrum.

8.5 MODEL TO EXPLAIN INDIVIDUAL VARIATION IN RESPONSE TO TRAINING

The starting point from which this thesis developed was to understand the basis of individual variation in response to standardized training programs such as the HERITAGE study [1]. Factors that may contribute to individual variation in response were reviewed in Chapter 2 and this content was used to prepare the following summary of an individual's adaptation to exercise training.

Every bout of exercise functions as a stimulus (in the form of specific perturbations to resting homeostasis) and elicits an acute adaptive response (in the form of changes in mRNA and protein levels in the hours after the cessation of exercise). With ongoing repetitions of the exercise stimulus, the adaptive responses following each exercise bout accumulate to produce detectable differences in phenotype and functional capacity and the homeostatic disturbance associated with the same external workload is reduced. However, the characteristics of an exercise stimulus and the process by which it is translated into an accumulated adaptive response may be modulated by the complex interaction of a number of variables. These variables could influence an individual's potential to improve a particular parameter of exercise response or affect the extent to which an individual is ready to adapt over the course of a training program. Figure 8.1 contains a proposed model of how variation in the magnitude of the exercise stimulus, an individual's potential to improve exercise response parameters and an individual's readiness to train might interact to produce a relatively large or relatively small adaptive response following an exercise bout. The relative contribution of each of these factors to the accumulated training response would be very difficult to quantify. However, one could speculate as to how an individual might incur a particular training response, as follows.

The proposed model in Figure 8.1 attempts to explain how individual training responses may arise from variation in the stimulus associated with the training sessions, variation in the potential to improve a particular exercise response parameter and variation in readiness to train. While general trends in the magnitude of training response across different exercise parameters may apply, this overview is best suited to explain the training response in one particular parameter (e.g. VO_2max). It is possible that the same individual may show a different magnitude of training response in another parameter (e.g. submaximal heart rate) over the same period of exercise training [10].

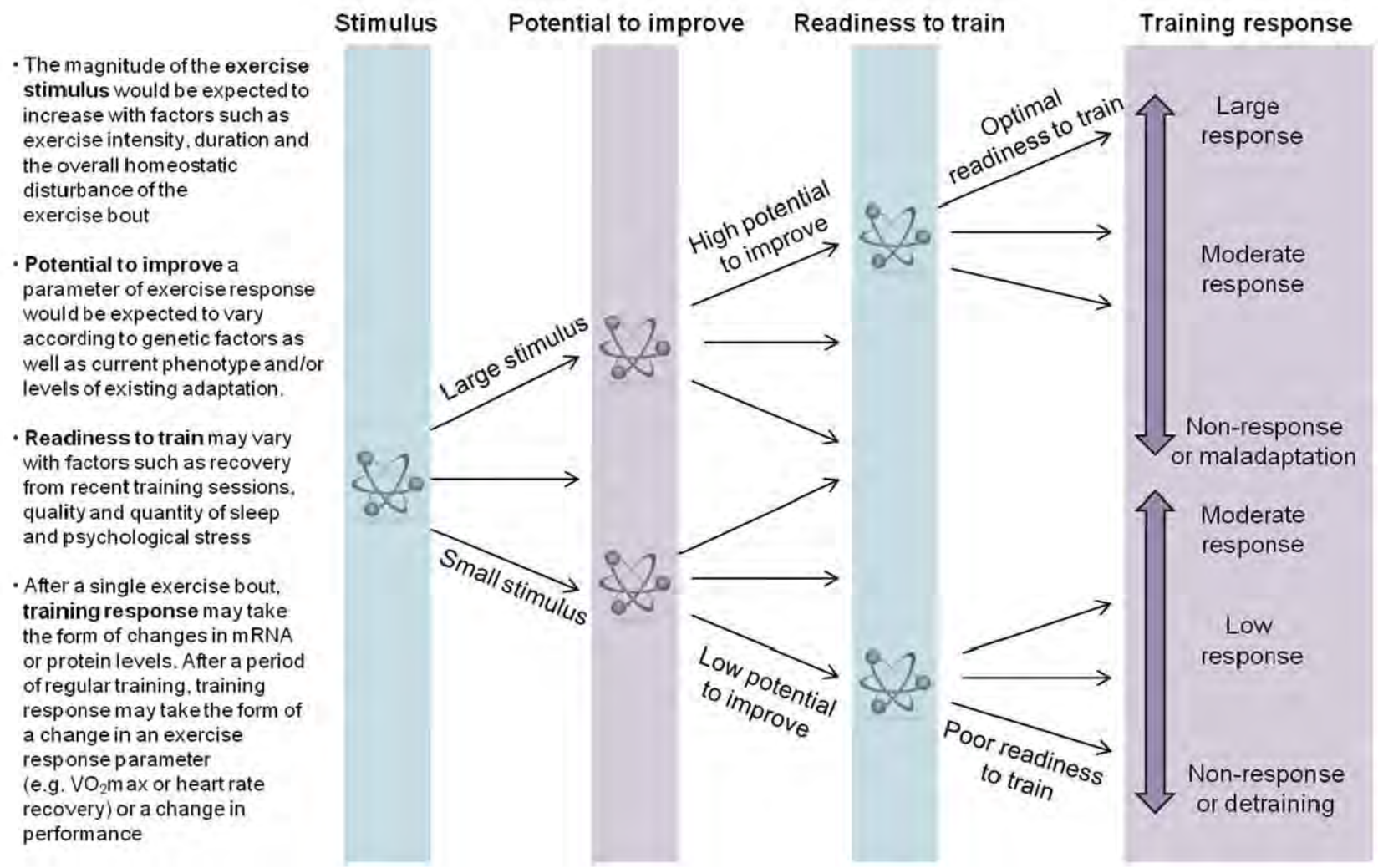


Fig 8.1 Proposed model explaining individual responses to training

8.5.1 Scenario 1 (Fig 8.2)

A previously untrained individual begins a training program and although the external load of the initial training sessions is relatively mild, each exercise bout is associated with a large homeostatic disturbance and exercise stimulus in the untrained individual. In the untrained state, this individual would be expected to have a relatively large potential to improve exercise response parameters compared to a trained individual. Although the potential to improve a particular response parameter may also be mediated by genetic factors, this individual's genotype is also conducive to a large potential to improve. However, this individual is typically only somewhat "ready to train" for each training session due to a stressful lifestyle and incomplete recovery from recent training sessions. Considering the interaction of all these factors, this individual might be expected to show a moderate-to-large improvement in training response parameters after several weeks of training.

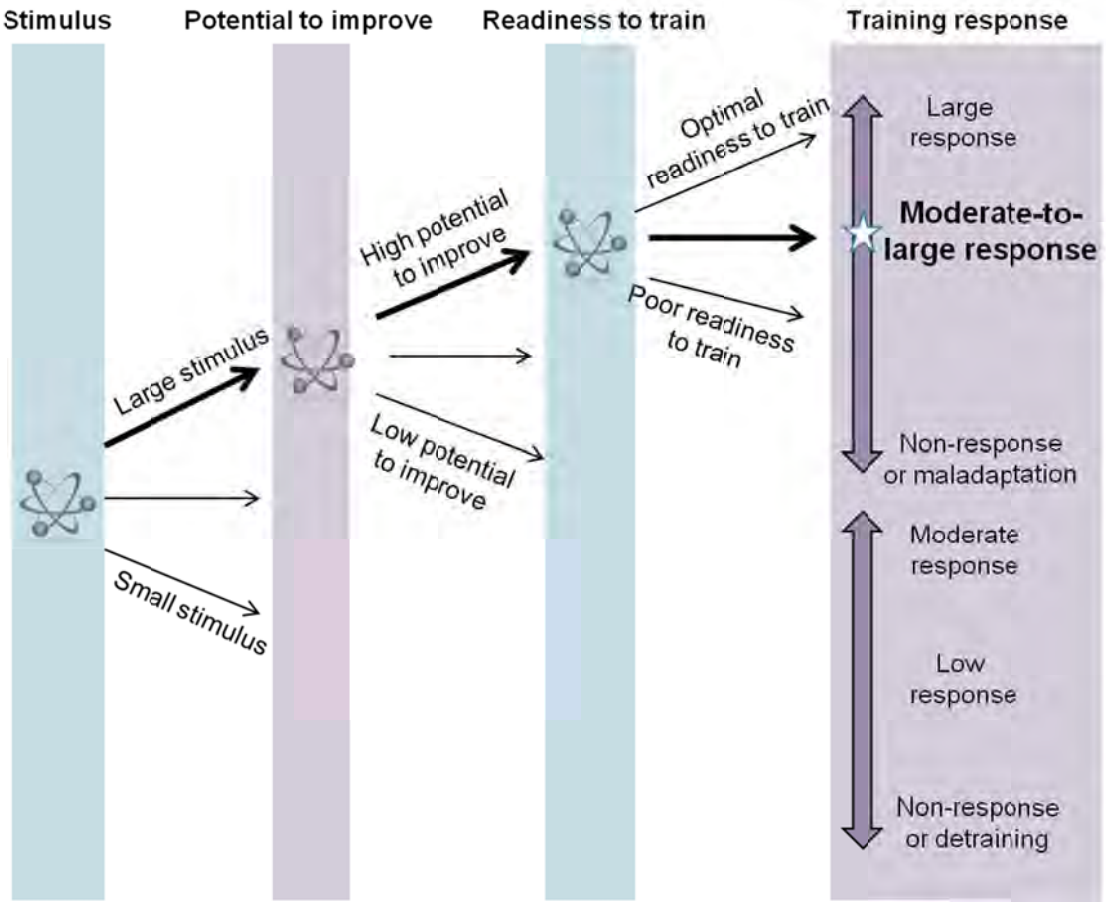


Fig 8.2 Predicted training responses for an untrained individual beginning a training program

8.5.2 Scenario 2 (Fig 8.3)

A moderately trained individual maintains a similar training regimen over several months with little variation in the relative intensity, frequency or duration of the training sessions. The individual becomes fully adapted to this training load and, as a result, the stimulus for further adaptation is small and the potential to improve exercise response parameters with the current training load is low. Even though the individual is generally well-recovered and "ready to train" for most training sessions, the interaction of these factors might be expected to maintain current training adaptations with little-to-know further improvement in training response parameters.

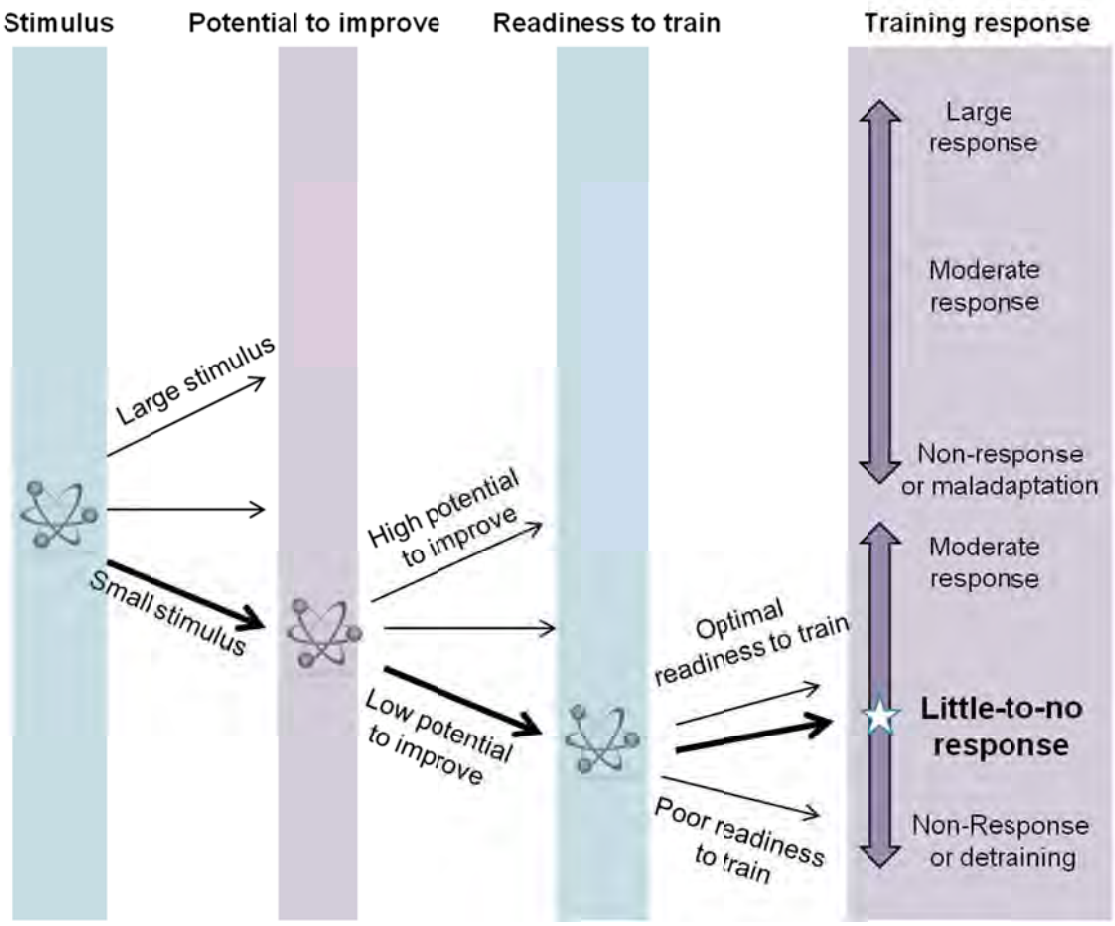


Fig 8.3 Predicted training responses for a moderately trained individual maintaining a training program

8.5.3 Scenario 3 (Fig 8.4)

A highly trained individual begins a period of overload training including frequent high intensity interval training sessions. These high intensity interval sessions are associated with a large homeostatic disturbance and exercise stimulus. However, the individual has already incurred a high level of training adaptation and the relative potential to improve exercise response parameters is only moderate. In the early stages of the overload period, the individual has moderate readiness to train and incurs a moderate training response. However, with further overload training, the individual accumulates fatigue from the frequent training sessions and has disturbed sleep, often resulting in poor readiness to train by the time of the next training session. This combination of high training load and poor recovery would be expected to produce unfavourable changes or maladaptation in certain exercise response parameters. It follows that rest or lighter training may produce an improvement in readiness to train and facilitate a return to moderate training responses.

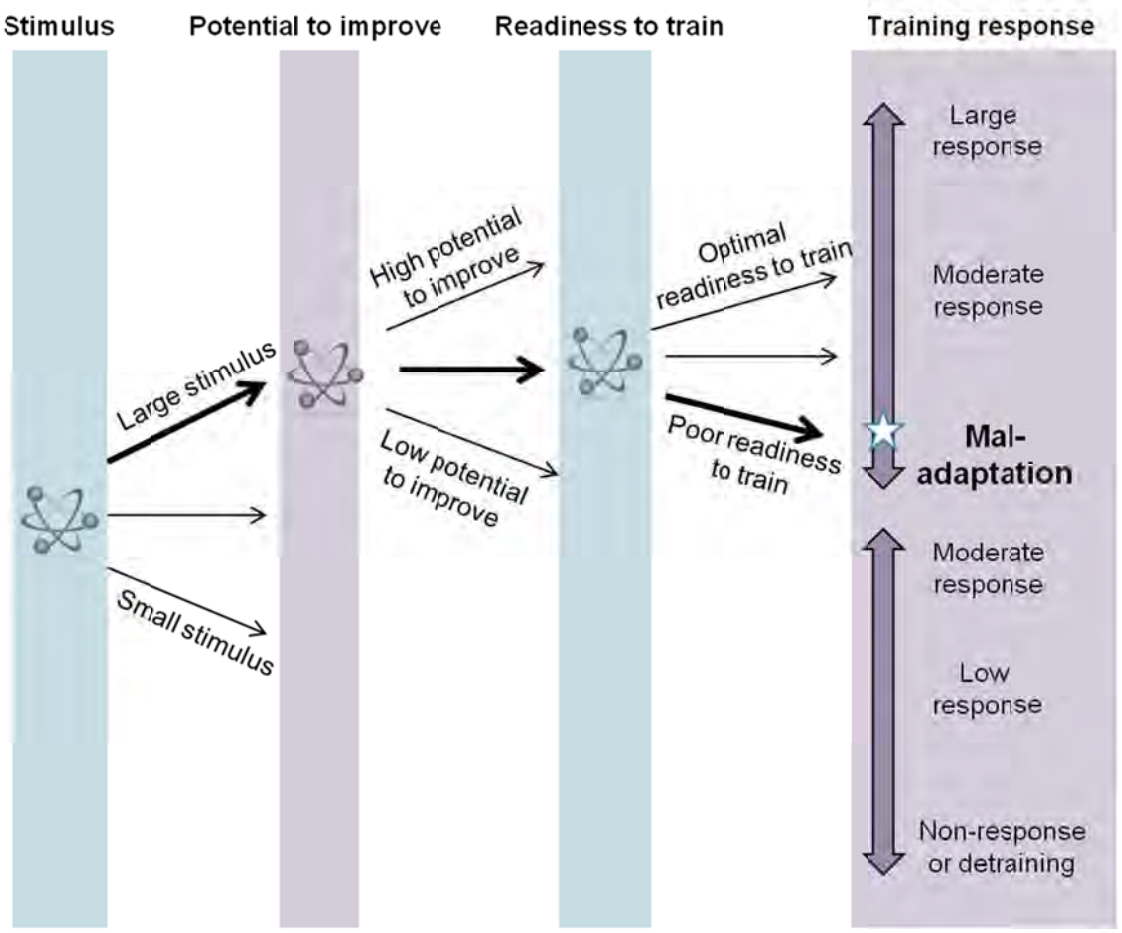


Fig 8.4 Predicted training responses for a highly trained individual completing a period of overload training

8.5.4 Model to explain individual variation in training response and the findings of the current thesis

Although one can speculate about the theoretical basis of the factors that might produce a relatively larger- or smaller- training response, to do so in practice requires a means to measure or quantify the magnitude of the exercise stimulus, the potential to improve a particular exercise response parameter and an individual's readiness to train. It is reasonable to suggest that the magnitude of the stimulus associated with an exercise bout would vary with the homeostatic stress of the exercise bout or internal training load. The findings of the current thesis showed that certain recovery measurements may have potential in this role as HRR_{60s} was related to inter-individual differences in RPE and $EPOC_{MAG}$ was related to intra-individual changes in exercise intensity. Furthermore, the findings of this thesis supported HRR_{60s} as a measurement sensitive to changes in readiness to train, although it would appear that changes in HRR_{60s} should be interpreted in the context of one or more additional measurements.

8.6 CONCLUSION

Although both post-exercise oxygen consumption and HRR represent the return towards resting homeostasis at the cessation of exercise, the findings of the current thesis suggest that different forms of these metabolic- or autonomic- recovery measurements show different sensitivity to inter-individual variation in the homeostatic stress of an exercise bout, intra-individual variation in the homeostatic stress of an exercise bout or intra-individual variation in readiness to train. Therefore, in response to the title of the thesis, *Post-exercise oxygen consumption and heart rate recovery as possible measures of the homeostatic stress of an exercise bout*, it can be concluded that both post-exercise oxygen consumption and HRR have potential as measures of homeostatic stress, depending on the form in which it is measured and the context of the application.

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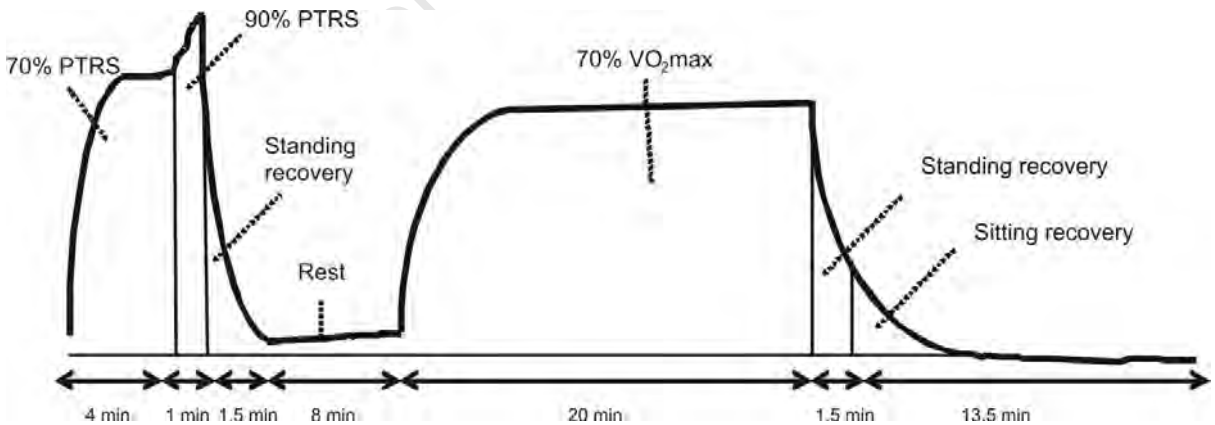
APPENDIX 1: MODIFICATIONS TO THE TESTING PROTOCOL FOR SUBSEQUENT STUDIES

A number of other changes were made to the laboratory protocols to be used in the subsequent studies of the thesis based on differences in study design and observations from the current study. These changes are briefly discussed in Table 1.1 and an overview of the adjusted protocol for submaximal exercise followed by recovery measurements appears in Fig 1.1.

Appendix Table 1.1 Changes in laboratory protocol between the current study and subsequent studies of the thesis

Protocol Change	Comment
Maximal Incremental test	
Bruce protocol to Peak Treadmill Running Speed Test to determine VO_2max	The Bruce protocol was used in the current study for the benefit of the untrained participants. However subsequent studies recruited only trained runners and the use of a peak treadmill running speed protocol to determine VO_2max was more appropriate.
Submaximal exercise with recovery measurements	
Introduction of a standardized warm-up	A 5 min warm-up was introduced before the main submaximal exercise to facilitate faster oxygen kinetics and attainment of a steady state during the submaximal exercise. The warm-up consisted of 4 min at 70% of the individual's peak treadmill speed followed by 1 min at 90% of the individual's peak treadmill speed.
3 km treadmill exercise to 20 min treadmill exercise	It was a limitation of the current study that an exercise bout standardized by distance produced variation in both exercise energy expenditure and exercise duration among participants. It is not possible to standardize both exercise energy expenditure, however we opted to standardize duration of the submaximal exercise for subsequent study by adjusting the 3 km treadmill exercise to a 20 min treadmill exercise.
60 min recovery period to 15 min recovery period	Although an individual may take up to an hour or more to fully restore resting homeostasis after exercise, the majority of heart rate recovery and metabolic recovery occurs within the first ~15 min post-exercise [322]. Therefore, the decision was taken to compare changes in recovery within the first 15 min post-exercise rather than the first 60 min post-exercise. A number of previous studies have used a 10-15 min recovery period when investigating heart rate recovery and/or metabolic recovery

	[33,34,323,324].
Positive incremental area under the curve to total area under the curve for determining EPOC magnitude	Jacobsen *et al.* [30] compared EPOC magnitude when corrected for baseline measurements ("positive incremental" area under the curve) and EPOC magnitude without correcting for baseline measurements (total area under the curve) and found that the latter measurement showed less intra-individual variation. This findings suggests that the total area under the curve may be more sensitive to changes in post-exercise metabolism within the same individual than the positive incremental area. If the research question required that the metabolic cost of the exercise be accurately quantified, then correcting for baseline measurements would be important. However, the research questions to be addressed in the subsequent studies of this thesis concerned the relative change in EPOC in response to an intervention rather than the absolute magnitude of EPOC. Therefore, the decision was taken to compare EPOC magnitude measurements calculated as the total area under the curve rather than the area under the curve corrected for baseline measurements. This approach also has the advantage of avoiding the methodological concerns associated with obtaining a meaningful measurement of resting metabolic rate [173].



Appendix Fig 1.1 Protocol for submaximal exercise and recovery trials. PTRS = Peak Treadmill Running Speed, VO_2max = maximal oxygen uptake

APPENDIX 2: PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)

Modified Physical Activity Readiness Questionnaire (PAR-Q)[217]

Name			**Date**
DOB	**Age**	**Home Phone**	**Work Phone**

Regular exercise associated with many health benefits, yet any change of activity may increase the risk of injury. Completion of this questionnaire is a first step when planning to increase the amount of physical activity in your life. Please read each question carefully and answer every question honestly:

Yes	**No**	**1) Has a physician ever said you have a heart condition and you should only do physical activity recommended by a physician?**
Yes	**No**	**2) When you do physical activity, do you feel pain in your chest?**
Yes	**No**	**3) When you were not doing physical activity, have you had chest pain in the past month?**
Yes	**No**	**4) Do you ever lose consciousness or do you lose your balance because of dizziness?**
Yes	**No**	**5) Do you have a joint or bone problem that may be made worse by a change in your physical activity?**
Yes	**No**	**6) Is a physician currently prescribing medications for your blood pressure or heart condition?**
Yes	**No**	**7) Are you pregnant?**
Yes	**No**	**8) Do you have insulin dependent diabetes?**
Yes	**No**	**9) Are you 69 years of age or older?**
Yes	**No**	**10) Do you know of any other reason you should not exercise or increase your physical activity?**

If you answered yes to any of the above questions, talk with your doctor BEFORE you become more physically active. Tell your doctor your intent to exercise and to which questions you answer yes.

If you honestly answered no to all questions, you can be reasonably positive that you can safely increase your level of physical activity gradually.

If your health changes so you then answer yes to any of the above questions, seek guidance from a physician.

Participant Signature	**Date**