Variation in Response and Recovery to Training Intensity in Highly Trained Rowers

Ana Christie Holt

BSc (The University of Auckland)

PgDipSpEx (Auckland University of Technology)

A thesis submitted to Auckland University of Technology in fulfilment of the requirements for the degree of

Master of Sport and Exercise

July 2016

School of Sport and Exercise Science

Table of Contents

List of Figures

List of Tables

List of Appendices

Attestation of Authorship

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person (except where explicitly defined), nor material which to a substantial extent has been submitted for the award of any other degree or diploma of university or institution of higher learning.

Ana Christie Holt

Date: 22 July 2016

Acknowledgements

I would firstly like to thank my primary supervisor Associate Professor Andrew Kilding for all the support and encouragement you have given me in both my academic and professional development throughout the past year and a half. Thank you for keeping me on track and helping me "tell the story I wanted to tell". Your guidance and reassurance in the face of setbacks has been invaluable and from which I have certainly learnt a lot.

To my secondary supervisor Dr Daniel Plews, your unwavering support and enthusiasm has been a huge inspiration not only towards my thesis, but also towards my development as a physiologist. Thank you for your honest feedback and for pushing me to produce my best work, even when that has meant more re-writing than I would have liked. Your perseverance in helping me through my statistical woes is something I will forever be extremely grateful for, as well as your constant reminders to "enjoy the process".

To Katherine Oberlin-Brown, thank you for your sacrifice of sensible wake-up times in order to help me throughout my data collection period. I hugely appreciate your time, support and friendship throughout this period; it wouldn't have been possible to do without you! On that note, thank you to Jessica Bush and Ben Scott for your mammoth efforts during preliminary testing, and to all those who assisted me in the collection of data for this thesis.

To Waikato University, School of Human Development and Movement Studies, specifically Dr Mathew Driller for your collaboration on the research undertaken in this thesis. I am so grateful for your accommodation of me in Cambridge and willingness to help, without which I would not have been able to conduct the research that interested me the most—thank you.

To Fabrice Merien and the AUT Roche diagnostics laboratory, for supporting the salivary cortisol analysis undertaken in the studies presented in this thesis. This would not have been possible to do without you, thank you.

To Dr Maheswaran Rohan, for your guidance with statistical analysis. Your input has helped me hugely in getting the most out of a complex data set, and it has been a relief to have your help.

To the athletes and coaches who were involved in these studies, thank you for your immense commitment, patience and enthusiasm, without you I would not have been able to conduct research in the area I wanted. It was a pleasure to get to know you all throughout the study and I wish you all the best for your future successes in rowing! In addition, to Allan Cotter and Rowing New Zealand for your accommodation of my research in the RNZ High Performance Centre. This support was pivotal in the conduction of my research, and for which I am extremely thankful.

To Sam, although you are useless at pipetting saliva and your psychologist skills are marginal, your presence throughout this process has been invaluable. Your support, encouragement, optimism, and ability to make me laugh no matter the situation has helped me through, and for that, I am so thankful to have you by my side.

Most importantly, Mum and Dad. Thank you for your unwavering support in everything I have I have set out to do in life. Your uncompromising love, encouragement, and belief in me is paramount to all that I have achieved, and I am eternally grateful for such amazing parents. I love you.

Intellectual Property Rights

Ethical Approval

Ethical approval for the studies undertaken in this thesis was granted from Auckland University of Technology's Ethics Committee (AUTEC) on 24 June 2015; Ethics Application Number: 15/36.

Abstract

Background: In endurance sports, where training loads are high, effective programming of day-to-day training is crucial to achieve sufficient training stimuli and adequate recovery for optimal adaptation. The time required for recovery to a pre-exercise state following a single exercise stimulus is influenced by exercise intensity, although studies to date investigating this influence implement passive recovery periods. This does not reflect a real-world setting, where high training frequencies often require subsequent training to be performed prior to complete recovery. Furthermore, no study has investigated the influence of energetic profile on recovery time-course, which may prove valuable in individualising training programming.

Aims: 1) To quantify the acute post-exercise deviation and time-course for recovery to baseline following different high-intensity interval training sessions throughout a non-passive recovery period. 2) To investigate the influence of energetic profile on the acute deviation and time-course for recovery to baseline.

Methods: Ten male and three female highly trained rowers (mean ±SD age: 20.2 ±3.7 yr; body mass: 83.4 ±9.4 kg; VO_2peak: 4.93 ±0.71 $L \cdot min^{-1}$) completed preliminary testing to determine energetic contribution to a 6 min maximal rowing test. On separate days, participants completed three interval training (IT) sessions on the rowing ergometer: 5 x 3.5 min, 4 min rest periods (VO_2); 10 x 30 s, 5 min rest periods (Glycolytic); and 5 x 10 min, 4 min rest periods (Threshold). Intervals were requested to be performed at the highest possible maintainable pace. Blood lactate and salivary cortisol were measured pre, 3 and 30 min post-exercise respectively. Resting heart rate (HR) variability (HRV), post-submaximal exercise HRV (HRVex), submaximal HR (HRex), HR recovery (HRR), modified Wingate peak power and mean power, and subjective recovery (REC-Q) were measured pre and 1, 10, 24, 34, 48, 58, and 72 h post-exercise. Study One involved the comparison of mean acute post-exercise deviation and time-course for recovery to baseline data between the three IT sessions. Study Two retrospectively selected four male and two female participants from Study One for matched pair comparison. Pairs were matched for performance capacity (<1% difference in 6 min mean test power or 2000 m test time) with differing energetic profiles (>6.8% difference in aerobic energy system contribution to the 6 min rowing test).

Results: Study One found either *trivial* or *unclear* differences in the acute deviation from baseline of blood lactate, salivary cortisol, HRV, HRVex, HRex, HRR, REC-Q, modified Wingate mean and peak power between IT sessions. HRVex had the longest time-course for baseline return: 37.8 ±14.2 h (mean ± CL) post-Threshold, 20.2 ±11.0 post-Glycolytic, and 20.6 ±15.2 h post-VO_2 IT. Partial correlations revealed participants with greater aerobic energetic contributions to have shorter recovery time-courses for HRR following Threshold IT (r = -0.52 ±0.51), but longer recovery time-courses for HRex following Threshold IT (r = 0.53 ±0.51) and HRVex following Glycolytic IT (r = 0.36 ±0.47) in the analysis of all thirteen participants.

In Study Two matched pair comparison revealed participants with greater anaerobic energetic contributions (AnT) had a 64.1 ±103.4% (mean ±SD) greater blood lactate response across IT sessions than participants with greater aerobic contributions (AeR). AeR illustrated larger acute HRV (17.7 ±216.2%), HRVex (40.1 ±68.7%), HRR (76.4 ±168.5%), cortisol (229.2 ±479%), and HRex (57.0 ±113.9%) responses across IT sessions. Larger acute mean power reduction (107.6 ±100.8%) in AnT across IT sessions. Longer HRVex (18.0 ±35.9 h) and HRex (10.5 ±18.0 h) recovery-time courses in AeR, with no consistent difference in recovery-time course for HRV, HRR, mean or peak power between AeR and AnT.

Conclusion: Acute deviations from baseline were similar following Threshold, Glycolytic, and VO_2 IT across all recovery variables measured in highly-trained male and female rowers. However, following resumption of training, return to a pre-exercise state is prolonged following Threshold compared to Glycolytic and VO_2 IT. Suggesting the existence of a durational effect on time to recover following exercise performed at HR intensities reflective of $\geq VT_2$. In addition, athletes presenting greater aerobic contributions demonstrate higher rates of parasympathetic recovery in comparison to athletes presenting greater anaerobic energetic contributions, however this did not correspond to differences in recovery time-course. These findings indicate energetic contribution to have limited practical influence on individualising the programming of high-intensity interval sessions, with regards to the time-course of recovery between acute sessions. However, the influence of individualising training programming with regard to energetic profile on the long-term adaptive response is unknown, and thus warrants further research.

Chapter One: Introduction

1.1 Background

Successful athletic performance at the elite level represents an interaction between training optimisation and genetic potential. Whereby the nature of the training stimulus and an athlete's inherent response to training regulate the overall adaptive outcome and resultant capacity for performance (Bouchard et al., 2011; Rankinen et al., 2012; Tucker & Collins, 2012). In order to achieve success in elite intermediate duration sports such as rowing, training typically reflects high training frequencies of up to three times per day, limiting the time available for recovery between training sessions to between 4-12 h (Fickerstrand, 2004; Seiler, 2010). However, training adaptation is maximised when the appropriate balance between training and recovery is achieved (Coutts, Wallace, & Slattery, 2007; Halson et al., 2002). Thus, the effective programming of training within a microcycle is of high importance, as the chronic programming of subsequent training sessions without sufficient recovery periods can lead to maladaptation and overtraining (A Fry et al., 1994; A Fry, Schilling, Weiss, & Chiu, 2006).

Performing strenuous exercise challenges the body's homeostatic state. This perturbation of homeostatic functioning represents a period of reduced exercise capacity, whereby physiological systems recover and adapt to the imposed stress (Petersen, Hansen, Aagaard, & Madsen, 2007). Resulting in a temporary super-compensatory rebound over and above baseline homeostasis, whereby performance capacity is enhanced (Garet et al., 2004; Jacks, Sowash, Anning, McGloughlin, & Andres, 2002). However, the duration of the post-exercise recovery period is variable and dependent on factors including the intensity of the training stimulus (Mourot, Bouhaddi, Tordi, Rouillon, & Regnard, 2004; Niewiadomski, Gąsiorowska, Krauss, Mroz, & Cybulski, 2007), cardiovascular fitness (R. Fry, Morton, Garcia-Webb, & Keast, 1991; Hautala et al., 2001; Seiler, Haugen, & Kuffel, 2007), training history (McDonald, Grote, & Shoepe, 2014), and lifestyle factors such as psychological stress (Perna & McDowell, 1995) and sleep (Skein, Duffield, Minett, Snape, & Murphy, 2013). Thus, complicating the art of training programming, particularly for sports of intermediate durations (3-8 min) such as rowing, track cycling, flat-water kayak, and middle-distance running, where a range of training intensities are performed throughout the training season (Schumacher & Mueller, 2002; Seiler & Tønnessen, 2009; Steinacker, Lormes, Lehmann, & Altenburg, 1998).

The mechanisms involved in restoring post-exercise homeostatic perturbation represent a multitude of integrated physiological systems. These mechanisms are involved in neuromuscular recovery (Garrandes, Colson, Pensini, Seynnes, & Legros, 2007), muscle metabolite clearance (Tesch, 1979), endocrine response (Ahtiainen, Pakarinen, Kraemer, & Häkkinen, 2003), glycogen and phosphocreatine (PCr) store repletion (Haff, Lehmkuhl, McCoy, & Stone, 2003; Hirvonen, Rehunen, Rusko, & Härkönen, 1987), thermoregulation (Nybo, 2008), cardiac parasympathetic reactivation (Stanley, Peake, & Buchheit, 2013), and performance recovery (Garrandes et al., 2007). However, a discordance exists between the recovery rate of the aforementioned parameters, with parameters such as neuromuscular recovery appearing to recover faster than that of muscle glycogen repletion (Andersson et al., 2008; Krustrup et al., 2011). In addition, subjective ratings of recovery have been shown to disagree with physiological measures of recovery (Saw, Main, & Gastin, 2015), making the identification of true homeostatic recovery difficult.

An accurate assessment of post-exercise homeostatic return may therefore require quantification of a variety of subjective and physiological recovery measures. Cardiac autonomic function represents the recovery of multiple physiological systems following exercise, thus providing a comprehensive assessment of homeostatic status (Delp & O'Leary, 2004; Edis & Shepherd, 1970). Furthermore, heart rate variability (HRV) and heart rate recovery (HRR) indices provide quick and non-invasive measurements of cardiac autonomic balance. In addition, performance capacity presents another key measure for the assessment of recovery, given the reduction in exercise capacity observed following fatiguing exercise (Krustrup et al., 2006). Finally, subjective measures demonstrate an important tool for assessing post-exercise recovery status, given their increased sensitivity to acute changes in training load and reflection of the athlete's psychological readiness to perform (Jokela & Hanin, 1999; Saw et al., 2015).

The time course of recovery following a training session has been shown to be longer following training performed above the first ventilatory threshold (VT_1) (Mourot et al., 2004; Niewiadomski et al., 2007; Parekh & Lee, 2005; Seiler, 2010; Seiler et al., 2007). Specifically, parasympathetic reactivation has been shown to require up to 24 h following low intensity aerobic training, 24-48 h following threshold intensities and more than 48 h following high intensities (Furlan et al., 1993; James, Barnes, Lopes, & Wood, 2002; Kiviniemi et al., 2010; Mourot et al., 2004; Seiler et al., 2007). Furthermore, the acute

post-exercise deviation from baseline has been found to correlate to recovery time-course, with greater indices of parasympathetic suppression relating to longer time-courses for parasympathetic recovery (Stanley et al., 2013). Extended running durations over 14 km appear to increase post-exercise parasympathetic suppression compared to 3 km of intensity matched running (Hynynen, Vesterinen, Rusko, & Nummela, 2010; Kaikkonen, Hynynen, Mann, Rusko, & Nummela, 2010). However interestingly, little evidence has found exercise duration to relate to recovery time-course when controlling for exercise intensity (Bernardi, Passino, Robergs, & Appenzeller, 1997; Hautala et al., 2001; Seiler et al., 2007; Stanley et al., 2013). While only minimal acute responses have been observed in endocrine markers following extended durations of exercise performed below VT_1 (Jacks et al., 2002; Nieman et al., 1999).

Cardiovascular fitness presents another factor influencing the time-course of homeostatic return, with enhanced metabolite clearance, substrate delivery, and autonomic recovery observed in individuals with more favourable levels of cardiovascular fitness (Dixon, Kamath, McCartney, & Fallen, 1992; Greiwe et al., 1999; Hautala et al., 2001; Hickner et al, 1997; Seiler et al., 2007; Stanley et al., 2013). Anatomical and physiological adaptations following endurance training are proposed to enhance the intrinsic ability to not only return the body to its resting homeostatic state, but also to induce less acute post-exercise homeostatic deviation (Huang, Webb, Zourdos, & Acevedo, 2007).

An additional factor found to influence post-exercise recovery dynamics is that of energetic profile. An athlete's energetic profile describes the nature of their adaptive response to training, and is thus influenced by a combination of training history and genetic factors (Bouchard et al., 2011; Rankinen et al., 2012; Tucker & Collins, 2012). Athletes exhibiting predominantly anaerobic energy system contributions to exercise have been found to induce greater acute parasympathetic suppression, metabolic response, neuromuscular fatigue and larger performance decrements than their predominantly aerobic energy system contribution counterparts (Buchheit, Hader, & Mendez-Villanueva, 2012; Bundle, Hoyt, & Weyand, 2003; Del Rosso, Nakamura, & Boullosa, 2016; Garrandes et al., 2007; McDonald et al., 2014; Otsuki et al., 2007). Unfortunately, a lack of research exists regarding the influence of energetic profile on post-exercise recovery time-course, and thus any influence is yet to be established. Nevertheless, given the association between acute post-exercise deviation and recovery time-course to baseline (Stanley et al., 2013), an influence is expected to exist.

The significance of energetic profile on training programming considerations is particularly evident in intermediate duration sports such as rowing, track cycling, flat-water kayak, and middle-distance running, which often require significant aerobic and anaerobic fitness. Accordingly, successful athletes in such sports have the potential to display a relatively wide range of energetic profiles (Craig & Norton, 2001; de Campos Mello, de Moraes Bertuzzi, Grangeiro, & Franchini, 2009; D. W. Hill, 1999; Schumacher & Mueller, 2002; Zouhal et al., 2012). For example, previous research has found the anaerobic contribution to a 1500 m race in eight female middle distance runners to differ by 7% (D. W. Hill, 1999). Thus, it is possible that several athletes performing the same training session may have inherently different recovery needs, which must be considered in order to maximise the adaptive response across all athletes within a team or training squad. Indeed, individualising training programming with regard to the individual's recovery state and readiness to train, measured via morning resting HRV, has been shown to be advantageous for maximising the adaptive response (Kiviniemi et al., 2010; Kiviniemi, Hautala, Kinnunen, & Tulppo, 2007; Vesterinen et al., 2016). However, the typical error associated with recordings of cardiac parasympathetic activity is relatively large (Al Haddad, Laursen, Chollet, Ahmaidi, & Buchheit, 2011), and research examining daily training prescription based on cardiac parasympathetic indices in elite athletes is currently lacking.

Regardless of the research examining the intensity effect on recovery time-course, there remains to be research conducted in a real-world setting. Current recommendations regarding time required for recovery to baseline are based off studies examining passive recovery following a single exercise bout, however the high training frequencies of endurance athletes typically do not allow for 48 h of passive recovery (Seiler et al., 2007). Thus, research examining recovery time-course from key high-intensity sessions and within a typical training week is warranted, particularly given the proposed effect of subsequent low-intensity exercise on hastening the recovery time-course following high-intensity exercise (Stanley et al., 2013). Finally, the influence of energetic profile on recovery time-course is yet to be established, whereby future research examining the influence of energetic profile in athletes performing intermediate duration sports would be of particular benefit.

1.2 Study aims

The objective of this thesis is to extend the current knowledge regarding the optimal recovery period preceding subsequent high-intensity training sessions, and to gain insight into factors influencing recovery time-course. The aims of thesis are therefore to:

1. Quantify the acute post-exercise deviation and recovery time-course to baseline following high-intensity interval training sessions performed at intensities reflecting the maximal oxygen uptake (VO_2), the second ventilatory threshold– VT_2 (Threshold), and maximal anaerobic glycolytic power (Glycolytic) throughout a non-passive recovery period; and
2. Investigate the influence of energetic contribution on inter-individual variation in the acute post-exercise deviation and time-course for recovery to baseline following the aforementioned interval training sessions.

1.3 Study hypotheses

1. It is hypothesised that homeostatic perturbation and time-course for recovery will be greatest following Glycolytic interval training, followed by VO_2, and with Threshold interval training generating the smallest acute post-exercise deviation and time-course for recovery to baseline.
2. Athletes presenting greater anaerobic energetic contributions will experience greater acute post-exercise deviation and longer time-courses for recovery to baseline following VO_2, Threshold, and Glycolytic interval training stimuli.

1.4 Thesis organisation

This Master's thesis is intended to examine variability in the acute deviation and time-course for recovery to baseline in differing high-intensity exercise stimuli, as well as its application to programming successive exercise bouts. This thesis adheres to pathway two, as classified by the Auckland University of Technology post-graduate thesis structure guidelines (AUT post-graduate handbook 2016). The sections in this thesis include an introduction, literature review, two studies, conclusion, and appendix.

Specifically, Chapter One includes the introduction, which provides context and presents an overview of the thesis. Chapter Two incorporates a literature review introducing the reader to the concept of an optimal recovery period following a single exercise bout for

the programming of subsequent training sessions. This involves an examination of the mechanisms involved in returning the body to its pre-exercise homeostatic state, and the various ways in which these mechanisms can be assessed throughout the post-exercise period, providing an index of recovery and readiness to train. Factors contributing to the variation of recovery time-course are then analysed, with specific reference to cardiovascular fitness, the nature of the exercise stimulus, energetic contribution, and lifestyle factors. Finally, directions for future research are discussed in reference to the implementation of training programming individualisation, with the purpose of identifying the potential and practicality of such a practice.

Chapter Three presents the first study of this thesis; an experimental study examining the mean magnitude of acute deviation and time-course for recovery to baseline following an exercise stimulus incorporating either VO_2, Threshold or Glycolytic intensities in highly trained rowers. The findings of this study raise questions of which are broached in Chapter Four. Chapter Four presents a case-study comparing athletes matched for current performance ability but presenting differing energetic contributions to a rowing performance test, to determine whether the incidence of inter-individual variation in the magnitude of acute deviation and time-course for recovery to baseline following the aforementioned training stimuli is related to energetic profile. The two studies presented in this thesis have been prepared specifically for publication in peer-reviewed journals, and thus have been formatted in consideration of word limits. All citations in this work have been presented in American Psychological Association (APA) referencing, and are collated in Chapter Six at the end of the thesis.

Finally, Chapter Five incorporates an overall discussion and conclusion, evaluating the findings of both studies 1 and 2, including practical applications of the findings from the research completed, as well as limitations and areas for future research.

1.5 Significance of thesis

The performance potential of elite athletes is enhanced through the precise execution of each training session, as each contributes to the overall accumulated physiological response. Thus, the advantage of maximising an athlete's adaptive response to each training session performed is evident, particularly given the often minor margin between success and failure in elite sport. The effective programming of subsequent training

stimuli is crucial in achieving adequate recovery for the optimal adaptive outcome, whilst maximising the training stimuli. However, the time required to achieve adequate recovery following exercise proves dependant on the exercise stimulus and appears to vary between individuals. Therefore, this thesis seeks to contribute to the literature regarding the time-course for homeostatic recovery following typically performed training sessions to inform optimal training programming strategies. Furthermore, the currently limited body of knowledge regarding inter-individual variation in recovery time-course is added to, with an objective of generalising this variation with regard to energetic profile, for its practical application in individualising training programming.

Chapter Two: Literature Review

Recovery from a single exercise bout: measurement and influencing factors

2.1. Introduction

Successful performance at the elite level often requires years of well-programmed training, allowing athletes to adapt optimally to meet the performance demands of the sport. Due to the highly specific nature of physiological adaptation, training must reflect the energy systems characterising the sport. In intermediate duration (3-8 min) sports such as rowing, middle distance running, flat-water kayak, and track cycling, substantial contributions from both the aerobic and anaerobic energy systems are evident (Craig & Norton, 2001; de Campos Mello et al., 2009; D. W. Hill, 1999; Zouhal et al., 2012). Therefore, effective adaptation to meet the energetic demands of racing requires a variety of training stimuli (Fiskerstrand & Seiler, 2004; Seiler, 2010). Importantly, adaptation to training is maximised when the appropriate balance between training and recovery is achieved, allowing super-compensation to occur. Conversely, high training loads with insufficient recovery periods compromise the adaptive ability (Jeukendrup, Hesselink, Snyder, Kuipers, & Keizer, 1992), which overtime can manifest as non-functional overreaching and overtraining (Meeusen et al., 2013). However, the type of training performed determines the degree of recovery required (Stanley et al., 2013), further complicating the art of training programming for intermediate duration sports.

The nature of the training session performed dictates the resultant physiological stress generated, and therefore the degree of homeostatic perturbation. Following a single training session perturbation to a range of physiological parameters occurs; including muscle metabolite (Tesch, 1979) and cortisol (Ahtiainen et al., 2003) accumulation, glycogen (Haff et al., 2003) and phosphocreatine store depletion (Hirvonen et al., 1987), suppression of cardiac parasympathetic activity (Seiler et al., 2007), and neuromuscular fatigue (Garrandes et al., 2007). However, these parameters each present a differing time-course for recovery and appear to be influenced by a number of factors including cardiovascular fitness, intensity and duration of the exercise stimulus, gender, and lifestyle factors such as sleep, nutrition and psychological stress (Hawley, Burke, Phillips, & Spriet, 2011; Kiviniemi et al., 2010; Perna & McDowell, 1995; Samuels, 2009; Seiler et al., 2007; Tomlin & Wenger, 2001). Therefore, the next training session programmed should consider the time period required for the recovery of homeostasis.

The time-course of recovery from a single exercise bout is illustrated by the super-compensation curve (*Figure 2.1*), whereby the capacity to deviate from homeostasis—and therefore adapt to a training stimulus—immediately following exercise can be inhibited for over 72 h (Sherman et al., 1983). After the return of homeostatic functioning to its pre-exercise state a temporary super-compensatory rebound is observed over and above baseline homeostasis, whereby performance capacity is enhanced (Buchheit, Laursen, Al Haddad, & Ahmaidi, 2009; Hautala et al., 2001). Given the appropriate balance between training and recovery is required to maximise training adaptation (Coutts et al., 2007; Halson et al., 2002), the effective programming of training within a microcycle is therefore of high importance, particularly as the chronic programming of subsequent training sessions without sufficient recovery periods can lead to maladaptation and overtraining (A Fry et al., 1994; A Fry et al., 2006). Moreover, achieving this balance proves difficult for elite athletes, notably in endurance sports where high training volumes are often achieved via high training frequencies (Fiskerstrand & Seiler, 2004; Seiler, 2010). Since the recovery time required to achieve a super-compensated state is dependent on the homeostatic stress of the exercise stimulus (and is thus influenced by the intensity and duration of the training session performed), a more practical means of achieving sufficient recovery is by interspersing the type of session performed. Therefore, knowledge of specific recovery characteristics following various types of training proves highly valuable for the optimisation training programming.

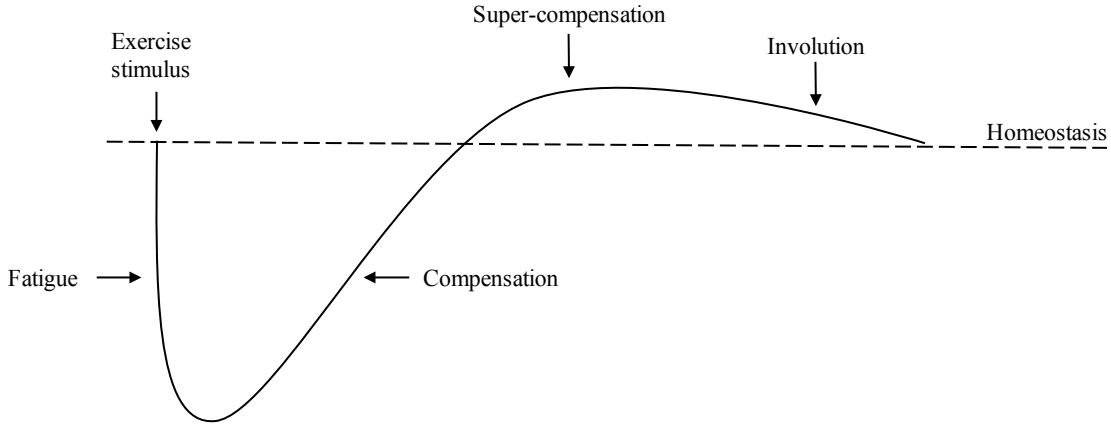

Figure 2.1 Super-compensation curve following an exercise stimulus. Redrawn from Bompa and Haff (2009).

The acute post-exercise deviation and time-course for recovery to baseline is also dependant on the individual. Both the magnitude of acute deviation and recovery time-course has been found to differ in athletes performing the same exercise stimulus (Lamberts, Swart, Capostagno, Noakes, & Lambert, 2010; Mann, Webster, Lamberts, & Lambert, 2014). Although, in the sport science literature examining physiological response and recovery following training it is widely accepted to report the group mean and standard deviation without presenting the range. This practice largely hides the observation that certain individuals within the study population will demonstrate a wide variation in response, in comparison to the population mean (Vollaard et al., 2009). This is described as inter-individual variation and in the case of this review represents the variation that is often found between individuals in the physiological response and recovery time-course following a standardized exercise stimulus. Although research in this area is currently limited, inter-individual variation has been found to account for up to 42% of the response in aerobic capacity to a standardized exercise stimulus (Kohrt et al., 1991). However, when considering the environmental, biological, and genetic diversity of the human race (Bouchard & Rankinen, 2001) this degree of variation is nevertheless unsurprising.

Given the wide range of exercise stimuli, recovery variables, and the individual nature of their responses; identification of the optimal time period between exercise bouts proves difficult. In addition, the magnitude of acute post-exercise homeostatic deviation appears to be influenced by training history, as endurance-trained athletes demonstrate reduced neuromuscular fatigue and metabolic response following high-intensity exercise compared to power-trained athletes (Garrandes et al., 2007; Paavolainen, Häkkinen, Nummela, & Ruskol, 1994). Considering these factors, identification of patterns of response and recovery based on the nature of the exercise stimulus and energetic profile of the athlete may simplify the act of individualising training programming for the maximisation of training adaptation.

2.1.1. Purpose of the review

The purpose of this review is to discuss the measurement of recovery following exercise and the various factors influencing the variation in recovery time-course between individuals. Specifically, this review will discuss measurement of the autonomic and endocrine systems, performance variables, and psychometric measures for the assessment

of homeostatic recovery following varying exercise stimuli. Factors contributing to the observation of inter-individual variation in physiological response and recovery from exercise will then be analysed; including the influence of cardiovascular fitness, nature of the training stimulus, training history, energetic profile, and lifestyle factors. Finally, the significance and practical potential of recovery variation on the optimisation of training programming for intermediate duration sports such as rowing will be discussed.

2.2 Measures of recovery

Recovery from exercise involves a multifaceted physiological response involving integrated systems, each presenting differing recovery time-courses. These systems work to return the body to its pre-exercise homeostatic state and are reflected by cardiac parasympathetic reactivation (Seiler et al., 2007; Stanley et al., 2013), the return of stress hormones to resting levels (Ahtiainen et al., 2003), performance capacity return (Garrandes et al., 2007; Krustrup et al., 2006), and subjective measures of readiness to train (Sikorski et al., 2013). Thus, the need for a variety of measures for the assessment of homeostatic return is warranted. The following section will discuss key measures for the assessment of homeostatic return and recovery from exercise, with each describing the underlying physiological mechanisms involved.

2.2.1 Cardiac parasympathetic measures

The autonomic nervous system regulates cardiovascular responses through activation of sympathetic and parasympathetic neural pathways. Sympathetic activity is responsible for instigating flight-or-fight responses, however also regulates exercise induced responses, acting to increase heart rate (HR) and blood pressure, redirect cardiac output to the working muscles via vasoconstriction, and instigate neuroendocrine stress responses. Conversely, parasympathetic activation stimulates rest-and-digest responses, and is responsible for the inhibition of HR, vasodilation, and blood pressure reduction, and is withdrawn during exercise with return occurring during the post-exercise recovery period (Aubert, Seps, & Beckers, 2003). This return of parasympathetic activity is referred to as cardiac parasympathetic reactivation and is reflective of homeostatic recovery following exercise, which has also been found to be associated with improved performance in athletes (Garet et al., 2004). Although the physiological mechanisms underlying cardiac parasympathetic reactivation following exercise are not wholly

known, its recovery kinetics appear to parallel many physiological systems involved in recovery from exercise including the thermoregulatory and vascular systems (Aubert et al., 2003; Stanley et al., 2013). Although, glycogen repletion and recovery of the neuromuscular system do not appear to concur with autonomic recovery (Delp & O'Leary, 2004). Nevertheless, the monitoring of cardiac parasympathetic reactivation provides a practical and relatively comprehensive measure of the body's recovery to its pre-exercise homeostatic state, particularly following high-intensity exercise (Seiler et al., 2007).

Cardiac parasympathetic assessment is achieved via the non-invasive and highly individualised measures of HRV and HRR. Heart rate variability describes the variance in time between successive heartbeats, with greater levels of parasympathetic activation reflected as greater variations in the time between beats. Recording of HRV encompasses detection of the time period between adjacent R-R intervals of a QRS complex during an electrocardiogram trace. Multiple methods of analysis exist for HRV, utilising time and frequency domain analysis, and non-linear methods—each describing differing physiological mechanisms (Task-Force, 1996). Time domain indices are regarded as the most appropriate analysis method for monitoring recovery in athletes, specifically rMSSD (square root of the mean of the sum of the squares of differences between adjacent R-R intervals) and SD1 (standard deviation of instantaneous beat-to-beat R-R variability measured from Poincaré plots) (Buchheit, 2014). These indices have the lowest coefficient of variation (CV) in terms of test re-test reliability (Al Haddad et al., 2011) and are less sensitive to the influence of respiratory rate, making them practical for the daily monitoring of athletes (Penttilä et al., 2001).

Heart rate variability measurements taken during resting conditions (i.e. after waking or a period of rest allowing HR stabilisation) are recommended for assessing parasympathetic activity in athletes (Buchheit, 2014). However, post-exercise measurement of HRV provides insight into the mechanisms driving parasympathetic reactivation. These mechanisms include blood pressure regulation, baroreflex activity, and metaboreflex response (Stanley et al., 2013). Thus, post-exercise HRV provides an assessment of homeostatic perturbation, with greater post-exercise stress responses reflecting lower HRV indices. Importantly, post-exercise HRV is influenced by the metaboreflex, so the use of exercise intensities bellow the first ventilatory threshold (VT_1) are optimal for the assessment of true autonomic regulation (Buchheit, Papelier, Laursen,

& Ahmaidi, 2007). Finally, assessment of both resting and post-exercise HRV requires approximately 180-300 s of HR data, and can be repeated throughout the recovery period (Stanley et al, 2013).

Heart rate recovery is reflective of sympathetic withdrawal following exercise, and can be used to track parasympathetic reactivation when repeated throughout the post-exercise recovery period. Physiological mechanisms influencing HRR include cardiac output and blood pressure regulation, as well as metaboreflex stimulation (Buchheit et al., 2007). However surprisingly, a lack of association has been found between HRR and post-exercise changes in these mechanisms (Buchheit, Al Haddad, Mendez-Villanueva, Quod, & Bourdon, 2011). As with post-exercise HRV measures, submaximal intensities of 3-5 min duration are recommended, allowing for HR stabilisation and inhibition of metaboreflex influence (Cerretelli & Di Prampero, 1971). Methods of analysis include assessment of the absolute change in post-exercise HR over a period of time (commonly 60 s), and signal modelling via linear, exponential, or mono-exponential models to derive parameters including the time-constant and asymptotic value (Perini et al., 1989). However, analysis of the absolute change in HR presents the more practical and reliable method for HRR assessment, considering its reflection of cardiac parasympathetic outflow (Kannankeril, Le, Kadish, & Goldberger, 2004), lower CV (Al Haddad et al., 2011), and the required equipment and time consuming nature of signal modelling methods. Practical considerations for implementing HRV and HRR recording include standardising body position, as standing, seated, and supine positions have been shown to have different effects on parasympathetic activity (Buchheit, Al Haddad, Laursen, & Ahmaidi, 2009). Environmental conditions should also be controlled, given the influence of light, noise and movement on the data obtained from these measures (Task-Force, 1996). Finally, reliability studies reveal post-exercise HRV to have a greater reliability (CV ~12.3) than HRR (CV ~25.7) (Al Haddad et al., 2011).

2.2.2 Endocrine measures

The endocrine system exerts precise control over various functions of the body via the regulation and release of hormones in order to maintain the homeostatic state. In response to exercise, the hypothalamus regulates the release of a number of hormones including cortisol and testosterone (Adlercreutz et al., 1986; E. Hill et al., 2008). These hormones in turn are responsible for the exercise induced changes in macronutrient release,

catabolism, and blood glucose level (Munck, 1971). Furthermore, they provide a measurable index of the post-exercise stress response when measured repeatedly throughout the post-exercise period (Adlercreutz et al., 1986; Papacosta & Nassis, 2011).

Cortisol is a glucocorticoid secreted from the adrenal cortex. Providing a marker of catabolic status, cortisol plays an important role in the up-regulation of glucose synthesis, protein catabolism, gluconeogenesis, lipolysis, and inhibition of cellular glucose uptake and oxidation (Eisenstein, 1973; Munck, 1971; O'Connor & Corrigan, 1987). Hypothalamic stimulation of the anterior pituitary gland leading to the vascular release of adrenocorticotropic hormone (ACTH) is responsible for the stimulation of steroid producing cells in the adrenal cortex, resulting in the release of cortisol into the circulation. This process is down-regulated via a negative feedback loop, whereby circulating cortisol acts to inhibit further ACTH and corticotropin releasing hormone (the mechanism of hypothalamic anterior pituitary axis stimulation) secretion (Tsigos & Chrousos, 2002). Simply, a rise in cortisol levels leads to down-regulation of the signal cascade, resulting in a decrease in cortisol secretion. Cortisol release is dependent on exercise intensity and duration, circadian rhythm, nutritional and fitness status (E. Hill et al., 2008; Suay et al., 1999). Cortisol measures are achieved via blood or salivary samples, with significant correlations existing between these measures at both rest and following exercise of varying intensities (Lippi et al., 2009). Peak concentrations occur 20 and 30 min post-exercise for blood and salivary samples respectively (Acevedo et al., 2007; O'Connor & Corrigan, 1987). Furthermore, salivary cortisol has been found to provide the more valid measure of circulating cortisol levels and hypothalamic-pituitary-adrenal (HPA) axis activity (Crewther, Cronin, Keogh, & Cook, 2008), as well as being the more practical field testing measure (Papacosta & Nassis, 2011).

Testosterone is also a steroid hormone—from the androgen group, and provides a marker of anabolic status. Responsible for up-regulating protein synthesis, down-regulating protein catabolism, neuromuscular structural changes, and the stimulation of growth hormone release (B. P. Brooks et al., 1998; Giustina & Veldhuis, 1998; Urban et al., 1995). Testosterone also acts to restore force production capacity by increasing neurotransmitter release and altering the neuromuscular junction (Nagaya & Herrera, 1995). Secretion of testosterone by reproductive organs is stimulated by the release of luteinising hormone and follicle stimulating hormone from the anterior pituitary gland, which in turn is stimulated by the hypothalamus and regulated via negative feedback

loops (Loebel & Kraemer, 1998). Testosterone is supressed following strenuous exercise, with its recovery signalling the return of an anabolic state following exercise induced catabolism (Adlercreutz et al., 1986). Due to its inverse response to cortisol, the testosterone/cortisol (T/C) ratio is commonly used as a post-exercise marker of recovery with a decrease in T/C ratio greater than 30% indicative of a catabolic state (Adlercreutz et al., 1986). As with cortisol, testosterone release follows a circadian rhythm, and can be assessed via blood and salivary samples throughout the post-exercise period. However, salivary samples have been shown to have a closer correlation with testosterone bioavailability and be more sensitive for assessing stress response to exercise (Crewther et al., 2008; Morley et al., 2006).

2.2.3 Performance measures

Following strenuous exercise of either high-intensities or long durations, subsequent exercise performance capacity has been found to be subdued (Krustrup et al., 2006; Mohr et al., 2010). This post-exercise fatigue effect is believed to be induced by several mechanisms including the accumulation of metabolites (Cairns, 2006), depletion of adenosine triphosphate (ATP) and phosphocreatine (PCr) stores (Karatzaferi, De Haan, Ferguson, Van Mechelen, & Sargeant, 2001), exercise induced muscle damage and inflammation (Cheung, Hume, & Maxwell, 2003), and reduced muscle glycogen availability (Jentjens & Jeukendrup, 2003). Measures of post-exercise performance recovery are usually in the form of time-trials, time-to-exhaustion, or strength tests; however, these are typically highly fatiguing and not practical for the assessment of recovery status in athletes performing frequent training sessions. A more practical means of assessing performance recovery is via peak and mean power achieved during a maximal 30 s modified Wingate test (Riechman, Zoeller, Balasekaran, Goss, & Robertson, 2002). When performed in competitive rowers mean power produced in this test has been found to predict up to 75.7% of the variance in 2000 m rowing ergometer test time, with peak power also demonstrating a strong relationship ($r = -0.85$, $p < 0.01$) with 2000 m performance (Riechman et al., 2002). The predictive ability of the modified Wingate test for 2000m ergometer performance is in part explained by the physiological demands of the tests, whereby anaerobic capacity and strength are suggested to play an important role in rowing race performance (Reilly, Secher, Snell, & Williams, 1990). Thus, the modified Wingate test provides a quick and valid, yet easily administered assessment of performance recovery in rowers.

Submaximal HR (HRex) at consistent exercise intensities provides another valuable measure for the assessment of performance within an athlete. Heart rate during continuous exercise is closely related to oxygen consumption and thus is a commonly observed adaptation to endurance training (Andrew, Guzman, & Becklake, 1966), whereby lower HRex at a consistent intensity generally indicates improved aerobic capacity (Mann, Lamberts, & Lambert, 2013). Furthermore, decreases in HRex have been found to have large and very large correlations with improvement in high intensity exercise performance (Buchheit, Chivot, et al., 2010; Buchheit et al., 2008; Lamberts, 2013). It is proposed that decreases in HRex in association with increases in aerobic capacity are due to reductions in sympathetic activity (Borresen & Lambert, 2008). Conversely, higher HRex are reflective of a reduced capacity for performance, with chronically increased HRex indicative of detraining, non-functional overreaching or overtraining (Hedelin, Kenttä, Wiklund, Bjerle, & Henriksson-Larsén, 2000; Jeukendrup et al., 1992). Therefore, following strenuous exercise HRex is expected to reflect the post-exercise fatigue response illustrated in the super-compensation curve, with recovery and rebound of HRex occurring as super-compensation is achieved and performance capacity is increased. Assessment of HRex typically requires 3-4 minutes of submaximal exercise for HR stabilisation to occur (Cerretelli & Di Prampero, 1971), with the average HR over the last 60-120 s generally taken. However, caution should be taken to standardize measurement protocols when assessing HRex in sports such as rowing, where an absence of HR stabilisation has been found to occur at submaximal intensities (Hartwell, Volberding, & Brennan, 2015).

2.2.4 Psychometric measures

Subjective stress is an important factor in recovery and is acknowledged as being a major contributor to under-recovery and under-performance (Kellmann, 2010). Thus, the monitoring of athlete's mood, general health and well-being, perceived physical recovery and fatigue status, and external life stressors provides important insight into factors effecting the recovery process and readiness to train. A number of psychometric monitoring tools exist in the form of questionnaires that can be applied across sporting modalities. These include the Profile of Mood States (POMS) (Mac Nair, Lorr, & Droppleman, 1971), the Recovery-Stress Questionnaire for Athletes (REST-Q Sport) (Kellmann, Altenburg, Lormes, & Steinacker, 2001), the Daily Analysis of Life Demands

in Athletes (DALDA) (Rushall, 1990), and the recovery questionnaire (REC-Q) (Halson et al., 2008). Interestingly, a recent review has found subjective measures to be more sensitive to acute changes in training load than physiological measures (Saw et al., 2015). This lack of association between subjective and physiological measures highlights the need for a range of recovery measures in the assessment of an athlete's readiness to train, incorporating that of a complementary holistic approach. Psychometric measures evaluating specifically athletes, across multiple constructs have been shown to provide the best reflection of performance capacity (Grove et al., 2014). However, for the practicality of assessing readiness to train following a single exercise bout measures should be quick and easily implemented in order to be performed daily.

2.3 Factors contributing to recovery time course

Given the optimisation of training adaptation occurs when the appropriate balance between fatigue and recovery is achieved, knowledge of specific recovery time-courses following various types of training would be highly valuable. However, the magnitude of acute post-exercise deviation and the time required for homeostatic return is not only dictated by the exercise stimulus, but also other factors. Cardiovascular fitness (Hautala et al., 2001; Seiler et al., 2007), the nature of the exercise stimulus (Niewiadomski et al., 2007; Seiler et al., 2007), training history (Garrandes et al., 2007; Otsuki et al., 2007), energetic profile (Del Rosso et al., 2016; McDonald et al., 2014), and lifestyle factors such as sleep (Samuels, 2009), nutrition (Hawley, Burke, Phillips, & Spriet, 2011), and psychological stress (Perna & McDowell, 1995) are factors that play a role in determining the time-course of homeostatic return. Thus, the following section will discuss the influence of these factors on the magnitude of post-exercise homeostatic perturbation, time-course of homeostatic return, and the mechanisms involved. Measures of parasympathetic activity will be favoured for the assessment of recovery in this section due to its comprehensive reflection of the integrated systems involved in the recovery process (Aubert et al., 2003). However, given the relatively scarce nature of the literature assessing homeostatic return throughout the post-exercise recovery period (i.e. 24-72 h post-exercise), the magnitude of homeostatic perturbation, as well as endocrine, muscle glycogen repletion, and performance recovery measures will be considered.

2.3.1 Cardiovascular fitness

Physiological and anatomical adaptations to long-term athletic training are numerous and influence the response and recovery time course following exercise. Endurance training alters haemodynamic loading of the heart at rest, as well as during submaximal exercise (Rost & Hollmann, 1983). These changes are induced by the heart's improved efficiency for the ejection of blood, as per the Frank-Starling relationship (G. A. Brooks, Fahey, & White, 1996). This is accomplished via increases to left ventricular end-diastolic volume, cardiac wall thickness and mass resulting in enhanced stroke volume and cardiac output (Ehsani, Hagberg, & Hickson, 1978; Wilmore et al., 2001). Furthermore, endurance training reduces catecholamine response to submaximal exercise, in turn subduing the tachycardic effect and therefore HRex (Orizio et al., 1988). Given the effect of autonomic balance on HR, the trainability of parasympathetic predominance presents a topic of interest in the literature, with findings showing promise for a positive effect on autonomic functioning following endurance training in previously untrained individuals (Hautala et al., 2003; Mourot et al., 2004; Yamamoto, Miyachi, Saitoh, Yoshioka, & Onodera, 2001). This is supported by studies showing well-trained individuals to demonstrate higher indices of cardiac parasympathetic activity than untrained controls (Buchheit & Gindre, 2006; Davy, Miniclier, Taylor, Stevenson, & Seals, 1996; Rennie et al., 2003).

Endurance training induced adaptations are associated with the enhanced time-course for homeostatic return observed in individuals with more favourable levels of cardiovascular fitness (Dixon, Kamath, McCartney, & Fallen, 1992; Hautala et al., 2001; Seiler et al., 2007; Stanley et al., 2013). Such findings likely reflect the enhanced blood flow dynamics (regulated by autonomic balance) associated with endurance training, which play an important role in post-exercise metabolite clearance, thermoregulation, glycogen repletion, and baroreflex response. Moreover, endurance training induced adaptations are likely related to the association between enhanced post-exercise cardiac parasympathetic reactivation and readiness to train (Lamberts et al., 2010). It therefore appears that individuals with greater indices of cardiovascular fitness present a superior physiological state to not only return homeostatic function but also illustrate markers of homeostatic return (via a greater parasympathetic predominance) (Huang et al., 2007).

Studies comparing the time course of parasympathetic reactivation following a single exercise bout in trained and untrained individuals are extremely limited (*Table 2.1*). Nevertheless, highly trained athletes (maximal oxygen uptake (VO_2max) 75 $ml \cdot kg^{-1} \cdot min^{-}$

[1]) demonstrated a 60-90 min faster recovery of parasympathetic control following a standardised high-intensity interval training session than trained athletes (VO_2max 60 $ml \cdot kg^{-1} \cdot min^{-1}$) (Seiler et al., 2007). This led the authors to question whether the observed accelerated autonomic recovery associated with enhanced aerobic capacity is a long-term adaptation to training, or rather an inherent trait of successful athletes allowing them to better cope and adapt to training stimuli. The concept that cardiac parasympathetic modulation is a determinant of training response is supported by the work of Hautala et al. (2003) who found baseline autonomic status to account for 27% of the change in peak oxygen uptake (VO_2peak) following an 8 wk training intervention in previously sedentary individuals. Additionally, a comprehensive review by Stanley et al. (2013) analysed the literature examining time-course of post-exercise parasympathetic reactivation. When adjusted for training intensity and duration, highly trained athletes were found to have reduced post-exercise suppression and faster recovery of parasympathetic activity compared to moderately trained and untrained, with untrained individuals having the longest time course of parasympathetic reactivation (Stanley et al., 2013). Consequently, more research in this area is needed to confirm the relationship between the time-course of parasympathetic reactivation after exercise and cardiovascular fitness status observed in these studies.

Cardiovascular fitness status is also proposed as responsible for enhancing metabolite clearance following anaerobically fatiguing exercise stimuli. The theoretical reasoning for this is two-fold: 1) athletes with greater aerobic capacities stress less non-oxidative energy sources and thus generate reduced metaboreflex responses (Buchheit et al., 2007); and 2) blood flow adaptations to aerobic training enhance the ability for post-exercise metabolite clearance, heat dissipation, and substrate delivery (Tomlin & Wenger, 2001). This is supported by adaptations to aerobic enzyme concentration, mitochondrial structure (Holloszy & Coyle, 1984), myoglobin concentration, capillarisation (Saltin & Rowell, 1980), and blood and haemoglobin volume (Kjellberg, Rudhe, & Sjostrand, 1949) associated with endurance training. As well as observations of reduced muscle and blood lactate levels at the same submaximal workload following endurance training (Karlsson & Saltin, 1971), and the strong correlation between VO_2max and the rate of oxygen uptake during repeated supramaximal intervals (Tomlin, 1998).

Furthermore, cardiovascular fitness is associated greater rates of post-exercise glycogen synthesis. Endurance training induced adaptions including increased GLUT-4

concentration (Greiwe et al., 1999), improved insulin signalling (Kirwan et al., 2000), glycogen synthase activity (Hickner et al., 1997), and blood flow contribute to enhance muscle glycogen uptake and synthesis (Greiwe et al., 1999). Furthermore, it appears individuals presenting lower indices of cardiovascular fitness demonstrate greater exercise induced muscle glycogen depletion (Greiwe et al., 1999; Hickner et al., 1997) as well as a reduced ability for fat oxidation (Hetlelid, Plews, Herold, Laursen, & Seiler, 2015), thus influencing a greater degree of post-exercise homeostatic perturbation. A study by Hickner et al. (1997) examined the influence of cardiovascular fitness on muscle glycogen synthesis following a glycogen depleting exercise stimulus. The authors found rates of muscle glycogen repletion two times faster in trained (VO_2max 60 ml·kg^{-1}·min^{-1}) subjects than untrained (VO_2max 38 ml·kg^{-1}·min^{-1}) 6 h post-exercise. Time for muscle glycogen content to return to resting levels was not assessed in the study by Hickner et al. (1997), and as with other recovery measures, limited comparisons of recovery time-course exist between trained and untrained individuals. Thus, the proposed advantageous influence of cardiovascular fitness on recovery time-course following a single exercise bout are largely theoretical, with further study in this area required to determine whether an association between time-course of homeostatic return following exercise and cardiovascular fitness status exists.

Table 2.1 Studies assessing the influence of cardiovascular fitness on post-exercise recovery time-course.

Study	Participants; training status	Training performed	Recovery measure	Measurement timing	Time to return to baseline
R. Fry et al. (1991)	14 M Varied fitness levels VO_2max: 42.6-75.4	25 x 1 min running at 1km·h^{-1} bellow v VO_2max, 2 min rest	Plasma cortisol and testosterone via venepuncture following 10 min resting	Pre, and 10 min, 2, 4, 8, and 24 h post-exercise	Cortisol: 2 h Testosterone: >24 h No association found with VO_2max.
Hautala et al. (2001)	10 M Varied fitness levels VO_2max: 39.5-58.6	75 km cross-country skiing race at 87 ±2.8% HRmax	HRV (Ln HF); 48 h continuous recording	Continuously for 24 h pre-exercise and 48 h post-exercise	8.2 ±5.4 h Range: 0-14 h Earlier recovery was strongly correlated with higher VO_2max values ($r = 0.745$, $p < 0.02$).
Seiler et al. (2007)	a.) 9 M Highly trained VO_2max: 75.0 ±5.0 b.) 8 M Trained VO_2max: 60.0 ±5.0	60 min running including 6 x 3 min at 96% VO_2max, 2 min of recovery	HRV (rMSSD); 5 min supine	Pre, and 5, 15, 30, 60, 90, 120, 180, and 240 min post-exercise	a.) 30 min b.) 120 min

Studies observing return to baseline presented only. Values presented as mean ± standard deviation. M – male; VO_2max – maximal oxygen consumption (ml·kg^{-1}·min^{-1}); v VO_2max – velocity at VO_2max; HRV – heart rate variability; HRmax – maximal heart rate; Ln HF – natural logarithm of the high-frequency spectral power (0.15-0.4 Hz); rMSSD – square root of the mean of the sum of the squares of differences between adjacent R-R intervals.

2.3.2 Training stimulus

The magnitude of homeostatic stress and subsequent time-course of recovery induced by an exercise bout is dependent on the nature of the stimulus performed (*Table 2.2*). The time-course of autonomic recovery following a training session has been shown to be longer following training of higher intensities, with the return of parasympathetic activity requiring 24 h following low intensity aerobic training, 24-48 h following threshold intensities and at least 48 h following high intensities (Stanley et al., 2013). This is related to the correlation of parasympathetic activity with post-exercise blood epinephrine (Perini et al., 1989), blood lactate (Buchheit, Al Haddad, et al., 2011), plasma acidosis (Buchheit, Chivot, et al., 2010), and arterial oxygenation concentrations (Ba, Delliaux, Bregeon, Levy, & Jammes, 2009). As well as post-exercise plasma volume changes (Buchheit, Laursen, et al., 2009), reflecting metabolite accumulation and homeostatic perturbation on the autonomic response. Studies examining the influence of intensity and duration on recovery time-course (time to return to baseline values) are presented in *Table 2.2*. Studies assessing cardiac parasympathetic activity have been selected for presentation due its reflection of multiple integrated mechanisms responsible for recovery (Stanley et al., 2013).

The aforementioned review by Stanley et al. (2013) also analysed the effect of intensity on the magnitude of parasympathetic suppression 1 h post exercise, finding the acute deviation from baseline was related to recovery time course, with higher intensities eliciting greater suppressions of parasympathetic activity. Additionally, this review identified low intensity exercise to demonstrate a decrease in cardiac parasympathetic activity occurring relatively soon (24-48 h post-exercise) after its initial recovery (Stanley et al., 2013). This subsequent reduction in parasympathetic recovery is indicative of a rapid onset of involution following the achievement of super-compensation (*Figure 2.1*). These findings are supported by the work of Seiler et al. (2007), who identified VT_1 to be the threshold intensity for increases in both the magnitude of parasympathetic suppression and time-course of parasympathetic recovery. Whereas exercise performed at intensities bellow VT_1 have not been found to influence either of these variables (Plews, Laursen, Kilding, & Buchheit, 2014; Seiler et al., 2007). Further, response in endocrine markers of stress magnitude appear to be minimal following exercise performed bellow VT_1 (Jacks et al., 2002; Nieman et al., 1999). With Nieman et al. (1999) reporting little change in blood levels of cortisol, growth hormone, epinephrine, and norepinephrine despite 2 h of rowing.

Little, if any influence of exercise duration on time course of autonomic recovery has been found when controlling for intensity (Seiler et al., 2007; Stanley et al., 2013). Studies examining autonomic recovery following ultra-endurance performance agree with the findings of Stanley et al. (2013), whereby parasympathetic recovery occurred within 24 h post-exercise, regardless of the longer duration of these events (271-370 min) (Bernardi et al., 1997; Hautala et al., 2001). However, HRV following Ironman performance (659 min) was not found to return to baseline values by 24 h, rather requiring 72h (Gratze et al., 2005). Although, the extent of this prolonged duration may be due to the sampling frequency, as no measures were obtained between these two time points.

Interestingly, Hynynen et al. (2010) found increased nocturnal HRV suppression following marathon running (217 ±28 min) compared to a moderate training run (52 ±26 min), where both exercise intensities were performed bellow VT_1. Kaikkonen et al. (2010) also found suppression of post-exercise HRV to be influenced by training duration, with 14km of running producing a substantially greater degree of post-exercise HRV suppression comparative to 3 km performed at the same submaximal intensity (60% velocity (v) at VO_2max). Although the same authors found no difference in post-exercise HRV suppression following 3.5 km and 7 km of running performed at either low (50% v VO_2max) or moderate intensity (63% v VO_2max) (Kaikkonen, Nummela, & Rusko, 2007), suggesting a threshold exists for durational influence on HRV suppression. Unfortunately, neither Hynynen et al. (2010); Kaikkonen et al. (2010); nor Kaikkonen et al. (2007) examined the time-course for HRV return to baseline following these exercise bouts. Consequently, the literature examining the effect of exercise duration on autonomic recovery when intensity is controlled proves limited and inconclusive and further research in this area would be beneficial in the validation previous findings.

2.3.2.1 Inter-individual variation in response to training intensity

Standardization of the training stimulus in most studies observing inter-individual variation involves the tightly regulated prescription of relative exercise intensities based on percentage VO_2max or maximal heart rate (HRmax). Although, the true standardization of exercise intensity prescribed by these methods is debated in the literature, with evidence of large inter-individual variation occurring in metabolite response at moderate and high relative intensities (Bouchard et al., 1999; Lortie et al.,

1984; Vollaard et al., 2009). The significance of this variation in metabolite response lies in the subsequent difference in the homeostatic stress of the exercise stimulus, which likely reflects inter-individual variation in the resultant recovery time course.

To date, it appears there is no research examining inter-individual variation in homeostatic recovery time-course following a single exercise bout. However, research examining the overall adaptive response to standardized training regimes have found inter-individual variation to be a product of variation of the metabolic exercise stimulus (Gaskill et al., 2001; McPhee et al., 2011; Vollaard et al., 2009). For example, Vollaard et al. (2009) reported that improved performance following training correlated to exercise induced responses in muscle lactate concentration and acytl carnitine. Furthermore, the authors found a large degree of inter-individual variation in the training response of metabolic factors to submaximal exercise performed at 70% VO_2max, including up to a 400% difference in muscle lactate concentration between individuals. These insights prompted the authors to caution against the practice of standardizing exercise intensity to a set percentage of VO_2max, given such large inter-individual responses.

Given the link between adequate recovery between exercise bouts and aerobic capacity adaptation (Hooper, Mackinnon, Gordon, & Bachmann, 1993) it is possible that a cascade effect exists. Whereby variability in the individual homeostatic stress stimulus relates to variability in the individual time-course for homeostatic recovery, and resultant accumulated adaptive response to the exercise program. This is exemplified by differences between endurance and power trained athletes in acute post-exercise neuromuscular fatigue and metabolic responses (Garrandes et al., 2007; Paavolainen et al., 1994). However, no evidence yet exists to substantiate this proposition for athletes presenting similar training histories, and research in the area of inter-individual variation in recovery time-course is first required. Nevertheless, caution should be taken when conducting research comparing the magnitude of homeostatic stress and subsequent recovery time-course dependant on exercise intensity, due to the observation of inter-individual variation following highly standardised exercise intensity prescription. Standardising exercise intensity relative to ventilatory threshold indices is recommended for future research examining the effect of intensity on recovery time-course following exercise (Mann et al., 2014; Vollaard et al., 2009).

Table 2.2 Studies assessing the influence of exercise intensity and duration on post-exercise recovery time-course of parasympathetic activity.

Study	Participants; training status	Exercise performed	Recovery measure	Measurement timing	Time to return to baseline
Terziotti, Schena, Gulli, and Cevese (2001)	12 M Untrained	a.) 20 min cycling at 80% VT_2 b.) 20 min cycling at 50% VT_2	HRV (Ln HF); 10 min seated; respiration controlled	Pre-exercise, and 15 min, 1 and 3 h post-exercise	a.) 1 h b.) 1 h
Mourot et al. (2004)	10 M Moderately trained	a.) 9 x 4 min cycling at VT_1 power, 1 min at Pmax b.) 50 min cycling at VT_1	HRV (Ln HF); 5 min supine	Pre-exercise, and 1, 24, and 48 h post-exercise	a.) 24 h b.) 1 h
Parekh and Lee (2005)	13 M Untrained	a.) 20 min running at 80% VO_2R b.) 25 min running at 50% VO_2R	HRV (Ln HF); 5 min supine	Pre-exercise, and 10, 15, 20, 25 min post-exercise	a.) 25 min b.) 10 min
Niewiadomski et al. (2007)	8 M Untrained	a.) 2 x 30 s maximal cycling (Wingate test), 3 min rest b.) 30 min cycling at 85% HRmax	HRV (Ln HF); 18 min supine; respiration controlled	Pre-exercise, and 1, 24, and 48 h post-exercise	a.) 48 h b.) 1 h
Seiler et al. (2007)	9 M Highly trained	a.) 6 x 3 min running at 95% VO_2max b.) 30 min running bellow VT_1, 30 min at 80-85% HRmax	HRV (Ln HF); 5 min supine	Pre-exercise, and 0, 10, 25, 55, 85, 115, 175 and 235 min post-exercise	a.) 30 min b.) 30 min

Study	Participants; training status	Exercise performed	Recovery measure	Measurement timing	Time to return to baseline
Seiler et al. (2007)	9 M Highly trained	c.) 60 min running bellow VT_1 d.) 120 min running bellow VT_1	HRV (Ln HF); 5 min supine	Pre-exercise, and 0, 10, 25, 55, 85, 115, 175 and 235 min post-exercise	c.) 5 min d.) 5 min
Kaikkonen, Rusko, and Martinmäki (2008)	8 M Moderately trained	a.) 7 x 3 min running at 93% v VO_2max b.) 7 x 3 min running at 85% v VO_2max c.) 21 min running at 80% v VO_2max d.) 21 min running at 85% v VO_2max	HRV (Ln HF); 1-2 min seated	Pre-exercise, and 0, 8, 18 and 28 min post-exercise	No return to baseline observed for any of the intensities

M – male; HRV– heart rate variability; Ln HF – natural logarithm of the high-frequency spectral power (0.15-0.4 Hz); VT_1 – first ventilatory threshold; VT_2 – second ventilatory threshold; HRmax – maximal heart rate; VO_2max – maximal oxygen uptake; VO_2R – VO_2 reserve; v VO_2max – velocity at VO_2max; HRmax – maximal heart rate; Pmax – maximal achievable power.

2.3.3 Training history and energetic profile

The time course for homeostatic return following a single exercise bout may be influenced by training history. Although, research examining post-exercise recovery time-course is scant, the magnitude of post-exercise stress response varies greatly between athletes exhibiting differing energetic profiles (*Table 2.3*). Specifically, endurance-trained athletes have been found to demonstrate reduced neuromuscular fatigue and metabolic responses following high-intensity exercise compared to power-trained athletes (Garrandes et al., 2007; Paavolainen et al., 1994). Furthermore, the responsiveness of measures used to asses recovery have been found to be influenced by training status, with Heffernan, Fahs, Shinsako, Jae, and Fernhall (2007) finding improved recovery in HRR, but not resting HRV, following 6 wk of resistance training in previously untrained individuals.

Differences in post-exercise homeostatic stress are attributed to the adaptive response of neuromuscular and metabolic function to training (Garrandes et al., 2007). Training history has been shown to dictate muscle fibre type distribution, with international level endurance trained athletes (5,000 m and marathon runners) demonstrating a greater proportion of type 1 fibres (69.4%), than their power trained (100 m sprinters) counterparts (27.4%) (Costill et al., 1976). Type II fibres are associated with higher resting concentrations of ATP, phosphocreatine, and glycogen, however exhibit a larger exercise induced depletion of these substrates post-exercise, thus reflecting their enhanced ability to induce homeostatic perturbation (Casey, Constantin-Teodosiu, Howell, Hultman, & Greenhaff, 1996; Greenhaff et al., 1994). Furthermore, a study by Hamada, Sale, MacDougall, and Tarnopolsky (2003) identified subjects with a predominant distribution of type II fibres to be associated with greater decreases in maximal force production following a bout of fatiguing exercise, compared to subjects exhibiting a predominant distribution of type 1 fibres.

Long-term training stimuli are responsible for the conversion of muscle fibre types between type IIa and type IIb (Allemeier et al., 1994; Ingjer, 1979), as well as inducing changes in mitochondrial size, number and function (Holloszy & Coyle, 1984). Thus, training history partly explains the variance observed in anaerobic and aerobic contribution to exercise between endurance and power athletes, describing their energetic profile. Endurance athletes demonstrating greater aerobic energy system contributions to exercise are suggested to illustrate faster post-exercise recovery time-courses due to their

limited recruitment of type II muscle fibres, and therefore reduced homeostatic perturbation (Buchheit et al., 2012; Bundle et al., 2003; Del Rosso et al., 2016). Furthermore, adaptations responsible for enhancing aerobic contribution are also related to recovery, with adaptations to blood flow dynamics and muscle capillarisation increasing the capacity for post-exercise substrate delivery and metabolite clearance (Pringle et al., 2003).

The research examining time-course of homeostatic recovery following a single exercise bout in athletes presenting different energetic profiles is extremely limited. However, previous research has identified differences in acute performance (Garrandes et al., 2007) and autonomic (Del Rosso et al., 2016; McDonald et al., 2014; Otsuki et al., 2007) responses with differing energetic profiles. McDonald et al. (2014) identified that differences in the post-exercise HRR of cyclists was related to training background, with "anaerobically trained" track cyclists illustrating slower HRR than "aerobically trained" road cyclists. This is supported by a recent study by Del Rosso et al. (2016), who found anaerobic capacity to be related to post-exercise HRR in males, with participants demonstrating higher indices of anaerobic speed reserve exhibiting slower indices of HRR. While these studies provide insight, further knowledge of the influence of energetic profile on recovery time course would be beneficial for the individualisation of training programming, particularly in intermediate duration sports where athletes demonstrate a range of energetic contributions and therefore likely present differing recovery needs (D. W. Hill, 1999).

2.3.3.1 Energetic contributions in rowing

Energetic contribution refers to the contribution of anaerobic and aerobic energy sources to a specific exercise or performance task. Differences in energetic contribution may exist between participants performing the same exercise task and these differences are likely reflective of variation in training state, underlying physiology, and genetic influences (Bray et al., 2009). Intermediate duration sports of 3-8 min, such as rowing, require a large contribution of both the aerobic and anaerobic energy systems, with previous research demonstrating mean contributions of 84% aerobic and 16% anaerobic energy to a 2000 m rowing ergometer test (de Campos Mello et al., 2009; Russell, Rossignol, & Sparrow, 1998). Similarly, using the accumulated oxygen deficit (AOD) method (Medbo et al., 1988), Pripstein, Rhodes, McKenzie, and Coutts (1999) estimated mean

contributions of 88% aerobic and 12% anaerobic energy sources to the same test. The AOD is considered an accurate (Gastin, 1994), reliable and valid measure (Noordhof, De Koning, & Foster, 2010) for quantifying energy system contribution. Nevertheless, the assumptions of this method have attracted attention in the literature, with suggestions that the relationship between oxygen uptake and exercise intensity is not linear at supramaximal intensities, resulting in an increasing under-estimation of oxygen deficit as intensity increases (Bangsbo, 1998).

Older studies examining the energetic contribution in rowing performance report aerobic contributions of approximately 70% (Hagerman, Connors, Gault, Hagerman, & Polinski, 1978; Mickelson & Hagerman, 1981; Roth, Hasart, Wolf, & Pansold, 1983; Secher, Vaage, & Jackson, 1982). This difference is likely reflective of the method used to assess energetic contribution, whereby the excess post-exercise oxygen consumption (EPOC) accumulated on cessation of the test was used to estimate anaerobic contribution in these studies (Artioli et al., 2012). This method provides a measure of both alactic and anaerobic energy, whereas only total anaerobic contribution can be derived from the AOD method (Artioli et al., 2012; Medbo et al., 1988). However, the use of EPOC to determine energetic contribution has also been found to over-estimate anaerobic energy contribution (Hagerman, 1994), and proves more time consuming than the AOD method (Artioli et al., 2012). Furthermore, the studies by Hagerman et al. (1978); Mickelson and Hagerman (1981); and Secher et al. (1982) used constant load mechanical resistance rowing ergometers. Whereas de Campos Mello et al. (2009); Pripstein et al. (1999); and Russell, Rossignol, and Sparrow (1998) performed tests on the air resistance ergometers, whereby the increases in resistance are exponentially proportional to that of power output (Concept II Inc., Morrisville, VT), likely producing differences in energetic contribution in comparison to older studies.

Table 2.3 Studies examining the influence of energetic profile on the acute post-exercise autonomic response.

Study	Participants; energetic profile	Training performed	Response measure	Measurement timing	Variation from baseline
Otsuki et al. (2007)	a.) 12 M AnT athletes b.) 12 M AeR athletes c.) 12 M Untrained controls	8 min cycling at 40% VO_2max	HRR; time constant of HR decay	Throughout the 30 s post exercise period	a.) 69.1 ±4 s b.) 65.9 ±4.3 s c.) 94.4 ±9.2 s
McDonald et al. (2014)	a.) 9 M, 1 F AnT cyclists b.) 11 M, 4 F AeR cyclists	Incremental cycling test to exhaustion (2 min stages, 50 W increments)	HRR; Absolute change in HR	Throughout the 2 min post-exercise period	a.) 52 ±15 bpm b.) 64 ±11 bpm
Del Rosso et al. (2016)	a.) 13 M; high ASR (AnT) b.)14 M; low ASR (AeR)	6 x 40 m maximal running, 20 s recovery	HRR; Time to reach 63% HR reduction	Throughout the 5 min post-exercise period	a.) 104.8 ±22.9 s b.) 87.5 ±15.5 s

Values presented as mean ± standard deviation. M – male; F – female; AnT – anaerobic energetic profile; AeR – aerobic energetic profile; HRR – heart rate recovery; HR – heart rate; VO_2max – maximal oxygen uptake; ASR – anaerobic speed reserve.

2.3.4 Gender

Gender appears to have some influence on the time-course of recovery, with females demonstrating delayed cardiac parasympathetic reactivation following high-intensity exercise (Kiviniemi et al., 2010; Mendonca et al., 2010). Gender differences in circulating reproductive hormone concentrations and thermoregulation may influence recovery (Charkoudian & Joyner, 2004; Kenny & Jay, 2007), however research investigating the influence of these factors on time to recover is limited. Additionally, gender differences in autonomic balance reveal females to possess significantly greater indices of parasympathetic activity at rest than males (Mendonca et al., 2010; Yamasaki et al., 1996), with menstrual cycle fluctuation further enhancing parasympathetic activity during the follicular phase in females (Saeki, Atogami, Takahashi, & Yoshizawa, 1997; Sato, Miyake, Akatsu, & Kumashiro, 1995). However, a study by Mendonca et al. (2010) investigating differences in acute autonomic recovery following a 30 s Wingate test in sedentary males and females found a larger post-exercise LF/HF ratio increase in females. Furthermore, autonomic balance demonstrated a greater post-exercise change from baseline in females, indicating the greater indices of parasympathetic activity observed in females at rest are not retained following high-intensity exercise (Mendonca et al., 2010). Moreover, Kiviniemi et al. (2010) found females to demonstrate delayed recovery of autonomic balance compared to males following two consecutive days of exercise at 85% peak HR leading the authors to propose a sustained effect of the greater acute post-exercise hypotension, and reduced total peripheral resistance exists in females compared to males (Carter, Watenpaugh, & Smith, 2001; Kiviniemi et al., 2010). Additionally, females demonstrate a reduced ability to lower body temperature following exercise (Kenny & Jay, 2007), while following endurance exercise females have been shown to illustrate reduced inflammatory responses than that observed in males (Apple et al., 1987; Shumate, Brooke, Carroll, & Davis, 1979). Despite differences in the acute exercise response, further research is required to determine whether a gender effect exists in the time taken to recover to a pre-exercise state.

2.3.5 Lifestyle factors

Various lifestyle factors have been found to influence an individual's ability to recover from subsequent training stimuli, these include sleep (Samuels, 2009; Spiegel, Leproult, & Van Cauter, 1999) psychological stress (Perna & McDowell, 1995), and nutrition (Hawley et al., 2011). These factors influence both the pre- and post-exercise

physiological state, and thus are responsible for predisposing an individual to poor recovery prior to the exercise stimulus, as well as inhibiting post-exercise recovery dynamics (H. Chen, 1991; Fukuda & Morimoto, 2001; Ivy, 1998; Spiegel et al., 1999). Research regarding factors such as sleep and psychological stress on recovery time-course proves problematic due to ethical factors and difficulties concerning the quantification of these variables.

2.3.5.1 Sleep debt

Chronic sleep debt is associated with altered metabolic and endocrine function, illustrated by raised cortisol secretion and sympathetic activity, as well as reduced insulin sensitivity, and cognitive performance (Spiegel et al., 1999; Van Dongen, Baynard, Maislin, & Dinges, 2004). These factors likely slow parasympathetic reactivation and muscle glycogen repletion mechanisms involved in post-exercise recovery, predisposing athletes experiencing chronic sleep debt to disadvantageous recovery states. Conversely, sleep debt has been observed to negatively influence training tolerance and performance (Samuels, 2009; Skein, Duffield, Edge, Short, & Mundel, 2011), potentially acting as a protective mechanisms by reducing the magnitude of training induced stress. A study by Skein et al. (2013) found a complete night's sleep deprivation to negatively impact post-match recovery in rugby league players, with delayed recovery of counter-movement jump and reaction time performances 16 h post-match, compared to post-match performances following 8 h of night sleep. The authors associated these findings to the reduced levels of neural drive and increased biomarkers of muscle damage (creatine kinase and C-reactive protein) observed following the sleep deprivation condition, although no significant difference was observed in these variables between sleep conditions (Skein et al., 2013). Additionally, studies investigating 30 h of sleep deprivation compared to regular sleep patterns note increased measures of resting HR, plasma catecholamines, and blood pH following sleep deprivation, while diminished indices of HRmax, VO_2peak, and time to exhaustion following an incremental cycling test to exhaustion (H. Chen, 1991). As well as extended sprint time, diminished distance covered during self-paced exercise, resting muscle glycogen concentration, neural drive, and increased ratings of mood disturbance and psychological stress in team-sport athletes (Skein et al., 2011). On the other hand, appropriate and consistent levels of sleep are recommended as an advantageous post-exercise recovery strategy, with day time napping

suggested to enhance recovery processes in instances of night sleep loss (Marshall & Turner, 2016; Samuels, 2009).

2.3.5.2 Psychological stress

For the purpose of this review psychological stress encompasses subjective mental and emotional stressors not necessarily related to (but not excluding) training, such as social, work or study related stress. In healthy populations psychological stress has been found to prolong wound healing (Marucha, Kiecolt-Glaser, & Favagehi, 1998; Walburn, Vedhara, Hankins, Rixon, & Weinman, 2009), impair strength development (Bartholomew, Stults-Kolehmainen, Elrod, & Todd, 2008), reduce aerobic capacity adaptation (Ruuska, Hautala, Kiviniemi, Mäkikallio, & Tulppo, 2012), and reduce immune function following exercise in recreational runners (Rehm, Elci, Hahn, & Marshall, 2013). Furthermore, Clarkson and Hubal (2002) identified 9% of the variance in maximal isometric force production 1 h post strenuous resistance exercise to be explained by perceived psychological stress.

Regulation of HPA axis negative feedback loops are proposed to alter with exposure to psychological stress, resulting in a chronic increase in cortisol secretion (Fukuda & Morimoto, 2001). This is substantiated by the findings of Perna and McDowell (1995) who identified elite athletes experiencing higher levels of psychological stress to illustrate a 33.3% greater post-exercise salivary cortisol response and extended time course for return to baseline. Furthermore, the enhanced sympathetic activation and subsequent parasympathetic withdrawal associated with psychological stress is reflected by reduced HRV indices (Delaney & Brodie, 2000). Additionally, Stults-Kolehmainen, Bartholomew, and Sinha (2014) identified psychological stress as reducing maximal isometric force, perceived energy, fatigue and muscle soreness recovery rate of over a 96 h period following strenuous exercise. This finding was unrelated to exercise workload, fitness, and body composition. This information—in addition to current findings illustrating psychological measures to be more sensitive than object measures (Saw et al., 2015)—highlights the need for an integrated monitoring system, including psychometric measures alongside that of physical measures for the assessment of athlete recovery. Although, considering the attention given to psychological stress altering physiological function, its effect on post-exercise recovery time-course is relatively understudied.

2.3.5.3 Nutritional status

Conflicting evidence currently exists regarding the influence of nutritional status on post-exercise recovery. Traditional studies observe an association between glucose availability (enhanced via carbohydrate consumption during and post-exercise) and increased post-exercise muscle glycogen synthesis rates (Doyle, Sherman, & Strauss, 1993; Ivy, 1998; van Loon, Saris, Kruijshoop, & Wagenmakers, 2000). Additionally, post-exercise glycogen repletion has been shown to be improved with carbohydrate consumption immediately post-exercise compared to within a few hours post-exercise (Ivy et al., 2002; Ivy, Lee, Brozinick, & Reed, 1988). The underlying mechanisms of this observation are associated with the insulin related activation of muscle glycogen synthesis. Insulin enhances muscle glucose transport and up-regulates glucose synthase—a rate limiting enzyme involved in muscle glycogen synthesis (Bergstrom, 1962; Ivy & Holloszy, 1981). In contrast, more recent research demonstrates no difference in post-exercise muscle glycogen repletion patterns between elite endurance athletes who consumed either high carbohydrate (47.2 g) or low carbohydrate (4.3g) shakes following 3 h of submaximal treadmill running (Volek et al., 2016). Nevertheless, protein consumption following strenuous exercise has been found to stimulate protein synthesis, enhancing the repair and recovery process of muscle (Tipton, Ferrando, Phillips, Doyle, & Wolfe, 1999). Although it is not the focus of this section, readers are directed to reviews by Burke, Kiens, and Ivy (2004); Phillips (2004); and Tipton and Wolfe (2004) for further information regarding post-exercise nutritional recommendations.

2.4 Future directions

The measurement of autonomic balance in the post-exercise period provides a valuable assessment of homeostatic return, due to its practical, non-invasive and relatively comprehensive reflection of the physiological systems involved in recovery. As such, recent research exemplifies the benefit of programming subsequent bouts of training following individualised recovery—measured as the return of autonomic balance to pre-exercise levels. Lamberts et al. (2010), examined the HRR responses of 14 highly trained cyclists throughout a 4-week high intensity training program. The authors were able to retrospectively group the athletes by those who illustrated increased HRR or decreased HRR after the 4-weeks. The decreased HRR group demonstrated an attenuated improvement to mean power output over a 40km time-trial compared to the

improved HRR group. This led the authors to suggest that accumulated fatigue in response to decreased training tolerance was responsible for blunting the training effect, presumably resulting from greater homeostatic stress and therefore insufficient recovery time between exercise bouts, in comparison to their peers (Lamberts et al., 2010). This emphasizes the potential for cardiac parasympathetic assessment to guide the individualization of training programming on the basis of recovery status and readiness to train.

The benefit of training programming in accordance with recovery status is further documented by Kiviniemi et al. (2007), Kiviniemi et al. (2010), and Vesterinen et al. (2016) who used cardiac parasympathetic reactivation via HRV to individualize training. These studies compared groups of moderately trained athletes performing either conventionally prescribed training or HRV guided training, whereby participants performed high-intensity training sessions when morning resting HRV was high and low-intensity sessions when HRV was found to be low. This was prescribed on the basis of decreased parasympathetic activity reflecting insufficient recovery from previous training sessions, indicating an unfavorable physiological condition for high-intensity exercise (Hautala et al., 2001). And is further supported by Stanley et al. (2013) who speculates low-intensity training performed in instances of low parasympathetic activity (induced by a previous strenuous training session) may accelerate recovery, given autonomic super-compensation occurs within 24 h of low-intensity exercise.

Despite the HRV guided training groups performing less overall high-intensity sessions than the conventional training groups in these studies, the HRV guided groups demonstrated greater VO_2max improvement, maximal attainable workload (Kiviniemi et al., 2010), and running performance improvement (Vesterinen et al., 2016) following the training intervention. Leading the authors to discover greater adaptability to training evident when low-intensity training was performed in circumstances of attenuated vagal modulation of HR. Although training prescription based on recovery status and readiness to train is promising for the optimization of training programming and subsequent adaptation, the practicality of prescribing training based on daily HRV measures may prove difficult considering the complexity of this measurement (Buchheit, 2014). Therefore, identifying the recovery time-course dependent on the nature of the training stimulus and athlete's energetic profile may provide a more practical and applicable means of individualizing training programming based on recovery requirements.

Given the potentially wide variation in energy system contributions illustrated in successful athletes competing in intermediate duration sports (D. W. Hill, 1999), knowledge regarding the influence of energetic profile on post-exercise recovery time-course may prove valuable for the individualisation of training programming within a training squad. However, current research in this area is extremely limited, with studies focussing on the acute deviation from baseline rather than recovery time-course (Del Rosso et al., 2016; Garrandes et al., 2007; McDonald et al., 2014; Otsuki et al., 2007). Thus, further research is first needed to establish whether any influence of energetic profile on recovery time-course exists before recommendations can be made.

Additionally, previous studies examining the influence of exercise intensity on recovery time-course implement passive recovery periods (Kaikkonen et al., 2008; Mourot et al., 2004; Niewiadomski et al., 2007; Parekh & Lee, 2005; Seiler et al., 2007; Terziotti et al., 2001). It is therefore likely that the recovery time-courses identified in previous studies are not applicable in a real-world setting, whereby due to the high training frequencies of endurance sports, athletes are likely to perform subsequent training prior to achieving complete homeostatic recovery. As such, future research considering a real-world setting—where athletes continue their usual programmed training—would prove valuable for the development of recovery-guided training and its practical application.

2.5 Conclusion

This literature review has discussed the current knowledge regarding the measurement of recovery time-course following a single exercise bout for optimising the timing of subsequent training stimuli. Current research investigating the influence of energetic profile on recovery from exercise examines the acute post-exercise deviation from baseline only, with a gap in the literature identified regarding the influence of energetic profile on time to recover to baseline. Nevertheless, the limited evidence available indicates athletes presenting greater anaerobic energetic profiles illustrate greater acute post-exercise cardiac parasympathetic suppression and greater reductions in force production following strenuous exercise.

In addition, current findings regarding the influence of exercise intensity on recovery time-course illustrate a positive relationship between the two variables. With typical

durations for recovery demonstrating up to 24 h following low-intensity exercise, 24-48 h following threshold intensity exercise, and at least 48 h following high intensity exercise. Exercise duration has been found to have no influence on the time to recover of autonomic balance at intensities bellow VT_1, with a lack of clear evidence for a durational influence at higher intensities. However, a large limitation identified in the literature examining recovery time-course to date is the utilisation of passive recovery periods.

Finally, additional parameters including lifestyle factors and cardiovascular fitness have been shown to influence post-exercise recovery time-course. Although research examining the influence of cardiovascular fitness on recovery time-course is scarce, reduced post-exercise homeostatic perturbation and accelerated recovery of cardiac parasympathetic activity has been observed in individuals presenting higher indices of cardiovascular fitness. While factors including psychological stress and sleep debt appear to inhibit mechanisms regulating the recovery process. Thus highlighting the value of an individualised approach to training programming, with recent research exemplifying the benefit of recovery guiding training based on the return of cardiac parasympathetic activity to baseline levels.

Analysis of the current literature regarding the time-course of recovery following a single exercise bout has identified several factors that may prove valuable in informing training programming. Further investigation to determine whether the influence of energetic profile on acute post-exercise deviation extends to differences in the time-course of homeostatic recovery may provide a means of individualising training programming in order to maximise the adaptive response. Furthermore, the examination of recovery parameters throughout non-passive recovery periods would provide a more practically applicable assessment of recovery time-course, given successful athletes typically perform multiple training sessions per day (Fiskerstrand & Seiler, 2004). In addition, while autonomic balance provides a comprehensive view of homeostatic recovery, observation of the parallel time-course of alternative measures of recovery may provide insight into the underlying mechanisms driving the recovery process.

Chapter Three: Study One

The acute post-exercise deviation and recovery time-course to baseline of autonomic and performance parameters following various training intensities in highly trained rowers

3.1 Abstract

Purpose: To investigate the effects of different interval training (IT) sessions on the acute post-exercise deviation and recovery time-course to baseline of autonomic and performance parameters during a non-passive post-exercise period. ***Methods:*** This study employed a repeated measures crossover design. Ten male and three female highly trained rowers (VO_2peak 4.93 ±0.71 $L \cdot min^{-1}$) completed preliminary testing for physiological assessments in the week prior to the experimental trials. Experimental trials required participants to complete three IT sessions on the rowing ergometer, separated by seven days: 5 x 3.5 min, 4 min rest periods (VO_2); 10 x 30 s, 5 min rest periods (Glycolytic); and 5 x 10 min, 4 min rest periods (Threshold). Participants were instructed to perform intervals at the highest maintainable pace. Blood lactate and salivary cortisol were measured pre and 3 min or 30 min post-exercise respectively. Resting heart rate (HR) variability (HRV), post-submaximal exercise HRV (HRVex), submaximal HR (HRex), HR recovery (HRR), modified Wingate peak power and mean power, and subjective recovery (REC-Q) were measured pre and 1, 10, 24, 34, 48, 58, and 72 h post-exercise. ***Results:*** Differences in the acute deviation across IT sessions were either *trivial* or *unclear* for all recovery variables. HRVex demonstrated the longest time-course for baseline return: 37.8 ±14.2 h (mean ± CL) post-Threshold, 20.2 ±11.0 h post-Glycolytic, and 20.6 ±15.2 h post-VO_2 IT. *Very large* (r = 0.7-0.9, $p < 0.05$) relationships existed between acute deviation and recovery time-course in HRV, HRex, HRR, peak and mean power following Threshold and Glycolytic IT. ***Conclusion:*** Acute deviations from baseline were similar following Threshold, Glycolytic, and VO_2 IT in highly-trained male and female rowers. However, following resumption of training, return to a pre-exercise state is prolonged following Threshold compared to Glycolytic and VO_2 focused IT, suggesting a durational influence on recovery time-course exists at HR intensities reflective of $\geq VT_2$.

3.2 Introduction

Adaptation to training is maximised when the appropriate balance between training and recovery is achieved, allowing super-compensation to occur. High training loads with insufficient recovery periods compromise the adaptive ability, which overtime can manifest as non-functional overreaching and overtraining (Meeusen et al., 2013). Programming training is further complicated by the influence of factors including training intensity (Stanley et al., 2013), cardiovascular fitness (Seiler et al., 2007), gender (Kiviniemi et al., 2010), and subsequent exercise (Carter, Wilson, Watenpaugh, Smith, & Crandall, 2002) on the time-course of recovery following a single training session. Furthermore, the magnitude of acute post-exercise deviation from baseline is expected to correspond to the time required for physiological functioning to return to a pre-exercise state (Buchheit et al., 2007; Seiler et al., 2007), however few studies have investigated such a relationship (Niewiadomski et al., 2007; Parekh & Lee, 2005).

Multiple integrated physiological mechanisms are involved in the post-exercise return of homeostasis, which is characterised by several factors including neuromuscular recovery (Garrandes et al., 2007), muscle metabolite (Tesch, 1979) and cortisol clearance (Ahtiainen et al., 2003), glycogen and phosphocreatine (PCr) store repletion (Haff et al., 2003; Hirvonen et al., 1987), cardiac parasympathetic reactivation (Stanley et al., 2013), and performance recovery. Monitoring the return of cardiac parasympathetic activity to its pre-exercise state is a method commonly employed in studies examining the post-exercise recovery time-course (Mourot et al., 2004; Niewiadomski et al., 2007; Seiler et al., 2007), as the return of autonomic balance is associated with many physiological mechanisms involved in recovery, thus providing a comprehensive measure of recovery status (Aubert et al., 2003). Furthermore, parasympathetic recovery has been shown to be dependent on exercise intensity, with recovery occurring within 24 h following low intensity ($<VT_1$) exercise, 24-48 h following $\sim VT_2$ intensity exercise, and >48 h following high-intensity ($>VT_2$) exercise (Stanley et al., 2013). However, studies examining cardiac parasympathetic recovery time-course following training sessions of differing intensities rarely observe the parallel time-course of alternative measures of recovery. Additionally, research to date has not examined homeostatic return in a real-world setting, whereby athletes typically perform multiple sessions per day, which is suggested to influence recovery time-course (Fiskerstrand & Seiler, 2004; Stanley et al., 2013).

Another factor influencing acute recovery is that of energetic contribution. Individuals presenting a greater capacity for the use of aerobic energy pathways in comparison to those with a greater capacity for the utilisation of anaerobic energy pathways, have been shown to demonstrate reduced acute post-exercise sympathetic stress (McDonald et al., 2014). If indeed acute post-exercise deviation is related to recovery time-course, energetic profile (capacity for aerobic or anaerobic energy system contribution to exercise) likely also influences recovery time-course, and may therefore provide a means of individualising training programming. Particularly in intermediate duration sports (3-8 min) such as rowing, track cycling, flat-water kayak, and middle distance running which require large contributions of both aerobic and anaerobic energy systems (Craig & Norton, 2001; de Campos Mello et al., 2009; D. W. Hill, 1999; Schumacher & Mueller, 2002; Zouhal et al., 2012). Thus allowing successful athletes to present relatively wide variations in energetic profile (D. W. Hill, 1999).

Therefore, to extend our understanding of the post-exercise recovery time-course in highly-trained endurance athletes, the aims of this study were to: 1) quantify the magnitude of acute deviation from baseline to high-intensity training sessions using a variety of measures; 2) determine practically applicable recovery time-courses for the purpose of optimising the programming of subsequent high-intensity training sessions in rowing; and 3) to investigate the influence of energetic contribution on recovery time-course within highly trained rowers.

3.3 Methods

3.3.1 Research design

This study employed a repeated measures crossover design for the assessment of acute post-exercise deviation and time-course for recovery to baseline following exposure to three different IT sessions. Participants first attended the laboratory for physiological assessments in the week prior to the experimental trials. Thereafter, participants performed one randomly assigned IT session every seven days, as shown in *Figure 3.1*. Several recovery status measures were repeatedly assessed pre-IT and over the ensuing 72 h period post-IT (*Figure 3.1*).

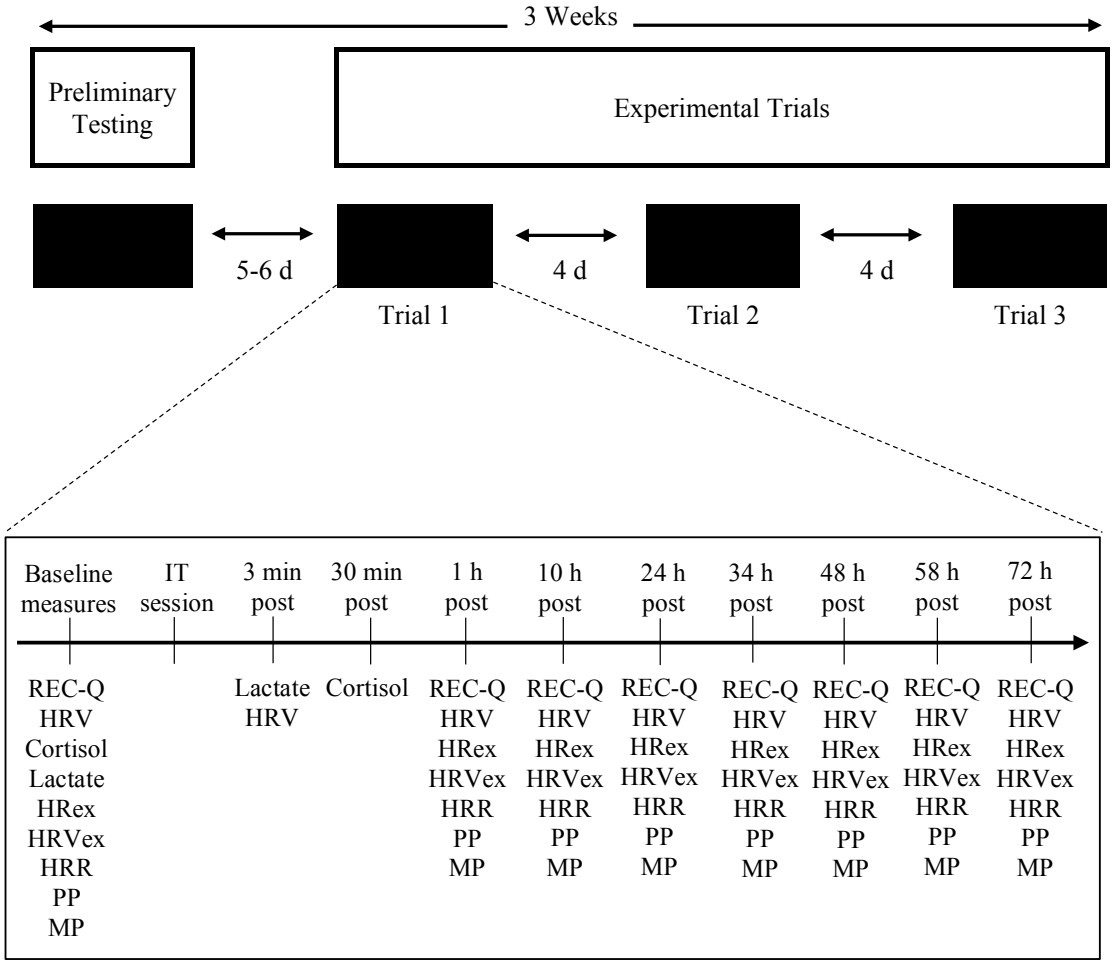

Figure 3.1 Schematic of experimental design and study timeline. Each experimental trial consisted of a randomly assigned interval training (IT) session followed by several recovery measures (in measurement order) taken throughout the 72 h post-IT period. A period of 5-6 d separated preliminary testing from the first experimental trial, with IT sessions separated by 7 days. REC-Q – perceived recovery; HRV – resting heart rate variability; HRex – submaximal exercise heart rate; HRVex – post-exercise HRV; HRR – heart rate recovery; PP – modified Wingate peak power; MP – modified Wingate mean power.

3.3.2 Participants

Thirteen highly-trained rowers participated in the study (*Table 3.1*), all of which belonged to the same training squad, with nine having competed in international age-group events and one in elite international events. All participants had been involved in regular training over the past 6 months with a mean (±SD) weekly training volume of 20 ±4.3 h at the time of the study, and had achieved the minimal performance time of <6:40 min (males) and <7:30 min (females) during a 2000 m rowing ergometer test for study inclusion

(*Table 3.1*). Participants or legal guardians where appropriate, provided informed consent prior to the commencement of the study. The study was approved by the Auckland University of Technology Ethics Committee (AUTEC).

Table 3.1 Participant Characteristics

Variable	**Men (N=10)**	**Women (N=3)**	**Total (N=13)**
Age (yr)	20.9 ±4.0	18.0 ±0.0	20.2 ±3.7
Height (cm)	189.1 ±6.7	181.8 ±9.4	187.4 ±8.0
Body mass (kg)	85.9 ±7.2	74.7 ±10.6	83.4 ±9.4
VO_2peak ($L \cdot min^{-1}$)	5.26 ±0.37	3.85 ±0.39	4.93 ±0.71
2000 m ergometer time (min)	06:21.9 ±0:22.9	07:07.3 ±0:09.7	06:34.3 ± 0:10.8

Data expressed as mean ±SD. VO_2peak – peak oxygen uptake.

3.3.3 Preliminary testing

All testing was performed in a temperature controlled laboratory (21 ±0.7 °C). Following a 24 h rest period, participants performed familiarization measures for HRV and the 5'-5' test. Thereafter, participants performed a step-test consisting of 5 x 3 min submaximal stages with 20 W power increments per stage on a rowing ergometer (Concept II Model E static ergometers, Concept II Inc., Morrisville, VT) to establish the power (W)–VO_2 ($L \cdot min^{-1}$) relationship. Power for the initial stage was dependant on the rower's profile (heavyweight males: 180 W; heavyweight females and lightweight men: 140 W; lightweight females: 120 W). Stroke rate was self-selected and the drag factor was adjusted to match the rower's gender (males: 130 units; females: 110 units) in accordance with Rowing New Zealand standards. Heart rate and expired air was collected throughout the test using a metabolic gas-analysis system (ParvoMedics TrueOne 2400, Salt Lake City, UT) which was calibrated prior to all tests using alpha standard gases (BOC gases, Auckland, NZ) and a 3 L syringe (Hans Ruldolph, Shawnee, USA).

A 45 min rest period followed completion of the submaximal step-test, after which a standardized warm-up consisting of 10 min rowing at a self-selected pace and stroke rate was performed. In the final 5 min of the warm up, participants performed three maximal 10-stroke bursts. After a subsequent 5 min preparatory period, participants performed a 6 min maximal rowing test. During the test, participants were instructed to implement the

same self-regulated stroke rate and pacing strategy as they would during a 2000 m ergometer test. Expired air, power output, and HR were collected throughout the test and VO_2peak established as the highest VO_2 ($L \cdot min^{-1}$) achieved over 30 s, with VO_2peak power established as power output (W) produced over the same 30 s period. Heart rate was recorded throughout the test to establish HRmax (highest HR attained). Recognising the effect of body mass on on-water rowing performance (Nevill, Beech, Holder, & Wyon, 2010), ergometer performance was expressed in both absolute (mean power output (W)) and relative terms, using the equation:

$$\text{Weight adjusted power ouput} = p\left(\frac{m}{100}\right)^{-0.666}$$

Where *p* is mean 6 min power output (W) and *m* is body mass (kg).

Energetic contribution

Oxygen uptake ($L \cdot min^{-1}$) during the final min of each stage of the step-test was plotted against actual power output (W), to establish a regression model at submaximal intensities. Extrapolation of the regression model allowed calculation of the maximal accumulated oxygen demand and uptake from the 6 min maximal test. Energetic contribution was then established as percentage aerobic energy system contribution to the test, which was calculated from the difference between accumulated oxygen demand and uptake, whilst accounting for oxygen stores (Tanner & Gore, 2013). This method has been accepted as the 'gold-standard' protocol for the quantification of an individual's energy system contributions, and established as a reliable and valid measure (Noordhof et al., 2010).

3.3.4 Experimental protocol

The experimental trials were performed over a three-week period, and consisted of three rowing ergometer IT sessions completed in random order, with each IT session separated by 7 d and performed following the same standardized 10 min warm-up previously described. The IT sessions were: 1) 5 x 3.5 min work periods; 4 min rest periods (VO_2); 2) 10 x 30 sec work periods; 5 min rest periods (Glycolytic); and 3) 5 x 10 min work periods; 4 min rest periods (Threshold). These IT sessions were selected to represent high-intensity exercise performed at: 1) maximal oxygen uptake (VO_2 IT); 2) maximal anaerobic glycolytic power (Glycolytic IT); and 3) VT_2 (Threshold IT), as would be prescribed in a real-world rowing setting. Participants were instructed to perform all

intervals at the highest maintainable pace, with active recovery between repetitions consisting of low-intensity rowing (instructed as applying minimal force whilst maintaining movement).

Heart rate was recorded throughout each IT session and analysed for time spent 80-90% and >90% HRmax, while mean power (W) of the work periods completed (i.e. excluding rest periods) was recorded from the ergometer's PM4 monitor (Concept II Inc., Morrisville, VT). Session RPE (sRPE) was recorded on IT session completion using Borg's 15-point scale (M. J. Chen, Fan, & Moe, 2002; Christen, Foster, Porcari, & Mikat, 2016). Blood samples were collected pre and 3 min post each IT session by earlobe capillary sample, and analysed immediately using a portable lactate analyser (Lactate Pro 2 LT-1730 analysers, Arkray, Tokyo, Japan). Prior to each IT session recovery measures were obtained and repeated throughout the 72 h post-IT period. The measurement timing for recovery measures is presented in *Figure 3.1*.

3.3.5 Recovery measures

The 5'-5' test

The 5'-5' test required participants to perform 5 min of submaximal ergometer rowing at target power outputs reflective of 60.8 ±5.9% VO_2peak power (mean ± SD) (heavyweight males: 240 W; heavyweight females and lightweight males: 200 W; lightweight females 160 W). Followed immediately by 5 min of seated rest, for the measurement of HRex, HRR, and HRVex (Buchheit, Mendez-Villanueva, Quod, Poulos, & Bourdon, 2010), at baseline and throughout the 72 h post-IT period (*Figure 3.1*). The ergometer drag factor was adjusted to match the rower's gender (males: 130 units; females: 110 units) in accordance with Rowing New Zealand standards, and held consistent for all ergometer tests throughout the experimental trial period.

Heart rate measures

All HR data measured throughout this study was recorded using Polar RS800CX HR monitors (Polar, Electro Oy, Kemplele, Finland) set to record R-R series at a 5 s sampling rate, and analysed via Polar Protrainer 5 Performance software (version 5.41.2, Kemplele, Findland). Ectopic beats were replaced automatically using adjacent R-R interval values. Resting HRV was measured for 10 min on arrival in a quiet, dimly lit room at baseline

and throughout the 72 h post-IT recovery period (*Figure 3.1*). Participants were seated, maintaining a still posture (Buchheit, Al Haddad, et al., 2009). The square root of the mean sum of the squared differences between R-R intervals (rMMSD) (Task-Force, 1996) was calculated from 2:30-7:30 min of the HRV recording. HRVex was calculated as rMMSD from the ninth minute of the 5'-5' test. Seated position and noise level were held consistent with HRV measures, for accurate assessment of parasympathetic activity (Buchheit, 2014). Resting HRV measures provide a comprehensive assessment of whole body autonomic balance and therefore homeostatic perturbation. While HRVex measures provide insight into the mechanisms driving parasympathetic reactivation, including blood pressure regulation, baroreflex activity, and metaboreflex responses (Stanley et al., 2013). Submaximal exercise HR (HRex) was recorded during the 5'-5' test and was defined as the average HR over the third to fifth minute for the assessment of performance (Buchheit, Chivot, et al., 2010; Mann et al., 2013). Reductions in HRex are associated with improvements to aerobic capacity, whereas increases to HRex reflect fatigue and reduced performance capacity (Hedelin et al., 2000; Mann et al., 2013), thus providing a measure of performance recovery. Heart rate recovery (HRR) represented the absolute difference in HR across the sixth minute of the 5'-5' test, providing a measure of post-exercise sympathetic withdrawal and parasympathetic reactivation throughout the recovery period (Kannankeril et al., 2004).

The modified Wingate test

The modified Wingate test consisted of a 30 sec maximal rowing ergometer effort from a stationary start, performed directly following the 5'-5' test. Peak and mean power (W), recorded using the ergometer's PM4 memory were used as measures of performance recovery, assessed at baseline and throughout the 72 h post-IT period (*Figure 3.1*). The modified Wingate test provides a practical and valid assessment of performance recovery in rowers. Mean power produced in this test has been found to predict up to 75.7% of the variance in 2000 m ergometer test time, with peak power also demonstrating a strong relationship ($r = -0.85$) with 2000 m ergometer performance (Riechman et al., 2002).

Salivary cortisol

Saliva was collected via the passive drool technique pre- and 30 min post-IT session. Saliva collections were obtained 10 min following mouth rinse with water, participants swallowed to clear their mouths before an unstimulated saliva sample was collected, utilising minimal orofacial movement. Participants were instructed to consume no food

or sports drink between pre and post saliva collections. Approximately 2 mL samples were collected into sterile bijou containers (7 ml-capacity with screw top, Labserve™, Auckland, NZ). Samples were stored at 4 °C for 2 h, after which they were pipetted into 2 mL-capacity 3810X Eppendorf tubes (Eppendorf, Hamburg, Germany) and frozen at -20 °C until batch analysis. Once thawed, samples were centrifuged (5424 R, Eppendorf, Hamburg, Germany) at 1000 g for 2 min and 500 µl from each sample was pipetted into a 1.5 mL-capacity Hitachi cup for cortisol analysis using a Roche Diagnostics™ Modular Analytics E170 instrument (Roche Diagnostics, Basel, Switzerland) at the Auckland University of Technology-Roche Diagnostics Laboratory. Salivary cortisol provides a practical and valid measure of HPA axis activity (Crewther et al., 2008; Papacosta & Nassis, 2011), reflecting the acute stress response to exercise (Adlercreutz et al., 1986).

Psychometric measures

Participants completed a psycho-physiological recovery questionnaire (REC-Q) at baseline and throughout the 72 h post-IT period (*Figure 3.1*) to provide insight into the association between perceived and physiological recovery. The questionnaire assessed participant's fatigue, leg soreness, physical recovery and mental recovery using a 10-point Likert scale ranging from 1 (minimum fatigue/soreness/recovery) to 10 (maximum fatigue/soreness/recovery) (Halson et al., 2008).

3.3.6 Dietary and exercise control

Participants maintained all regular squad training sessions throughout the duration of the study, replicating the same sessions each week (*Table 3.2*). All sessions were quantified using the training impulse (TRIMP) method (Foster et al., 2001), calculated from session duration (h) and sRPE, and are displayed as arbitrary units (AU) in *Table 3.2*. Target HR intensities are presented in *Table 3.2* for each session. Dietary consumption for the 24 h period prior and following each IT session were recorded and replicated week-to-week. Participants were requested to refrain from consuming caffeine 12 h preceding and throughout the 72 h post-IT measurement period.

Table 3.2 Training performed following each experimental trial (IT session).

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Morning training	IT session (Intensity characteristics in *Table 3.3*) TRIMP: 14.7 ±4.0	24 h post-IT Row: 16 km (<VT_1) TRIMP: 14.1 ±1.6	48 h post-IT Row: 5000 m race (~VT_2) TRIMP: 11.1 ±1.2	72 h post-IT Row: 10 x 2 min work, 2 min rest with bungees; 2 x 500 m without bungees (~VT_2) TRIMP: 22.0 ±2.0	Row: 18 km (<VT_1) TRIMP: 21.5 ±2.9	Row: 3 x 3000 m at 28, 30, open spm (~VT_2) TRIMP: 16.9 ±1.3	Rest day
Afternoon training	10 h post-IT Erg: 70 min (<VT_1) Resistance training: 45 min TRIMP: 28.6 ±2.8	34 h post-IT Erg: rate changes 5, 4, 3, 2, 1, 2, 3, 4, 5 min at 20, 22, 24, 26, 30, 26, 24, 22, 20 spm (VT_1 - VT_2) TRIMP: 7.9 ±0.9	58 h post-IT Erg: 30 min (<VT_1) Resistance training: 45 min TRIMP: 17.6 ±2.0	Rest session	Resistance training: 45 min TRIMP: 10.6 ±1.2	Cycling: 90 min (<VT_1) TRIMP: 19.4 ±2.1	

Recovery measures were taken prior to each training session throughout the 72 h post-IT period. IT – interval training; TRIMP – training impulse (mean ±SD); VT_1 – first ventilatory threshold; VT_2 – second ventilatory threshold; spm – strokes per minute. Target heart rate intensity presented in brackets.

3.3.7 Statistical analysis

IT sessions were treated as independent groups for statistical analysis, given the seven-day gap between the performance of each IT session. Participant numbers for each IT session were: 10 (VO_2), 12 (Threshold), and 13 (Glycolytic). Descriptive statistics are shown as mean ± standard deviation (SD) and 90% confidence limits (CL) when stated. Acute deviation was assessed as change from baseline to 3 min post-IT (blood lactate), 30 min post-IT (salivary cortisol), or 1 h post-IT session (REC-Q, HRV, HRex, HRVex, HRR, peak power, mean power). All acute deviation and mean percent change from baseline data was log-transformed prior to analysis to reduce bias arising from non-uniformity of error. Qualitative inferences were used to assess the magnitude of effect and practical detection of change from baseline. This was achieved using a modified statistical spreadsheet (Hopkins, 2006) which calculated standardized changes (std change) and 90% CL with the threshold values ≤0.2 (*trivial*), >0.2 (*small*), >0.6 (*moderate*), >1.2 (*large*), and >2.0 (*very large*). Qualitative chances of response eliciting values higher or lower than baseline were assessed as: 25-75% *possibly*, 75-95% *likely*, 95-99% *very likely*, >99% *most likely*. If the chance of a higher or lower difference from baseline was >5%, then the true difference was deemed *unclear*. Paired samples t-tests were conducted to evaluate the standardized difference in mean change between each training session. Eta squared values were calculated, and 90% CL with qualitative chances of differences occurring in mean acute deviation between VO_2, Threshold, and Glycolytic training were achieved using a modified statistical spreadsheet (Hopkins, 2007).

Recovery time-courses were considered as the time difference between baseline and the post-IT return to baseline values. Where the measure did not return to baseline values within the 72 h measurement period, the time-point closest to baseline was taken. Recovery time-course data was log transformed, with qualitative chances of differences occurring between IT sessions in recovery time-course achieved using a modified statistical spreadsheet (Hopkins, 2006). Spearman rho correlations were used to establish the relationship between acute deviation and time-course for recovery from each IT session, since data violated the assumptions of linearity and homoscedasticity. Partial correlations were used to establish the relationship between energetic contribution (percentage aerobic contribution to the 6 min test) and recovery time-course, while controlling for the effect of 6 min test performance. Partial correlations controlled for 6 min test performance in both absolute and relative terms, using either raw or weight

adjusted mean 6 min power output. Both Spearman rho and partial correlation analyses were accomplished using SPSS 22 (SPSS Inc, Chicago, IL, USA). Spearman rho and partial correlations were evaluated using the thresholds: ≤0.1 (*trivial*), >0.1 (*small*), >0.3 (*moderate*), >0.5 (*large*), >0.7 (*very large*), and >0.9 (*almost perfect*) (Hopkins, Marshall, Batterham, & Hanin, 2009). Where 90% CL overlapped *small* negative and positive values the magnitude of correlation was deemed *unclear* (Hopkins et al., 2009).

3.4 Results

Mean (±SD) power during the 6 min maximal test was 365.1 ±52.0 W. Mean aerobic contribution to the 6 min maximal test was 88.6 ±6.4%. Mean weekly TRIMP was 184.5 ±3.6 AU. Intensity characteristics of the three IT sessions are displayed in *Table 3.3*.

Table 3.3 Physiological responses to interval training sessions (mean ± SD).

Measure	**VO_2 IT**	**Threshold IT**	**Glycolytic IT**
sRPE	19 ±0.7	18.3 ±0.9	16.2 ±1.7
Mean power (W)	336.1 ±55.8	290 ±29.9	544.5 ±114.0
Intensity (% VO_2peak power)	97.0 ±5.4	79.6 ±6.8	156.4 ±15.6
TRIMP	10.6 ±0.4	16.7 ±7.9	12.1 ±1.3
Time >90% HRmax (m:s)	14:46 ±2:03	30:30 ±11:45	2:42 ±3:37
Time 80-90% HRmax (m:s)	7:34 ±3:39	18:06 ±9:40	7:12 ±2:01
Peak HR (% HRmax)	98.1 ±2.4	99.1 ±1.5	94.6 ±2.8
Blood lactate ($mmol \cdot L^{-1}$)	11.6 ±2.5	8.2 ±2.9	11.8 ±3.8

IT – interval training; sRPE – session rating of perceived exertion; TRIMP – training impulse; HRmax – maximal heart rate; HR – heart rate.

3.4.1 Acute post-exercise deviation from baseline

Magnitudes of acute post-exercise deviation from baseline are presented in *Table 3.4*. The std mean change (±90% CL) in cortisol values post-VO_2 IT was 1.11 ±0.52, presenting the largest IT session response. Post-training blood lactate responses were similar between VO_2 (6.13 ±0.61) and Glycolytic (6.55 ±0.7) IT. The most substantial suppression of HRV and HRVex was observed following Threshold IT (-1.09 ±0.7). Whereas, REC-Q scores demonstrated the greatest effect following VO_2 IT (2.12 ±0.63).

Difference in the magnitude of acute deviation between IT sessions is presented in *Table 3.5* as standardised Cohen's units. *Trivial* differences were found between Threshold and VO_2 IT in salivary cortisol and HRR responses; between Glycolytic and VO_2 IT in blood lactate, HRV, HRex, HRR, and mean power responses; and between Threshold and Glycolytic IT in salivary cortisol, HRVex, HRR, peak power, mean power, and REC-Q responses. Differences between the IT sessions for the responses of all other measures were *unclear* (*Table 3.5*).

Table 3.4 Within standardised mean pre-post change, and qualitative chances of change following three different interval training sessions (VO_2, Threshold, and Glycolytic).

Variable measured	**VO_2 IT**			**Threshold IT**			**Glycolytic IT**		
	Std change in mean (±90% CL)	% Chances (+/trivial/-)	Qualitative inference	Std change in mean (±90% CL)	% Chances (+/trivial/-)	Qualitative inference	Std change in mean (±90% CL)	% Chances (+/trivial/-)	Qualitative inference
Cortisol	1.11 (0.52)	99/1/0	Very likely moderate	0.45 (0.61)	77/19/4	Unclear	0.31 (0.53)	64/30/6	Unclear
Blood lactate	6.13 (0.61)	100/0/0	Most likely very large	4.78 (0.57)	100/0/0	Most likely very large	6.55 (0.70)	100/0/0	Most likely very large
HRV	-0.97 (0.35)	0/0/100	Most likely moderate	-1.09 (0.70)	1/2/98	Very likely moderate	-0.46 (0.44)	1/15/84	Unclear
HRVex	-0.43 (0.51)	3/19/79	Unclear	-1.09 (0.70)	0/0/100	Most likely moderate	-0.42 (0.49)	2/19/78	Unclear
HRex	0.07 (0.20)	13/85/2	Unclear	0.45 (0.20)	83/16/2	Unclear	0.00 (0.21)	6/89/5	Unclear
HRR	-0.05 (0.60)	23/44/32	Unclear	-0.63 (0.49)	1/6/93	Unclear	-0.47 (0.39)	1/11/88	Unclear
Peak power	-0.08 (0.09)	0/98/2	Very likely trivial	-0.04 (0.14)	1/96/3	Very likely trivial	0.03 (0.18)	6/91/2	Unclear
Mean power	-0.05 (0.11)	0/98/2	Very likely trivial	0.02 (0.18)	5/92/3	Unclear	0.05 (0.16)	6/93/1	Unclear
REC-Q	2.12 (0.63)	100/0/0	Most likely very large	1.65 (0.53)	100/0/0	Most likely large	1.41 (0.57)	100/0/0	Most likely large

Change from baseline measured 3 min post-IT (blood lactate), 30 min post-IT (salivary cortisol), and 1 h post-IT session (REC-Q, HRV, HRex, HRVex, HRR, peak power, mean power). IT – Interval training; Std – standardised; CL – confidence limits; HRV – resting heart rate variability; HRVex – post-submaximal exercise HRV; HRex – post-submaximal exercise heart rate; HRR – heart rate recovery; REC-Q – perceived recovery.

Table 3.5 Between standardised mean pre-post differences, and qualitative chances of difference between three different interval training sessions (VO_2, Threshold, and Glycolytic).

Variable measured	**Threshold vs. VO_2 IT**			**Glycolytic vs. VO_2 IT**			**Threshold vs. Glycolytic IT**		
	Std diff in mean (±90% CL)	% Chances (+/trivial/-)	Qualitative inference	Std diff in mean (±90% CL)	% Chances (+/trivial/-)	Qualitative inference	Std diff in mean (±90% CL)	% Chances (+/trivial/-)	Qualitative inference
Cortisol	-0.08 (0.29)	2/98/1	Very likely trivial	-0.38 (0.65)	37/61/2	Unclear	0.13 (0.22)	1/99/0	Very likely trivial
Blood lactate	-0.57 (0.39)	63/37/0	Unclear	0.00 (0.02)	1/100/0	Most likely trivial	-0.37 (0.30)	22/78/0	Unclear
HRV	-0.35 (0.38)	23/77/0	Unclear	0.09 (0.19)	0/100/0	Most likely trivial	0.38 (0.30)	24/76/0	Unclear
HRVex	0.18 (0.42)	10/89/1	Unclear	0.45 (0.36)	41/59/0	Unclear	0.02 (0.33)	1/98/1	Very likely trivial
HRex	-0.42 (0.53)	38/61/1	Unclear	-0.07 (0.20)	0/100/0	Most likely trivial	0.47 (0.45)	45/54/0	Unclear
HRR	-0.02 (0.15)	0/100/0	Most likely trivial	-0.05 (0.16)	0/100/0	Most likely trivial	0.14 (0.31)	4/96/1	Very likely trivial
Peak power	-0.32 (0.42)	21/78/1	Unclear	-0.06 (2.50)	36/31/33	Unclear	0.07 (0.34)	3/96/2	Very likely trivial
Mean power	-0.56 (0.51)	59/40/1	Unclear	-0.02 (0.11)	0/100/10	Most likely trivial	-0.14 (0.32)	4/96/1	Very likely trivial
REC-Q	-0.15 (0.40)	8/92/1	Unclear	-0.20 (0.38)	9/90/0	Unclear	-0.04 (0.12)	0/100/0	Most likely trivial

Change from baseline measured 3 min post-IT (blood lactate), 30 min post-IT (salivary cortisol), and 1 h post-IT session (REC-Q, HRV, HRex, HRVex, HRR, peak power, mean power). IT – Interval training; Std diff – standardised difference; CL – confidence limits; HRV – resting heart rate variability; HRVex – post-submaximal exercise HRV; HRex – post-submaximal exercise heart rate; HRR – heart rate recovery; REC-Q – perceived recovery.

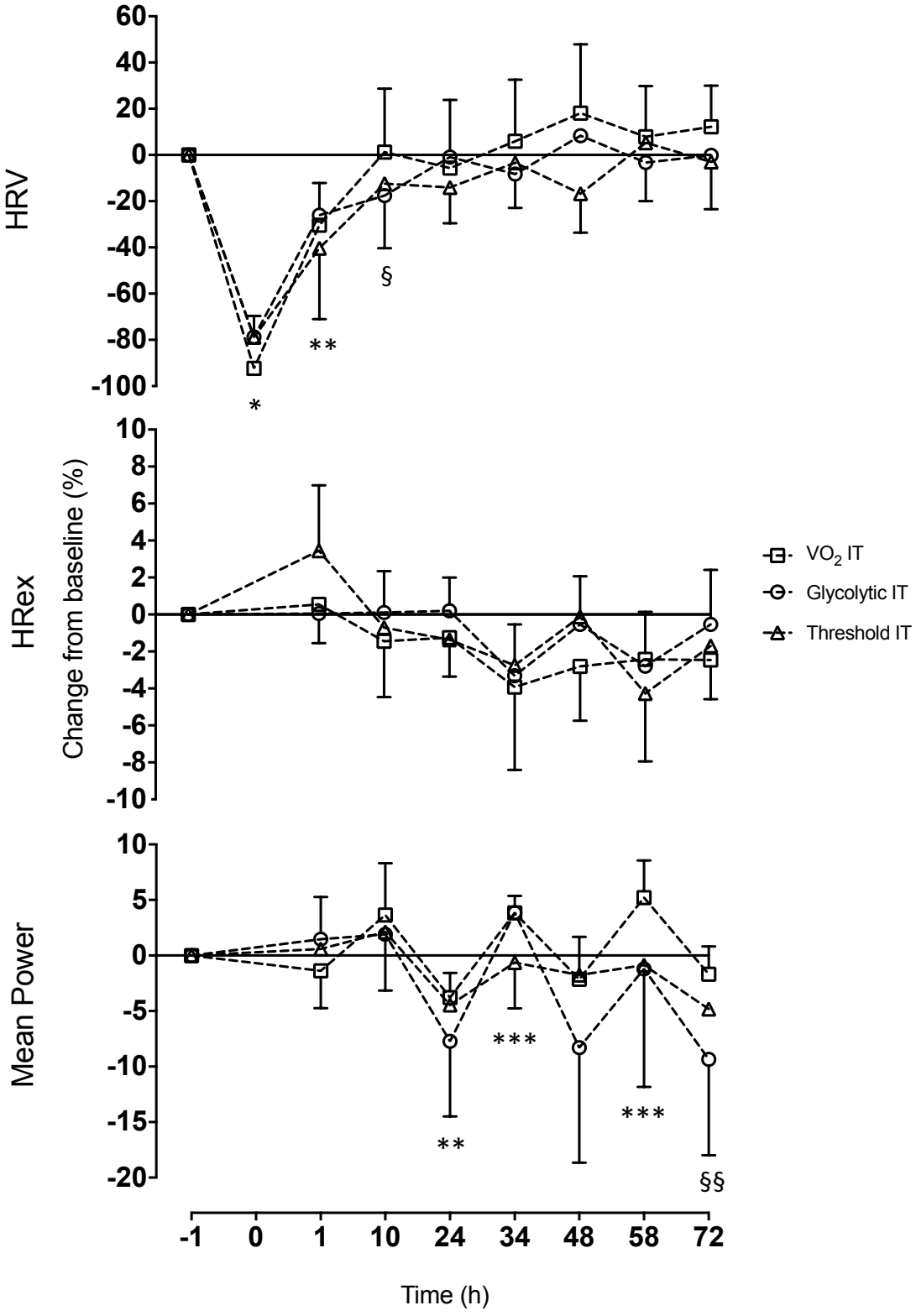

Figure 3.2 Mean percent change (±90% CL) from baseline over time following the three interval training (IT) sessions (VO_2, Glycolytic, and Threshold). HRV – resting heart rate variability; HRex – submaximal exercise heart rate; Mean Power – modified Wingate mean power. * *Most likely* difference from baseline post-VO_2, Glycolytic and Threshold

intervals. ** *Very likely* difference from baseline post-VO_2, Glycolytic and Threshold intervals. § *Likely* difference from baseline post-Glycolytic intervals. *** *Very likely* difference from baseline post-VO_2 intervals. §§ *Very likely* difference from baseline post-Glycolytic intervals. Error bars omitted from one interval training session for clarity.

3.4.2 Recovery time-course

Mean (±CL) time-course of recovery from each IT session is presented in *Table 3.6*. Time-course of return to baseline illustrated substantial inter-individual variation, with variances of ≥24 h demonstrated in all recovery variables. Between IT session analysis revealed HRVex to have a *likely large* difference in recovery time-course between Threshold and Glycolytic IT (-1.61 ±1.54; std diff in mean ±CL), however differences between IT sessions for all other variables were *unclear*. Nevertheless, HRV demonstrated the longest mean recovery time-course following Threshold IT (29.2 ±12.1 h) compared to VO_2 (15.7 ±11.2 h) and Glycolytic (17.8 ±9.6 h) IT. Threshold IT also demonstrated the longest mean recovery time-course in HRVex, with similarly shorter mean recovery time-courses observed following VO_2 and Glycolytic IT. Conversely, mean recovery time-course was longest post-VO_2 in HRex, HRR, mean and peak power, with Glycolytic IT eliciting the quickest mean recovery time-course for these measures (*Table 3.6*).

Across IT sessions HRVex presented the longest recovery time-course of the recovery variables, followed by HRV. While peak power and mean power demonstrated the most rapid recovery time-courses across IT sessions (*Table 3.6*). In this study total REC-Q score did not return to baseline throughout the measurement period following all IT sessions. Mean percent change (± CL) from baseline over time for HRV, HRex, and mean power following each IT session is presented in *Figure 3.1*.

Table 3.6 Mean (±90% CL) recovery time-course following three different interval training sessions (VO_2, Threshold and Glycolytic).

	VO_2 IT		**Threshold IT**		**Glycolytic IT**	
	Time-course (h)	Range (h)	Time-course (h)	Range (h)	Time-course (h)	Range (h)
HRV	15.7 ±11.2	1-39	29.2 ±12.1	1-61	17.8 ±9.6	1-58
HRVex	20.6 ±15.2	1-66	37.8 ±14.2	7-72	20.2 ±11.0	1-55
HRex	16.0 ±13.2	1-58	12.7 ±6.3	1-34	7.4 ±4.6	1-24
HRR	20.1 ±13.7	1-58	14.2 ±9.2	1-44	12.0 ±7.6	1-48
Peak power	14.1 ±8.7	1-33	11.3 ±8.7	1-35	6.1 ±6.1	1-34
Mean power	13.1 ±9.5	1-31	8.1 ±7.5	1-38	7.8 ±6.6	1-34

IT – interval training; CL –confidence limits; HRV – resting heart rate variability; HRVex – post-submaximal exercise heart rate variability; HRex – submaximal exercise heart rate; HRR– heart rate recovery; REC-Q – perceived recovery.

Correlations between acute deviation and time-course to return to baseline are presented in *Table 3.7*. *Very large* negative correlations were found between the acute change in mean power and HRVex with recovery time-course following VO_2 IT, with greater suppression of these variables 1 h post-exercise associated with longer recovery time-courses. Additionally, HRV, HRR, peak power, and mean power demonstrated *large* and *very large* negative correlations between the variables following both Threshold and Glycolytic IT, while a *very large* negative correlation was found in HRVex following Glycolytic IT only. A *very large* positive relationship was also found in HRex following Threshold and Glycolytic IT, with greater increases to acute post-exercise HRex associated with longer time-courses for baseline return.

3.4.3 Influence of energetic contribution on recovery time-course

Partial correlation coefficients for time to return to baseline vs. aerobic contribution to the 6 min test, while controlling for either raw 6 min test mean power or weight adjusted 6 min test mean power are presented in *Figure 3.3*. When controlling for raw 6 min test mean power *large* and *moderate* positive relationships were found between recovery time-course and aerobic contribution in HRex following Threshold (r = 0.53 ±0.51) and HRVex following Glycolytic IT (r = 0.36 ±0.47) respectively. In addition, a *large* negative relationship was found between recovery time-course and aerobic contribution in HRR following Threshold IT (r = -0.52 ±0.51). All other relationships were *unclear*.

Similar findings were demonstrated when controlling for weight adjusted 6 min test mean power, with *large* and *moderate* positive relationships also found between recovery time-course and aerobic contribution in HRex following Threshold (r = 0.55 ±0.42) and HRVex following Glycolytic IT (r = 0.36 ±0.47) respectively. While a *moderate* negative relationships was found between recovery time-course and aerobic contribution in HRR following Threshold IT (r = -0.48 ±0.46), as well as an additional *large* negative relationship in peak power following Threshold IT (r = -0.55 ±0.46). Thus the relationship between energetic contribution and recovery time-course across a range of recovery variables appears limited and conflicting, with little difference identified in these findings when controlling for raw or weight adjusted performance parameters.

Table 3.7 Spearman's correlation coefficients between the acute deviation and time-course for recovery to baseline following three IT sessions.

	VO_2 IT	**Threshold IT**	**Glycolytic IT**
HRV	-0.20	-0.79*	-0.62*
HRVex	-0.87**	-0.68	-0.71*
HRex	-0.63	0.88**	0.85**
HRR	-0.50	-0.83**	-0.73*
Peak power	-0.46	-0.71*	-0.84**
Mean power	-0.82*	-0.83*	-0.89**

IT – interval training; HRV – resting heart rate variability; HRVex – post-exercise heart rate variability; HRex – submaximal heart rate; HRR – heart rate recovery. *$p < 0.05$ (2-tailed); **$p < 0.01$ (2-tailed).

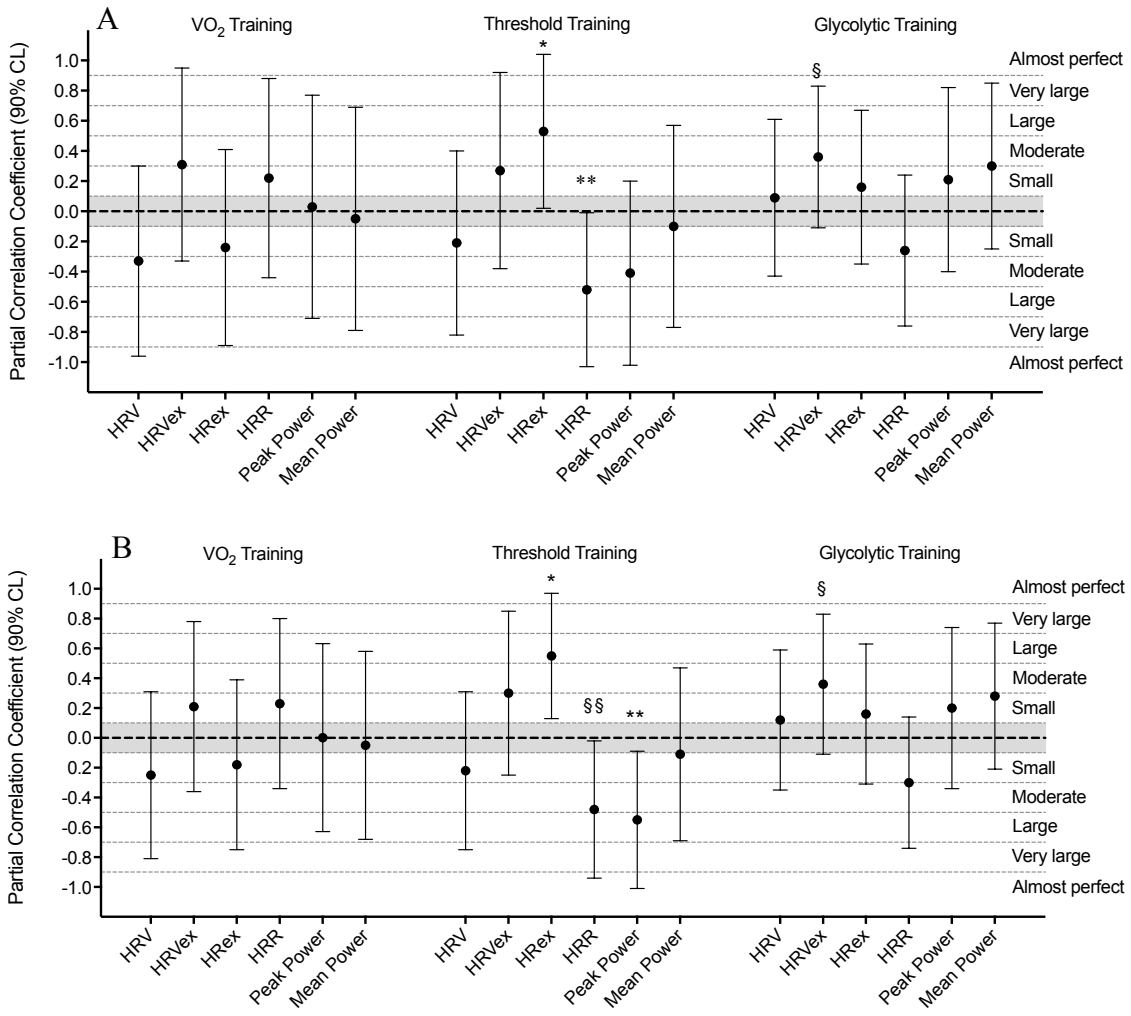

Figure 3.3 Mean partial correlation coefficient with 90% confidence limits (CL) for recovery time-course and percentage aerobic contribution to the 6 min test (energetic contribution), while controlling for 6 min test mean power output (A); or weight adjusted 6 min test mean power output (B). HRV – resting heart rate variability; HRVex – post-submaximal exercise heart rate variability; HRex – submaximal exercise heart rate; HRR– heart rate recovery. **Large* positive relationship; ***large* negative relationship; §*moderate* positive relationship; §§*moderate* negative relationship. The shaded grey area represents trivial correlations.

3.5 Discussion

The aim of the present study was to determine the effect of differing IT sessions on the acute deviation and time-course of recovery to baseline in highly trained rowers, across a typical training week. The main findings of this study suggest that similar magnitudes of acute deviation from baseline exist following Threshold, Glycolytic and VO_2 IT in highly trained male and female rowers. Nevertheless, relationships exist between the

acute deviation and recovery time-course to baseline. Furthermore, duration of time spent close to HRmax demonstrated an association with recovery time-course, while the influence of energetic contribution on recovery time-course was limited and inconclusive.

3.5.1 Acute post-exercise deviation from baseline

Intensity characteristics of the three IT sessions (*Table 3*.3) reveal actual intensities performed to reflect targeted intensities, with VO_2 IT performed close to VO_2peak (97.0 ±5.4% VO_2peak power; mean ±SD); Threshold IT reflecting intensities within the range typical of VT_2 (79.6 ±6.8% VO_2peak power) (Meyer, Gabriel, & Kindermann, 1999); and Glycolytic IT performed well above VO_2peak (156.4 ±15.6% VO_2peak power). Peak HR achieved during Threshold and VO_2 IT sessions were similarly high (99.1 ±1.5 and 98 ±2.4 bpm, respectively), with the lower peak HR achieved in Glycolytic IT (94.6 ±2.8 bpm) explained by HR lag rather than exercise intensity (Cerretelli & Di Prampero, 1971). Lag in HR also explains the greater time spent 80-90% than >90% HRmax during Glycolytic IT, even so Glycolytic IT demonstrated the shortest total time spent >80% HRmax. Threshold IT illustrated substantially longer durations spent 80-90% and >90% HRmax than either VO_2 or Glycolytic IT (*Table 3.3*), likely reflecting greater HR drift experienced in response to the longer interval durations performed in Threshold IT (Hartwell et al., 2015). However despite differences in intensity (%VO_2peak power) and time spent close to HRmax, blood lactate response was *most likely very large* across all IT sessions, with only a *most likely trivial* difference between Glycolytic (11.8 ±3.8 $mmol \cdot L^{-1}$) and VO_2 IT (11.6 ±2.5 $mmol \cdot L^{-1}$) observed, with all other differences in blood lactate response *unclear*. Similarly, although cortisol demonstrated a *very likely moderate* increase post-VO_2 IT, *very likely trivial* and *unclear* differences were observed between IT sessions (*Table 3.5*). These findings highlight the variable nature of cortisol as a measure of stress response (Suay et al., 1999), and disagree with previous research demonstrating a positive relationship between cortisol response and exercise duration performed at intensities above VT_2 (Kindermann et al., 1982; Snegovskaya & Viru, 1993).

Additionally, only *trivial* and *unclear* differences were found in the acute deviation from baseline across a range of recovery measures used to reflect recovery status (*Table 3.5*). This included differences in parasympathetic suppression between IT sessions, with only

a *most likely trivial* difference in acute HRV deviation observed between Threshold and Glycolytic IT. Although, parasympathetic suppression was expected to be substantially larger in Threshold IT given the extended time spent close to HRmax during this IT session. Previous research demonstrates time spent at intensities above VT_2 to be related to catecholamine accumulation (Kindermann et al., 1982; Manetta, Brun, Prefaut, & Mercier, 2005; Urhausen, Weiler, Coen, & Kindermann, 1994), reflecting a larger sympathetic response and corresponding parasympathetic suppression (Christensen & Galbo, 1983; Perini et al., 1989). However, any durational influence on sympathetic response was not observed on measures of autonomic balance taken 1 h post-IT in the current study.

3.5.2 Recovery time-course

The current study observed Threshold IT to induce the longest time-course for autonomic balance across recovery measures (*Table 3.6*), with HRV return to baseline presenting 29.2 ±12.1 h for Threshold IT, followed by Glycolytic (17.8 ±9.6 h) and VO_2 IT (15.7 ±11.2 h). These findings oppose that of previous research, whereby higher intensities have been shown to elicit longer HRV recovery time-courses, irrespective of exercise duration (Mourot et al., 2004; Niewiadomski et al., 2007; Seiler et al., 2007). However, the association between time spent 80-90% and >90% HRmax with recovery time-course in the current study suggests a durational effect for intensities relative to $\geq VT_2$. Whereby longer time spent close to HRmax relates to greater indices of sympathetic activity, thus influencing the regulation of cardiovascular parameters and prolonging cardiac autonomic recovery. Comparison between studies investigating post-exercise HRV recovery time-course proves difficult given the influence of exercise intensity (Buchheit et al., 2007), measurement position (Buchheit, Al Haddad, et al., 2009), and cardiovascular fitness (Hautala et al., 2001) on the assessment of HRV. Even so, the current study observed an accelerated HRV recovery time-course by ~30 h following VO_2 and Glycolytic IT compared to that predicted from the interpolation of data from several studies by Stanley et al. (2013) for exercise performed above VT_2.

Differences between the present findings and that observed by Stanley et al. (2013) may be explained by the inclusion of a non-passive recovery period in the current study, i.e. participants continued with their normal habitual training (*Table 3.2*), unlike those analysed by Stanley et al. (2013). Some evidence exists supporting the benefit of

performing low intensity exercise on the recovery process, with enhanced thermoregulation (Carter et al., 2002), metabolite removal (Gill, Beaven, & Cook, 2006), and no influence of parasympathetic activity suppression evident (Plews et al., 2014; Seiler et al., 2007). As low intensity training was performed 10 and 48 h post-IT (*Table 3.2*), it is possible that these subsequent low-intensity sessions hastened recovery time-course. Although, resistance training was also performed 10 h post-IT, which has been shown to supress parasympathetic activity (Kingsley et al., 2014; Teixeira, Ritti-Dias, Tinucci, Júnior, & de Moraes Forjaz, 2011), likely minimising beneficial effects of the low-intensity sessions and potentially prolonging recovery time-course. Further research examining a non-passive recovery period is required to confirm the influence of subsequent training sessions on recovery time-course. Elite athletes typically perform high training frequencies (Fiskerstrand & Seiler, 2004) and are therefore likely to exercise prior to the achievement of complete homeostatic recovery. Therefore, if research examining the post-exercise recovery time-course is to have a practical impact on training programming, it should seek to determine the influence of varying intensities and types of subsequent exercise on time taken to return to baseline. If indeed an influence exists, recovery time-courses observed in the current study are likely specific to the training performed throughout the recovery period (*Table 3.2*).

The current study demonstrates recovery time-course to vary depending on the recovery variable assessed. Performance parameters illustrated longer time to recover following VO_2 IT than either Threshold or Glycolytic IT in HRex, HRR, mean and peak power. Although differences in acute salivary cortisol, blood lactate, and REC-Q deviation from baseline did not differ between IT sessions, VO_2 IT demonstrated the greatest mean sRPE score (19 ±0.7; mean ± SD), indicating greater indices of fatigue and peripheral stress. Similarly, VO_2 IT demonstrated the longest time-course for HRR recovery, demonstrating that although acute sympathetic response did not differ between IT sessions, sympathetic withdrawal was delayed to a greater extend following VO_2 IT. As such, VO_2 IT demonstrates delayed recovery of both performance and autonomic (albeit to a lesser extent than Threshold IT) variables. Whereas, Threshold IT predominantly delayed the recovery of autonomic variables, likely due to the substantially smaller mean power produced (*Table 3.3*) reducing the incidence of muscle damage and inflammation, and thus recovery time-course of performance variables (Armstrong, Warren, & Warren, 1991). Interestingly, Glycolytic IT induced the shortest recovery time-courses for both autonomic and performance variables. Although mean power was highest in Glycolytic

IT (*Table 3*.3), the shorter duration of intervals (30 s) and the longer rest periods (5 min) allowing for partial restoration of plasma pH, phosphocreatine and ATP stores (Buchheit et al., 2013; McCartney et al., 1986) presumably resulted in the accumulation of less overall peripheral and sympathetic stress, in comparison to Threshold and VO_2 IT. This disagreement between recovery variables highlights the need for a comprehensive assessment of recovery status, considering the recovery of performance and autonomic variables did not reflect whole system recovery in the current study.

Participant's energetic contributions to a 6 min maximal rowing test were determined to gain insight into whether variance in the utilisation of energy systems is related to recovery time-course, when controlling for participant's performance ability. Aerobic contributions (88.6 ±6.4%; mean ±SD) were consistent with those previously presented in the literature (de Campos Mello et al., 2009; Pripstein et al., 1999; Russell et al., 1998). Findings were inconsistent with a tendency for participants with greater aerobic contributions to have shorter recovery time-courses for HRR following Threshold IT (r = -0.52 ±0.51; mean ±CL), but longer recovery time-courses for HRex following Threshold IT (r = 0.53 ±0.51) and HRVex following Glycolytic IT (r = 0.36 ±0.47). Furthermore, although a range of recovery variables were assessed, all other relationships were *unclear,* suggesting no definitive influence of energetic contribution on recovery time-course exists. The same correlational analysis was performed while controlling for weight adjusted 6 min test performance to investigate whether a more appropriate measure of on-water rowing performance capacity revealed a more conclusive relationship between aerobic contribution and recovery time-course. Interestingly, very little difference between the two conditions was evident (***Figure 3.3***), with participants possessing greater aerobic contributions additionally demonstrating shorter recovery time-courses for modified Wingate peak power following Threshold IT (r = -0.55 ±0.46) when controlling for weight adjusted performance.

Although not assessed in the current study, previous research demonstrates athletes exhibiting greater aerobic profiles to have faster acute HRR responses (Del Rosso et al., 2016; McDonald et al., 2014; Otsuki et al., 2007), indicating a smaller activation of sympathetic activity and earlier parasympathetic reactivation to the same exercise, than participants illustrating greater anaerobic energy contributions. Thus, it is possible participants with greater aerobic contributions in the current study induced less

sympathetic stimulation from the initial IT session, but this did not translate to a more rapid recovery of autonomic balance in comparison to participants with greater anaerobic contributions. However, further research examining both the acute deviation from baseline and recovery time-course of autonomic balance relative to energetic contribution is required to confirm this hypothesis. Furthermore, partial correlations accounted for the influence of performance ability in the current study, as cardiovascular fitness has been shown to influence recovery time-course (Hautala et al., 2001; Seiler et al., 2007). However, future research considering polarised energetic groups (i.e. groups demonstrating highly aerobic and anaerobic contributions) matched for performance ability may provide a clear difference in the acute deviation and recovery time-courses to baseline.

Lastly, the inclusion of both males and females in the current study should be considered in the interpretation of results presented. Gender differences in circulating reproductive hormone concentrations and thermoregulation may influence recovery (Charkoudian & Joyner, 2004; Kenny & Jay, 2007); however, research investigation the influence of these factors on time to recover proves limited. Menstrual cycle phase has previously been shown to influence blood lactate response to high-intensity exercise, with females in the luteal phase of the menstrual cycle demonstrating reduced lactate production than during other phases (Jurkowski, Jones, Toews, & Sutton, 1981). Menstrual cycle phase has also been found to influence autonomic balance at rest, with greater indices of parasympathetic activity evident during the follicular phase in females (Saeki et al., 1997; Sato et al., 1995). Additionally, previous studies reveal females to have greater parasympathetic withdrawal (Mendonca et al., 2010), and delayed parasympathetic recovery (Kiviniemi et al., 2010) following high-intensity exercise than males. Thus, the inclusion of females may explain some of the differences observed between the current study and previous findings (Kaikkonen et al., 2008; Mourot et al., 2004; Niewiadomski et al., 2007; Seiler et al., 2007).

3.6 Conclusion

In summary, the acute deviation from baseline in measures of autonomic balance, performance, and perceived recovery is similar following Threshold, Glycolytic, and VO_2 IT in highly-trained male and female rowers. Conversely, the recovery of parasympathetic activity during a non-passive recovery period is longest after Threshold,

followed by Glycolytic and VO_2 IT respectively. However, some discordance in recovery duration exists between recovery measures, with the longest recovery of performance variables occurring after VO_2 IT. The wide range of inter-individual variation in recovery time-course observed highlights the need for individualised training programming. Furthermore, the acute deviation from baseline is largely related to recovery time-course, with greater indices of acute deviation associated with longer recovery. Finally, the influence of energetic contribution on recovery time-course revealed limited and inconclusive findings, with larger aerobic contributions associated with longer recovery of HRex following Threshold and HRVex following Glycolytic IT, as well as shorter HRR following Threshold IT. Thus, a more in-depth investigation regarding the influence of energetic contribution on recovery time-course is required to determine the practicality of individualising training programming based on energetic profile.

Chapter Four: Study Two

The influence of energetic contribution to rowing on the acute post-exercise deviation and time to recover to baseline following a single exercise bout: a case study in highly trained rowers

4.1 Abstract

Purpose: To investigate the influence of energetic contribution on inter-individual variation of the acute post-exercise deviation from baseline and time-course of homeostatic recovery following three exercise intensities. ***Methods:*** Two female and four male highly trained rowers (VO_2peak 4.95 ±0.77 $L \cdot min^{-1}$) were selected for pairwise comparison. Participants were matched for performance ability (mean 6 min rowing test power) and then paired with >6.8% difference in percentage aerobic energy contribution to a 6 min rowing performance test, measured in preliminary testing. Participants completed three interval training (IT) sessions on the rowing ergometer at the highest maintainable pace: 5 x 3.5 min, 4 min rest periods (VO_2); 10 x 30 sec, 5 min rest periods (Glycolytic); and 5 x 10 min, 4 min rest periods (Threshold). Blood lactate and salivary cortisol were measured pre, 3 and 30 min post-exercise respectively. Resting heart rate (HR) variability (HRV), post-submaximal exercise HRV (HRVex), submaximal HR (HRex), HR recovery (HRR), modified Wingate peak power and mean power were measured pre and 1, 10, 24, 34, 48, 58, and 72 h post-exercise. ***Results:*** Participants exhibiting greater anaerobic energetic contributions (AnT) demonstrated 64.1 ±103.4 % (mean ±SD) greater blood lactate responses across IT sessions than participants with greater aerobic contributions (AeR). Trends for AeR illustrate larger acute HRV (17.7 ±216.2 %), HRVex (40.1 ±68.7 %), HRR (76.4 ±168.5 %), cortisol (229.2 ±479 %), and HRex (57.0 ±113.9%) responses across IT sessions. Larger acute mean power reduction (107.6 ±100.8 %) in AnT across IT sessions. Longer HRVex (18.0 ±35.9 h) and HRex (10.5 ±18.0) recovery-time courses in AeR, with no consistent difference in recovery-time course for HRV, HRR, mean or peak power between AeR and AnT. ***Conclusion:*** Highly trained rowers illustrate widely varying energetic contributions to rowing performance. AnT demonstrate greater anaerobic contribution to exercise, whereas acute cardiac parasympathetic suppression is greater in AeR. However, no consistent difference in recovery time-course was observed between AeR and AnT, suggesting AeR possess a higher rate of parasympathetic recovery than AnT.

4.2 Introduction

In research examining the acute deviation and time-course of homeostatic recovery to baseline it is widely accepted to report the group mean and standard deviation without presenting the range. This practice largely hides the likelihood that a response highly varied from the mean will be observed in certain participants, irrespective of exercise stimulus standardisation (Vollaard et al., 2009). This observation represents inter-individual variation and is evident in a number of studies examining the adaptive response following training (Bouchard & Rankinen, 2001; Hautala et al., 2006; Hautala et al., 2003; Kohrt et al., 1991; McPhee, Williams, Degens, & Jones, 2010; Scharhag‐Rosenberger, Walitzek, Kindermann, & Meyer, 2012; Sisson et al., 2009; Vollaard et al., 2009), including the acute post-exercise response (Mann et al., 2014). Inter-individual variation can also be expected to occur in the post-exercise recovery time-course, given the association between acute deviation and recovery time-course to baseline (Kaikkonen et al., 2010; Kaikkonen et al., 2007; Kaikkonen et al., 2008). However, due to the limited nature of studies examining the time taken for recovery to baseline, only one has been found to report inter-individual variation (Hautala et al., 2001).

Energetic contribution refers to the contribution of anaerobic and aerobic energy sources to a standardised performance variable. Where differences between participants are likely reflective of variances in the adaptive response to training, however a genetic influence may also exist (Bray et al., 2009). Endurance training induces anatomical and physiological adaptations enhancing aerobic capacity (Ingjer, 1979). Conversely, power and sprint training induces adaptations enhancing anaerobic capacity (Allemeier et al., 1994; Holloszy & Coyle, 1984). However, intermediate duration sports of 3-8 min, such as rowing, require a large contribution of both the aerobic and anaerobic glycolytic energy systems, with previous research demonstrating mean contributions of 84% aerobic and 16% anaerobic energy to a 2000 m rowing test (de Campos Mello et al., 2009; D. W. Hill, 1999; Russell et al., 1998). Thus allowing rowers to present varied energetic contributions to rowing performance, whilst achieving similar performance outcomes. It has been suggested that energetic profile influences acute post-exercise deviation from baseline, with individuals presenting larger anaerobic profiles experiencing greater neuromuscular fatigue, lactate accumulation, and parasympathetic suppression (Del Rosso et al., 2016; Garrandes et al., 2007; Paavolainen et al., 1994). Unfortunatley, limited research has considered the influence of energetic contribution on recovery time-course. Nevertheless, given the positive association between the magnitude of acute deviation from baseline

and recovery time-course observed in Chapter Three (*Table 3.7*), we hypothesise participants demonstrating greater anaerobic energetic contributions to present longer time-courses for homeostatic return following high-intensity IT sessions in comparison to participants demonstrating greater aerobic energetic contributions.

Knowledge of the recovery time-course following different intensities of training is important for the maximisation of training adaptaton and prevention of fatigue accumulation, which overtime can manifest as non-functional overreaching and overtraining (Meeusen et al., 2013). Individualising the programming of subsequent high-intensity training sessions based on recovery status has been shown to benefit training adaptation (Kiviniemi et al., 2010; Kiviniemi et al., 2007), however the practicality of individualising training programming for a squad or team of athletes remains complex. Understanding an athlete's post-exercise recovery requirements relative to their energetic profile would likely ease this impracticality. Thus, the aim of this study was to determine whether the inter-individual variation observed in the acute deviation and recovery time-course to baseline following various high-intenisty exercise stimuli was related to energetic contribution in highly-trained rowers.

4.3 Methods

4.3.1 Research design

This study employed a repeated measures crossover design for the assessment of the acute deviation and time-course to recover to baseline following exposure to three different IT sessions of varying intensity. To observe the influence of energetic contribution, three pairs of highly-trained rowers matched for performance ability but presenting either greater anaerobic (AnT) or aerobic (AeR) energetic contributions to a 6 min rowing performance test were compared. The selection criteria for pairs included: <1% difference in either 2000 m time or 6 min test mean power to allow for matched performance ability (Smith & Hopkins, 2012) and a >6.8% difference in aerobic contribution during the 6 min test because it has previously been established that the coefficient of variation for energetic contribution determined by the AOD method is 6.8% (Doherty, Smith, & Schroder, 2000). Participants first attended the laboratory to determine energetic contribution in the week prior to the experimental trials. Thereafter, participants performed one IT session per week over a period of three weeks, as shown in *Figure 4.1*.

Before and following each IT session, several recovery status measures were repeatedly assessed over a 72 h period (*Figure 4.1*).

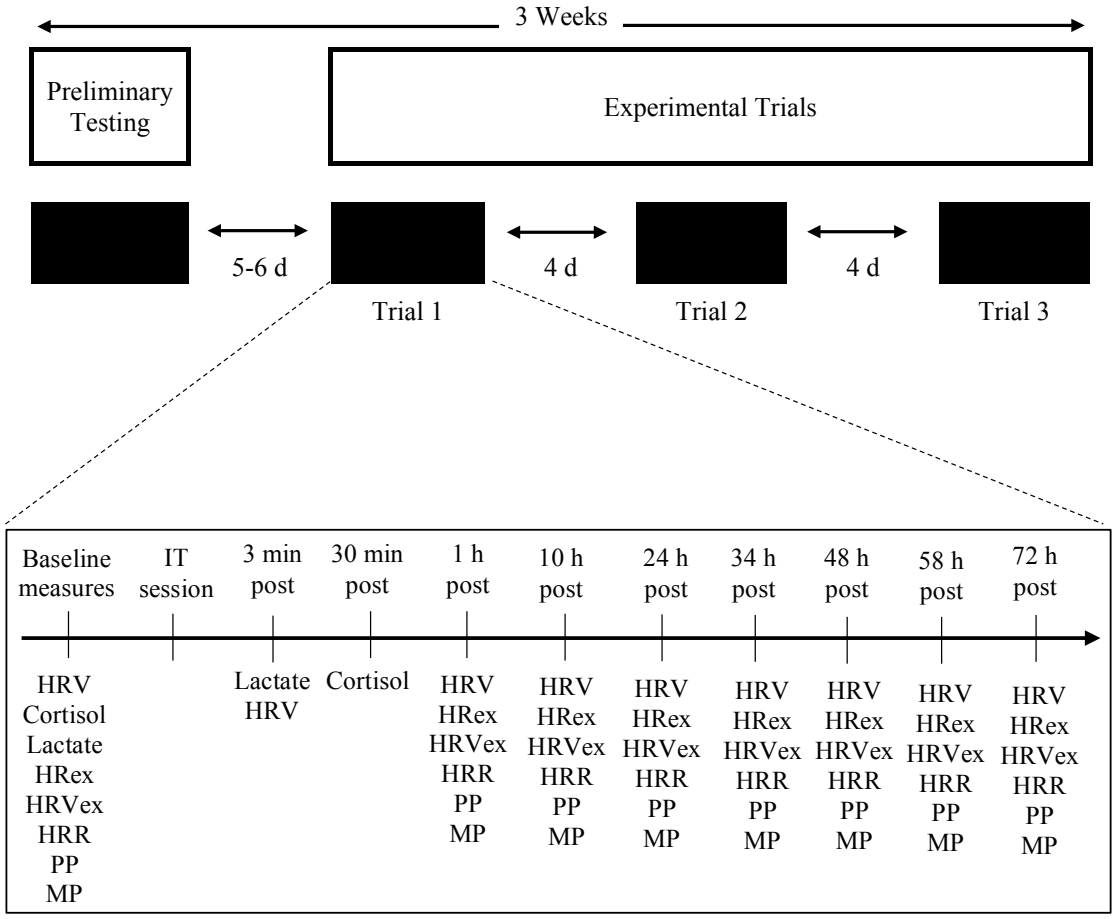

Figure 4.1 Schematic of experimental design and study timeline. Each experimental trial consisted of a randomly assigned interval training (IT) session followed by several recovery measures (in measurement order) taken throughout the 72 h post-IT period. A period of 5-6 d separated preliminary testing from the first experimental trial, with IT sessions separated by 7 days. HRV – resting heart rate variability; HRex – submaximal exercise heart rate; HRVex – post-exercise HRV; HRR – heart rate recovery; PP – modified Wingate peak power; MP – modified Wingate mean power.

4.3.2 Participants

Six highly-trained rowers (2 females, 4 males) were included in this study. All of which belonged to the same training squad. All participants had been involved in regular training over the past 6 months with an average weekly training volume of 22.5 ±1.5 h (mean ±SD) at the time of the study. Participants or legal guardians where appropriate, provided

informed consent prior to the commencement of the study. The study was approved by the Auckland University of Technology Ethics Committee (AUTEC).

Table 4.1 Participant characteristics for matched pair comparison.

Variable	Pair 1		Pair 2		Pair 3	
	AeR	**AnT**	**AeR**	**AnT**	**AeR**	**AnT**
Height (cm)	192.6	185.9	199.9	191.0	185.5	190.9
Weight (kg)	85.7	75.5	99.1	92.4	78.3	85.5
Gender	Male	Male	Male	Male	Female	Female
Aerobic contribution (%)	92.7	79.5	89.5	81.4	91.1	80.4
VO_2peak ($L \cdot min^{-1}$)	5.20	4.85	5.88	5.62	4.07	4.07
6 min mean power (W)	379	369	426	430	298	308
2000 m test time	06:26.9	06:28.5	06:03.6	06:11.8	7:01.0	7:00.2

AeR – participants with greater aerobic energetic contribution; AnT – participants with greater anaerobic energetic contribution to the 6 min rowing test.

4.3.3 Preliminary testing

All testing was performed in a temperature controlled laboratory (21 ± 0.7 °C). Following a 24 h rest period, participants performed familiarization measures for HRV and the 5'-5' test. Thereafter, participants performed a step-test consisting of 5 x 3 min submaximal stages with 20 W power increments per stage on a rowing ergometer (Concept II Model E static ergometers, Concept II Inc., Morrisville, VT). Power for the initial stage was dependant on the rower's profile (heavyweight males: 180 W; heavyweight females and lightweight men: 140 W). Stroke rate was self-selected and the drag factor was adjusted to match the rower's gender (males: 130 units; females: 110 units) in accordance with Rowing New Zealand standards. Heart rate and expired air was collected throughout the test using a metabolic gas-analysis system (ParvoMedics TrueOne 2400, Salt Lake City, UT) which was calibrated prior to all tests using alpha standard gases (BOC gases, Auckland, NZ) and a 3 L syringe (Hans Ruldolph, Shawnee, USA).

A 45 min rest period followed completion of the submaximal step-test, after which a standardized warm-up consisting of 10 min of steady-state rowing at a self-selected pace and stroke rate was performed. In the final 5 min of the warm up, participants performed three maximal 10-stroke bursts. After a 5 min preparatory period following the warm-up, participants performed a 6 min maximal rowing performance test. During the test, participants were instructed to implement the same self-regulated stroke rate and pacing strategy as they would during a 2000 m ergometer test. Expired air, power output, and HR were collected throughout the test and VO_2peak established as the highest VO_2 ($L \cdot min^{-1}$) achieved over 30 s, with VO_2peak power established as power output (W) produced over the same 30 s period. Heart rate was recorded throughout the test to establish HRmax (highest HR attained).

Energetic contribution

Oxygen uptake ($L \cdot min^{-1}$) during the final min of each stage of the step-test was plotted against actual power output (W), to establish a regression model at submaximal intensities. Extrapolation of the regression model allowed calculation of the maximal accumulated oxygen demand and uptake from the 6 min maximal test. Energetic contribution was then established as percentage aerobic energy system contribution to the test, which was calculated from the difference between accumulated oxygen demand and uptake, whilst accounting for oxygen stores (Tanner & Gore, 2013). This method has been accepted as the 'gold-standard' protocol for the quantification of an individual's energy system contributions, and established as a reliable and valid measure (Noordhof et al., 2010).

4.3.4 Experimental protocol

The experimental trials were performed over a three-week period, and consisted of three rowing ergometer IT sessions completed in random order, with each IT session separated by 7 d and performed following the same standardized 10 min warm-up previously described. The IT sessions were: 1) 5 x 3.5 min work periods; 4 min rest periods (VO_2); 2) 10 x 30 sec work periods; 5 min rest periods (Glycolytic); and 3) 5 x 10 min work periods; 4 min rest periods (Threshold). These IT sessions were selected to represent high-intensity exercise performed at: 1) maximal oxygen uptake (VO_2 IT); 2) maximal anaerobic glycolytic power (Glycolytic IT); and 3) VT_2 (Threshold IT), as would be

prescribed in a real-world rowing setting. Participants were instructed to perform all intervals at the highest maintainable pace, with active recovery between repetitions consisting of low-intensity rowing (instructed as applying minimal force whilst maintaining movement).

Heart rate was recorded throughout each IT session and analysed for time spent 80-90% and >90% HRmax, while mean power (W) of the work periods completed (i.e. excluding rest periods) was recorded from the ergometer's PM4 monitor (Concept II Inc., Morrisville, VT). Session RPE (sRPE) was recorded on IT session completion using Borg's 15-point scale (M. J. Chen et al., 2002; Christen et al., 2016). Blood samples were collected pre and 3 min post each IT session by earlobe capillary sample, and analysed immediately using a portable lactate analyser (Lactate Pro 2 LT-1730 analysers, Arkray, Tokyo, Japan). Prior to each IT session recovery measures were obtained and repeated throughout the 72 h post-IT period. The measurement timing of recovery measures is presented in *Figure 4.1*.

4.3.5 Recovery measures

The 5'-5' test

The 5'-5' test required participants to perform 5 minutes of submaximal ergometer rowing at a target power output reflective of intensities below the first ventilatory threshold (VT_1) (heavyweight males: 240 W; heavyweight females and lightweight males: 200 W; lightweight females 160 W). Followed immediately by 5 min of seated rest, for the measurement of HRex, HRR, and HRVex (Buchheit, Mendez-Villanueva, et al., 2010), performed at baseline and throughout the 72 h post-IT period (*Figure 4.1*). The ergometer drag factor was adjusted to match the rower's gender (males: 130 units; females: 110 units) in accordance with Rowing New Zealand standards, and held consistent for all ergometer tests throughout the experimental trial period.

Heart rate measures

All HR data measured throughout this study was recorded using Polar RS800CX HR monitors (Polar, Electro Oy, Kemplele, Finland) set to record R-R series at a 5 s sampling rate, and analysed via Polar Protrainer 5 Performance software (version 5.41.2, Kemplele, Findland). Ectopic beats were replaced automatically using adjacent R-R interval values.

Resting HRV was measured for 10 min on arrival in a quiet, dimly lit room at baseline and throughout the 72 h post-IT period (*Figure 4.1*). Participants were seated, maintaining a still posture (Buchheit, Al Haddad, et al., 2009). The square root of the mean sum of the squared differences between R-R intervals (rMMSD) (Task-Force, 1996) was calculated from 2:30-7:30 min of the HRV recording. HRVex was calculated as rMMSD from the ninth minute of the 5'-5' test. Seated position and noise level were held consistent with HRV measures, for accurate assessment of parasympathetic activity (Buchheit, 2014). Resting HRV measures provide a comprehensive assessment of whole body autonomic balance and therefore homeostatic perturbation. While HRVex measures provide insight into the mechanisms driving parasympathetic reactivation, including blood pressure regulation, baroreflex activity, and metaboreflex responses (Stanley et al., 2013). Submaximal exercise HR (HRex) was recorded during the 5'-5' test and was defined as the average HR over the third to fifth minute for the assessment of performance (Buchheit, Chivot, et al., 2010; Mann et al., 2013). Reductions in HRex are associated with improvements to aerobic capacity, whereas increases to HRex reflect fatigue and reduced performance capacity (Hedelin et al., 2000; Mann et al., 2013), thus providing a measure of performance recovery. Heart rate recovery (HRR) represented the absolute difference in HR across the sixth minute of the 5'-5' test, providing a measure of post-exercise withdrawal and parasympathetic reactivation throughout the recovery period (Kannankeril et al., 2004).

The modified Wingate test

The modified Wingate test consisted of a 30 sec maximal rowing ergometer effort from a stationary start, performed directly following the 5'-5' test. Peak and mean power (W), recorded using the ergometer's PM4 memory, were used as measures of performance recovery, assessed at baseline and throughout the 72 h post-IT period (*Figure 4.1*). The modified Wingate test provides a quick and valid assessment of performance recovery in rowers. Mean power produced in this test has been found to predict up to 75.7% of the variance in 2000 m ergometer test time, with peak power also demonstrating a strong relationship ($r = -0.85$) with 2000 m ergometer performance (Riechman et al., 2002).

Salivary cortisol

Salivary cortisol was measured via passive drool pre- and 30 min post-IT session. Saliva collections were obtained 10 min following mouth rinse with water, participants swallowed to clear their mouths before an unstimulated saliva sample was collected,

utilising minimal orofacial movement. Participants were instructed to consume no food or sports drink between pre and post saliva collections. Approximately 2 mL samples were collected into sterile bijou containers (7 ml-capacity with screw top, Labserve™, Auckland, NZ). Samples were stored at 4 °C for 2 h, after which they were pipetted into 2 mL-capacity 3810X Eppendorf tubes (Eppendorf, Hamburg, Germany) and subsequently frozen at -20 °C until analysis. Samples were thawed for batch analysis, and spun in a centrifuge (5424 R, Eppendorf, Hamburg, Germany) at 1000 g for 2 min. Following centrifuging, 500 μl from each sample were pipetted into 1.5 mL-capacity Hitachi cups for analysis by Roche Diagnostics™ Modular Analytics E170 instrument (Roche Diagnostics, Basel, Switzerland) at the Auckland University of Technology-Roche Diagnostics Laboratory. Salivary cortisol provides a practical and valid measure of HPA axis activity (Crewther et al., 2008; Papacosta & Nassis, 2011), reflecting the acute stress response to exercise (Adlercreutz et al., 1986).

4.3.6 Dietary and Exercise Control

Participants maintained all regular squad training sessions throughout the duration of the study, replicating the same sessions week-to-week, which were quantified using the training impulse (TRIMP) method (Foster et al., 2001), calculated from session duration (h) and sRPE. Dietary consumption for the 24 h period prior and following each IT session was recorded and replicated week-to-week. Participants were requested to refrain from consuming caffeine 12 h preceding and throughout the 72 h post-IT measurement period.

4.3.7 Statistical Analysis

IT sessions were treated as independent groups for statistical analysis, given the seven-day gap between the performance of each IT session. Pair 2 has been removed from mean data presented for VO_2 IT due to the absence of one participant from this Pair during the VO_2 IT data collection period. Descriptive statistics are shown as mean ± standard deviation (SD). Comparison of the acute post-exercise deviation between matched pairs was assessed as percentage difference in change from baseline to 3 min (blood lactate), 30 min (salivary cortisol), and 1 h post-IT (HRV, HRVex, HRex, HRR, and modified Wingate peak and mean power). Recovery time-courses were considered as the time difference between baseline and the post-IT return to baseline values. Where the measure

did not return to baseline values within the 72 h measurement period, the time-point closest to baseline was taken.

4.4 Results

Matched pair characteristics are presented in *Table 4.1*. Mean (±SD) weekly TRIMP score was 183.7 ±1.2 in the AeR pairs, and 184.6 ±1.6 in the AnT pairs. Intensity characteristics of the three IT sessions are displayed in *Table 4.2*.

4.4.1 Acute post-exercise deviation from baseline

Percentage differences in change from baseline to measures taken within 1 h post-exercise are presented in *Table 4.3*. Trends across matched pairs indicate 64.1 ±103.4 % (mean ±SD) greater response in blood lactate and 107.6 ±100.8 % greater acute reduction in mean power production in AnT across IT sessions. Whereas AeR tended to demonstrate larger deviations from baseline in HRV (17.7 ±216.2 %), HRVex (40.1 ±68.7 %), HRex (57.0 ±113.9 %), HRR (76.4 ±168.5 %), and cortisol (229.2 ±479 %) across IT sessions.

4.4.2 Recovery time-course

Percentage deviation from baseline over time in three key measures (HRV, HRex, and modified Wingate mean power) following each IT session is illustrated in *Figure 4.2*. Consistent trends between pairs for recovery to baseline time-course demonstrate 18.0 ±35.9 h (mean ±SD) longer HRVex, and 10.5 ±18.0 longer HRex durations in AeR across all IT sessions. Differences in recovery time-course dependant on energetic contribution were inconsistent between pairs for the remaining recovery variables measured, with a wide range of inter-individual variation (≥33 h) unrelated to energetic contribution evident across all recovery variables.

Table 4.2 Physiological responses to interval training sessions (mean ± SD).

Measure	VO_2 IT		Threshold IT		Glycolytic IT	
	AeR	**AnT**	**AeR**	**AnT**	**AeR**	**AnT**
sRPE	19.3 ±0.6	18.5 ±0.7	18.3 ±1.2	17.7 ±1.2	16.7 ±1.5	16.7 ±2.5
Power (W)	353.7 ±65.6	336.0 ±58.7	304.3 ±60.0	275.3 ±63.2	452.3 ±127.0	575.7 ±104
Intensity (% VO_2peak power)	94.6 ±3.1	95.5 ±9.6	81.3 ±2.4	80.6 ±8.7	155.5 ±12.9	165.7 ±10.0
TRIMP	10.8 ±0.3	10.4 ±0.4	20.2 ±1.3	19.4 ±1.3	12.5 ±1.1	12.5 ±1.9
Time >90% HR_{max} (min:s)	14:21 ±2:19	14:24 ±2:56	26:01 ±18:37	38:35 ±10:02	1:04 ±1:12	2:07 ±1:04
Time 80-90% HR_{max} (min:s)	5:52 ±1:26	9:45 ±6:22	22:56 ±16:28	13:55 ±4:34	6:48 ±1:58	7:56 ±3:39
Peak HR (% HRmax)	97.8 ±2.3	98.9 ±0.7	96.9 ±0.8	100.0 ±0.0	93.1 ±2.8	94.0 ±1.2
Blood lactate ($mmol \cdot L^{-1}$)	9.4 ±1.2	9.3 ±3.1	6.6 ±1.2	9.9 ±4.4	8.5 ±1.8	13.0 ±2.9

IT – interval training; AeR – participants presenting greater aerobic energetic contribution; AnT – participants presenting greater anaerobic energetic contribution; sRPE – session rating of perceived exertion; TRIMP – training impulse.

Table 4.3 Percentage difference between matched pairs in acute post-exercise deviation from baseline.

Matched Pairs	IT session	sRPE	Cortisol	Blood Lactate	HRV	HRVex	HRex	HRR	Peak power	Mean power
		Percentage difference between pairs in the acute post-exercise deviation from baseline (%)								
Pair 1	VO_2	-10.0	-37.0	19.3	-25.4	-122.6	-122.2	-387.5	-66.7	22.9
AeR vs.	Threshold	-10.5	-430.0	-24.6	81.7	76.6	~	~	~	~
AnT	Glycolytic	13.3	-67.0	16.7	167.1	-85.8	~	-88.2	-79.6	147.4
Pair 2	VO_2	~	~	~	~	~	~	~	~	~
AeR vs.	Threshold	0.0	-121.3	17.5	-476.4	-2.5	-83.3	166.7	20.0	212.9
AnT	Glycolytic	28.6	44.4	-45.6	245.0	34.9	166.7	-150.0	~	~
Pair 3	VO_2	0.0	-1321.1	131.7	-37.9	-69.2	-120.0	-12.5	70.6	-20.0
AeR vs.	Threshold	0.0	204.7	260.6	-20.6	-82.7	-50.0	-51.9	-41.4	175.0
AnT	Glycolytic	11.8	-106.3	137.3	-75.1	-69.2	-133.3	-11.1	~	~
Mean		4.2	-229.2	64.1	-17.7	-40.1	-57.0	-76.4	-19.4	107.6
SD		13.1	476.0	103.4	216.2	68.7	113.9	168.5	63.2	100.8

Acute deviation is represented as change in pre-post scores following three different interval training (IT) sessions (VO_2, Threshold and Glycolytic). Change scores calculated from pre- post measures. IT – interval training; sRPE – session rating of perceived exhaustion; HRV – resting heart rate variability; HRVex – post-submaximal exercise heart rate variability; HRex – submaximal exercise heart rate; HRR – heart rate recovery; AeR – participants with greater aerobic energetic contribution; AnT – participants with greater anaerobic energetic contribution; ~ missing data.

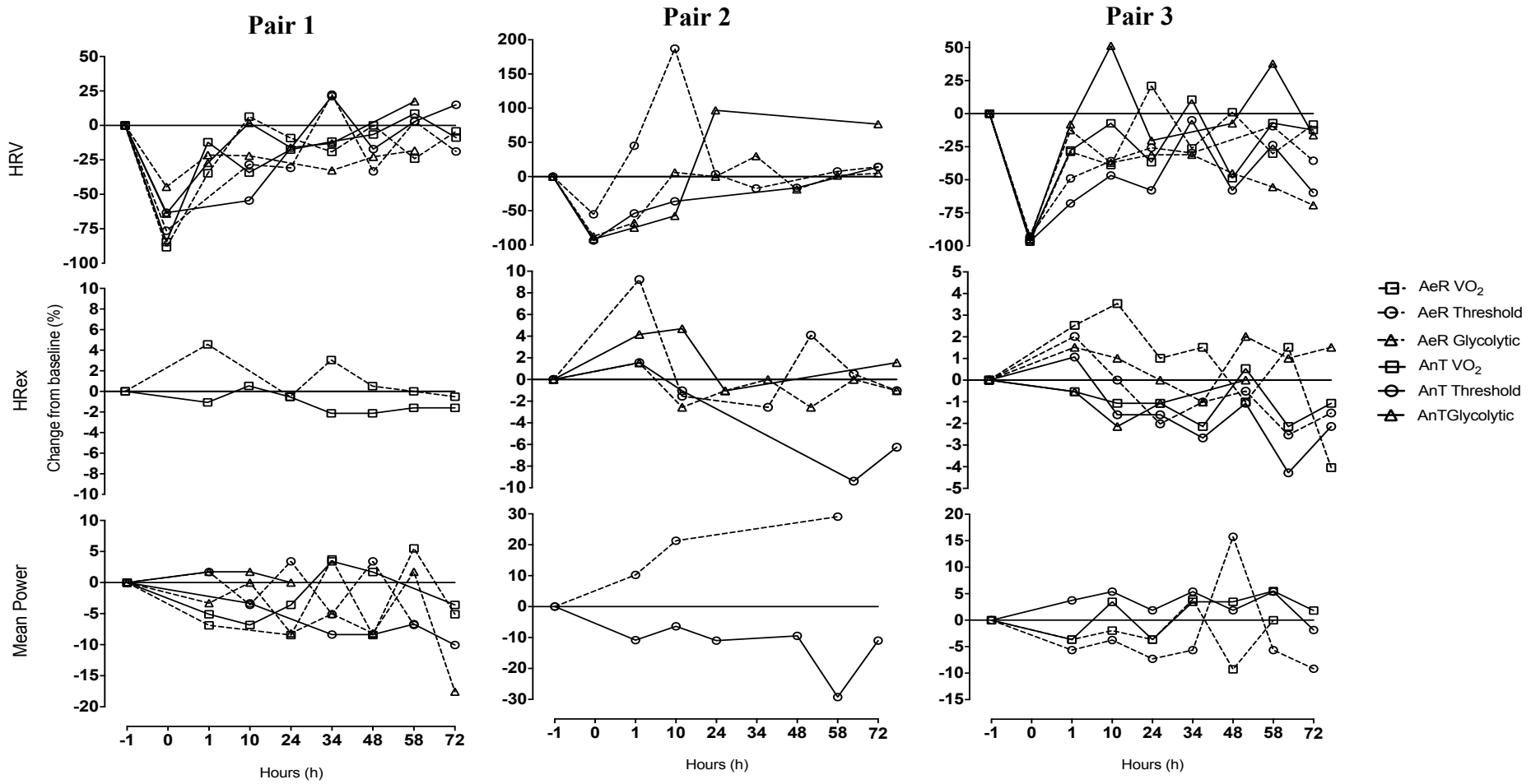

Figure 4.2 Percentage deviation from baseline over time. HRV – resting heart rate variability; HRex – submaximal exercise heart rate; AeR – participants with greater aerobic energetic contribution; AnT – participants with greater anaerobic energetic contribution. Measured following VO_2, Threshold, and Glycolytic interval training sessions.

4.5 Discussion

The aim of the present study was to investigate the influence of energetic contribution on the magnitude of acute deviation and time-course of return to baseline following three different high intensity IT sessions. The main findings of this study suggest that AnT demonstrate greater utilisation of anaerobic energy sources, whereas AeR elicit greater acute suppression of parasympathetic activity. However, these findings did not translate to recovery time-course, with limited differences observed between AeR and AnT. Furthermore, a wide range of inter-individual variation unrelated to energetic contribution appears to exist in recovery time-course.

4.5.1 Acute post-exercise deviation from baseline

In the present study participants with superior anaerobic contributions were hypothesised to experience greater acute deviation from baseline in all recovery variables assessed. Indeed, the 64.1 ±103.4% (mean ±SD) greater blood lactate accumulation and 107.6 ±100.8% greater decrement in modified Wingate mean power observed in AnT across IT sessions supports this hypothesis. These findings illustrate AnT as generating enhanced utilisation of non-oxidative pathways in comparison to AeR. Although surprisingly, these findings were not observed consistently across all three pairs, with similar responses only observed in blood lactate following VO_2 IT, and mean power decrement following Threshold IT (*Table 4.3*)—which may be due to the comparison of just two pairs for these measures.

Nevertheless, the current findings of greater neuromuscular fatigue and blood lactate accumulation in AnT may be reflective of a greater distribution and subsequent recruitment of type II muscles fibres (Costill et al., 1976), higher muscle glycogen, ATP and PCr content, and enhanced anaerobic enzyme activity (Casey et al., 1996; Greenhaff et al., 1994), contributing to a greater ability to induce homeostatic perturbation in these participants (Hamada et al., 2003). Furthermore, our findings are consistent with the work of Garrandes et al. (2007) who found power athletes to have a 25% greater decrement in concentric knee-extension torque than their endurance-trained counterparts following fatiguing exercise. Similarly, Paavolainen et al. (1994) found a 40.5% greater blood lactate response following high-intensity anaerobic intervals to correspond with a larger impairment of muscle contractile capacity in sprint athletes compared to endurance-trained athletes. Although, these studies compare athletes of differing training histories,

the present study appears to be the first to compare athletes presenting similar training histories and matched for performance ability. Therefore indicating factors other than training history and performance ability as responsible for the differences observed in anaerobic function between participants presenting differing energetic contributions (Schumacher & Mueller, 2002; Simoneau & Bouchard, 1995).

The current finding of larger acute HRV (17.7 ±216.2%; mean ±SD) and HRVex (40.1 ±68.7%) suppression, as well as HRR acceleration (57.0 ±113.9%) and salivary cortisol response (229.2 ±479%) in AeR following all IT sessions was unexpected. This is given similar studies examining the acute suppression of cardiac parasympathetic activity in athletes with differing energetic profiles report positive associations between cardiac parasympathetic withdrawal and anaerobic profile (McDonald, 2014; Del Rosso, 2016). Specifically, McDonald et al. (2014) observed an 8% slower HRR in anaerobically trained track cyclists than their aerobically trained road cyclist counterparts, following a VO_2max test. Furthermore, Buchheit et al. (2007) found anaerobic contribution, metabolite accumulation, and factors involved with type II muscle fibre recruitment to have a significant influence on parasympathetic suppression following high-intensity exercise. Previous findings of larger cardiac parasympathetic inhibition following exercise in athletes with greater anaerobic contributions can be explained by the greater metaboreflex stimulation likely occurring in these athletes (Buchheit, Chivot, et al., 2010; Buchheit & Gindre, 2006; Buchheit et al., 2007; Huang et al., 2007; Seiler et al., 2007). However, the present study illustrates a greater accumulation of blood lactate in AnT following Threshold and Glycolytic IT (*Table 4.2)*, demonstrating higher metabolic stress induced in AnT. Furthermore, previous research demonstrates time spent at HR intensities reflective of $\geq VT_2$ to be related to catecholamine and cortisol accumulation (E. Hill et al., 2008; Kindermann et al., 1982; Manetta et al., 2005; Urhausen et al., 1994), reflecting a larger sympathetic response and corresponding parasympathetic suppression (Christensen & Galbo, 1983; Perini et al., 1989). However, our results illustrate AnT as achieving higher peak HR across all IT sessions, with significantly longer time spent >90% HRmax during Threshold, and at 80-90% HRmax during VO_2 IT sessions (*Table 4.2*). Thus, it appears mechanisms other than metaboreflex and time spent close to HRmax to explain the greater cortisol response and inhibition of parasympathetic activity observed in AeR following most IT sessions in the present study.

4.5.2 Recovery time-course

The current study observed an 18.0 ±35.9 h (mean ±SD) longer recovery time-course for HRVex and 10.5 ±18.0 h longer for HRex in AeR across IT sessions, with measures of HRV, HRR, mean and peak power displaying a wide (≥33 h) range of inter-individual variation between pairs. The tendency for longer HRVex recovery time-course in AeR corresponds to the 40.1 ±68.7% (mean ±SD) larger acute suppression of this variable observed in AeR. This observation agrees with the findings presented in Chapter Three (*Table 3.6*), and that of previous studies, whereby greater HRV suppression measured 20 min (Parekh & Lee, 2005) and 1 h (Niewiadomski et al., 2007) post-exercise was found to be associated with longer recovery-time courses. It can be presumed that the extended recovery time-course of HRVex observed in the present study is resultant from the greater acute deviation from baseline observed in this variable (Niewiadomski et al., 2007; Parekh & Lee, 2005). However, the same association is not apparent for HRex, nor HRV and HRR, which both illustrated similar acute deviations to HRVex in AeR (*Table 4.3*). In addition, given the highly variable nature of HRVex (Buchheit, 2014; Buchheit et al., 2008), the degree of acute deviation from baseline in AeR does not appear to be related to recovery time-course in the current study.

The longer HRVex and HRex recovery time-course observed in AeR, as well as the wide range of inter-individual variation unrelated to energetic contribution observed in the recovery time-course of HRV, HRR, mean and peak power does not support our hypothesis of AeR illustrating earlier recovery across all measures. However, the greater acute suppression of cardiac parasympathetic activity evident in AeR (*Table 4.3*) suggests these participants possess a faster rate of autonomic recovery, thus restoring the greater acute cardiac parasympathetic suppression within a similar time-course to that of AnT. Adaptations to blood flow dynamics and heart structure related to aerobic energetic contribution are likely responsible for the enhanced rate of autonomic recovery observed in AeR, via improving thermoregulation, metabolite clearance, and blood pressure regulation (Douglas, O'Toole, Hiller, Hackney, & Reichek, 1987; Greenhaff, 1989; MacRae, Dennis, Bosch, & Noakes, 1992). Further, these parameters are associated with cardiac parasympathetic activity (Buchheit, Laursen, et al., 2009; Buchheit et al., 2007; Buchheit, Voss, Nybo, Mohr, & Racinais, 2011b; Convertino, 2003). Nevertheless, the current study demonstrates greater aerobic energetic contributions, when matched for performance ability and training history, do not correspond to earlier absolute recovery

of autonomic or performance parameters in comparison to participants presenting greater anaerobic energetic contributions. Therefore, individualising the programing of high-intensity interval sessions in relation to energetic profile and recovery time-course is unlikely to have any practical benefit. Rather, given the wide degree of inter-individual variation in recovery time-course unrelated to energetic profile observed in the current study, the individual monitoring of athletes on a case-by-case basis appears to be the most appropriate method for optimising training programming (Kiviniemi et al., 2007; Plews, Laursen, Stanley, Kilding, & Buchheit, 2013).

4.5.3 Limitations and future research

To our knowledge no previous research has investigated the influence of energetic profile on recovery time-course in participants matched for performance capacity. Matching participants for performance capacity proves valuable due to the influence of cardiovascular fitness on recovery time-course (Hautala et al., 2001; Seiler et al., 2007), as well as the positive association between aerobic contribution and performance capacity (own unpublished observation). However, the performance measure used to match performance capacity may influence the results observed. For example, in rowing ergometer performance does not necessarily correspond to on-water performance (Mikulić, Smoljanović, Bojanić, Hannafin, & Matković, 2009), therefore a more appropriate means of matching performance capacity in rowers may be via weight adjusted ergometer performance (Nevill, Beech, Holder, & Wyon, 2010). Although, in the current study weight adjusted mean 6 min power output limited the number of participants who met the inclusion criteria to just one pair, preventing the observation of trends related to energetic contribution across pairs. Furthermore, the correlations between recovery time-course and energetic contribution performed in Chapter 3 revealed very limited differences when controlling for either weight adjusted ergometer performance or raw ergometer performance (***Figure 3.3***). Nevertheless, given the lack of alternative analysis methods in the literature, future research examining the influence of energetic contribution on recovery time-course should investigate matching participants using alternative variables of performance capacity.

Given the influence of cardiovascular fitness on recovery time-course (R. Fry et al., 1991; Hautala et al., 2001; Seiler et al., 2007), in order to limit confounding factors in the comparison of recovery time-course in differing energetic contributions, inclusion criteria

for matched pairs in the current study required <1% difference in performance ability and >6.8% difference in energetic contribution. These inclusion criteria considerably limited the sample size available for investigation, consequently restricting statistical analysis and likely contributing to the wide sample variation observed. Furthermore, it can be argued that the inclusion of two performance variables (the 6 min and 2000 m rowing tests) does not reflect pairs equally matched for performance ability. Nevertheless, although differences between pairs on both tests were not consistently less than 1%, differences did not exceed 3.3%.

Therefore, future research examining a larger cohort of participants equally matched for performance ability, while presenting differing energetic contributions is necessary to confirm the current study's findings. However, a large degree of difficulty is associated with obtaining such a sample due to the strict requirements regarding performance ability, given participants presenting greater aerobic contributions likely possess greater performance abilities than those presenting greater anaerobic contributions (Riechman et al., 2002). Additionally, the present study examined highly trained rowers from the same training squad. Whereas future research investigating a wider population of participants from other intermediate duration sports, such as middle distance running, track cycling, and flatwater kayak would further the current understanding regarding the influence of energetic contribution on recovery time-course in athletes of similar training histories.

4.6 Conclusion

The present study provides evidence that endurance trained rowers with greater anaerobic energetic contributions demonstrate greater utilisation of non-oxidative pathways during VO_2 IT sessions. Additionally, participants demonstrating greater aerobic energetic contributions illustrated greater acute suppression of cardiac parasympathetic activity following all IT sessions. However, these findings did not translate to recovery time-course, with AeR demonstrating consistently longer times to recover following all IT sessions in HRVex and HRex alone. While a wide degree of inter-individual variation unrelated to energetic contribution was evident in recovery time-course for all other recovery variables. These findings provide insight into variances in the recovery characteristics of athletes presenting differing energetic contributions. Whereby AeR appear to demonstrate a faster rate of autonomic recovery, given the greater acute suppression of cardiac parasympathetic activity in AeR, but lack of consistent difference in absolute time to recover autonomic balance between AeR and AnT. Consequently,

there appears to be no apparent benefit of programming training based on energetic profile; rather, an individualised approach to monitoring recovery is recommended.

Chapter Five: Discussion and conclusion

5.1 Summary of findings

Adaptation to training is maximised when the appropriate balance between fatigue and recovery is achieved. Thus emphasising the importance of the effective programming of training within a microcycle, as the chronic programming of subsequent training sessions without sufficient recovery periods can lead to maladaptation and overtraining (Meeusen et al., 2013). This is particularly the case in endurance sport where high training frequencies of up to three times per day limit the time available for recovery between training sessions (Fiskerstrand & Seiler, 2004; Seiler, 2010). Therefore, knowledge of the time-course for recovery following various training sessions, and the factors influencing recovery time-course is valuable for the optimisation of training programming. The objective of this thesis was to extend the current knowledge regarding the optimal recovery period preceding subsequent high-intensity training sessions, and to gain insight into factors influencing recovery time-course.

The literature review in this thesis did not reveal any studies investigating recovery time-course over a non-passive recovery period, as is typical of many endurance sports such as rowing (Fiskerstrand & Seiler, 2004; Seiler, 2010). Additionally, studies examining the influence of energetic contribution on recovery proved sparse, with none identified to observe time-course for recovery (i.e. time to return to baseline). Furthermore, although a range of physiological mechanisms are involved in restoring post-exercise homeostatic perturbation (Seiler et al., 2007), a lack of research investigating differences in recovery time-course between measures exists. Further research in this area is warranted given a discordance exists between the recovery rate of various factors involved in the recovery process (Andersson et al., 2008; Krustrup et al., 2011; Saw et al., 2015).

In light of the limitations identified in the literature, Chapter Three investigated the recovery time-course from three interval training sessions of differing intensities over a 72 h recovery period. Whereby subsequent training sessions reflective of a typical rowing program were performed throughout (*Table 3.2*), rather than prescribing rest, as performed in previous studies (Kaikkonen et al., 2008; Mourot et al., 2004; Niewiadomski et al., 2007; Parekh & Lee, 2005; Seiler et al., 2007; Terziotti et al., 2001). A range of recovery measures were assessed throughout the 72 h recovery period, providing insight into differences in the recovery time-course of autonomic balance,

performance, and perceptual parameters. This study observed Threshold IT to require the longest recovery period for parasympathetic activity, whereas VO_2 and Glycolytic IT, although performed at higher intensities (*Table 3.2*) required substantially less time to return to baseline values (*Table 3.5*). These findings are in conflict with previous literature, whereby studies comparing the recovery of parasympathetic activity following differing exercise intensities demonstrate a positive association between intensity and recovery time-course (Mourot et al., 2004; Niewiadomski et al., 2007; Seiler et al., 2007). Conversely, the results presented in Chapter Three indicate time spent at intensities reflective of $>VT_2$ to be responsible for recovery time-course, with autonomic recovery time-course reflecting IT session duration spent 80-90% and >90% HRmax (*Table 3.2*).

Although previous research illustrates little, if any, influence of exercise duration on the time-course of autonomic recovery when controlling for intensity (Bernardi et al., 1997; Hautala et al., 2001; Seiler et al., 2007; Stanley et al., 2013; Terziotti et al., 2001), such studies have only examined the relationship between exercise duration and autonomic recovery time-course at intensities bellow VT_1 (Seiler et al., 2007). While VT_1 appears to mark a threshold for further increases in the acute post-exercise deviation from baseline (Jacks et al., 2002; Nieman et al., 1999; Plews et al., 2014; Seiler et al., 2007), VT_1 may also present a threshold for further increases to autonomic recovery time-course. With greater time spent close to HRmax corresponding to a greater sympathetic activation, resultant catecholamine release, and corresponding parasympathetic suppression prolonging recovery time-course (Christensen & Galbo, 1983; Urhausen et al., 1994). Which is supported by the *very large* and *large* correlations observed between acute autonomic deviation and recovery time-course to baseline in Chapter Three (*Table 3.7*).

Discordance existed between the various measures of recovery assessed in Chapter Three, with performance measures illustrating substantially faster recovery time-courses than that of autonomic balance across all IT sessions (*Table 3.5*). The longer recovery time-course of parasympathetic activity compared to that of performance measures likely reflects the comprehensive nature of parasympathetic activity. Although the physiological mechanisms underlying cardiac parasympathetic reactivation following exercise are not wholly known, its recovery kinetics appear to parallel many physiological systems involved in exercise recovery including the thermoregulatory and vascular systems (Aubert et al., 2003; Stanley et al., 2013). Interestingly, however, HRVex consistently illustrated longer recovery time-courses than HRV across all IT sessions.

This discrepancy is likely related to the differing mechanisms driving the recovery of the two measures, whereby metaboreflex stimulation is a key determinant of HRVex status (Buchheit et al., 2007), whereas baroreflex stimulation is a prominent regulator of HRV status at rest (Aubert et al., 2003). Furthermore, although it can be reflective of recovery status, HRVex proves a complex measure and is influenced by multiple mechanisms, with exercise intensity having a large effect (Buchheit et al., 2007), and is thus deemed to be a less effective measure of autonomic balance than HRV (Buchheit et al., 2012; Buchheit et al., 2013). In contrast, HRR demonstrated a reduced recovery time-course compared to HRV, across all IT sessions. Heart rate recovery provides a measure of sympathetic withdrawal and parasympathetic reactivation, rather than complete autonomic recovery as represented by HRV. Furthermore, while HRR is regulated by cardiac output, blood pressure regulation, and metaboreflex stimulation (Buchheit et al., 2007), a lack of association has been found between HRR and post-exercise changes in these mechanisms (Buchheit, Voss, Nybo, Mohr, & Racinais, 2011a), which may further explain its earlier return to baseline, compared to other measures of parasympathetic status reported in Chapter 3. In addition, the large CV (~25.7) associated with HRR (Al Haddad et al., 2011), makes true variation from baseline difficult to identify for this measure.

In addition, measures of perceived recovery illustrated no return to baseline throughout the 72 h measurement period, following all IT sessions. This can be explained by the non-passive recovery period reflective of that typically performed in a real-world setting by highly performing rowers, whereby the first programmed rest session occurred 82 h post-IT, whereas each IT session was preceded by a 24 h rest period (*Table 3.2*). Psychometric measures have previously been shown to more sensitive to training load than physiological measures (Saw et al., 2015). Thus, the accumulation of training load throughout the 72 h measurement period explains why participants' ratings of overall fatigue, leg soreness, mental recovery, and physical recovery remained elevated without returning to their post-rest day baseline scores. These findings highlight the importance of programming rest days, or days where training load is substantially reduced, for the prevention of fatigue accumulation.

Finally, Chapter Three examined the relationship between recovery time-course and energetic contribution while controlling for performance ability. Such a relationship appears promising given previous data has revealed a relationship to exist between energetic contribution and the acute sympathetic response, assessed as HRR in track and

road trained cyclists (McDonald et al., 2014). Furthermore, knowledge of the influence of energetic contribution on recovery time-course would benefit the individualisation of training programming based on recovery needs. This analysis produced some evidence supporting an influence of energetic contribution on recovery time-course, with greater aerobic contributions illustrating longer recovery time-courses for HRex following Threshold (r = 0.53 ±0.51; mean ±CL), and HRVex following Glycolytic IT (r = 0.36 ±0.47), when controlling for raw 6 min maximal test performance. Although interestingly, greater aerobic contributions also demonstrated reduced recovery time-courses for HRR following Threshold IT (r = -0.52 ±0.51). In addition, the same relationship was examined while controlling for weight adjusted 6 min maximal test, however, very little difference between the two conditions was observed (***Figure 3.3***), with greater aerobic contributions additionally demonstrating reduced recovery time-courses for modified Wingate peak power following Threshold IT (r = -0.55 ±0.46). These findings indicate the influence of energetic contribution on recovery time-course to be limited and inconclusive, whether controlling for raw or weight adjusted performance ability.

The impact of energetic contribution on recovery time-course for some measures led to a more in-depth investigation of energetic contribution influence on recovery time-course, presented in Chapter Four, whereby AeR and AnT participants matched for performance ability were compared. In agreement with the findings of Chapter Three, a lack of association between energetic contribution and recovery time-course was evident for most variables assessed. However, AeR demonstrated greater acute HRV, HRVex, and HRR deviation from baseline following all IT sessions (*Table 4.3*). Furthermore, the *large* and *very large* correlations observed between recovery time-course and the acute deviation from baseline across most recovery variables in Chapter Three (*Table 3.6*) were not reflected in Chapter Four, with only HRVex and HRex illustrating longer recovery time-courses in AeR. The findings from these studies do not support our hypothesis of AnT demonstrating longer recovery time-courses following all three IT sessions. Rather, our results suggest AeR (when matched for raw performance ability) possess a greater capacity to induce parasympathetic suppression, but yet recover at a faster rate than AnT, given differences in recovery time-course between AeR and AnT were sparse and not consistently related to the acute deviation from baseline.

5.2 Limitations of the research

Several limitations should be considered in the interpretation of findings from this thesis:

1) Recovery was examined across an acute time period in the studies presented in this thesis. However, examination of a longer time period allowing for participants to perform each IT session multiple times, and fatigue accumulation to occur, would have benefited the results' reproducibility, while shedding light on whether participants respond differently over time or to the same repeated stimulus.

2) The sample size of the studies undertaken in this thesis are somewhat limited due to the inclusion criteria of participants from the same regional performance centre squad, and presenting <6:40 (males) and <7:30 min:sec (females) during a 2000 m rowing ergometer test, as well as present <1% difference in performance ability in Chapter Four. In addition, a variety of methods for deriving pairs was explored, including separating AeR and AnT participants based on aerobic contributions >1 SD either side of the mean, and matching pairs based on weight-adjusted 6 min performance or weight-adjusted 2000 m performance. These methods of deriving pairs may have provided a more appropriate comparison of differing energetic profiles matched for performance ability. However, these criteria restricted sample size to just one pair in Study Two, preventing the observation of trends related to energetic contribution across pairs. Furthermore, some participants did not complete all three IT sessions, likely weakening the results observed.

3) The transferability of recovery time-course findings from the studies presented in this thesis are limited to highly trained athletes (VO_2peak ~4.9 L·min^{-1}) given the inverse association between VO_2max and autonomic recovery time-course (Hautala et al., 2001; Seiler et al., 2007). Also likely to limit the transferability of findings to a wider population is the programming of subsequent training sessions, as the non-passive recovery period performed (*Table 3.2*) is expected to have influenced the recovery time-course of parasympathetic activity (Carter et al., 2002; Kingsley et al., 2014; Seiler et al., 2007).

4) Lifestyle factors including sleep quantity (Samuels, 2009), psychological stress (Perna & McDowell, 1995), and diet (Hawley et al., 2011) have been shown to influence physiological mechanisms involved in the recovery process. Due to the

difficulties associated with the quantification and control of these factors, they were not controlled for in the studies presented in this thesis and therefore any confounding effect cannot be established.

5) The inclusion of both male and female participants in the studies presented in this thesis may have influenced the results obtained. Females have previously been shown to demonstrate delayed cardiac parasympathetic reactivation following high-intensity exercise in comparison to males (Kiviniemi et al., 2010; Mendonca et al., 2010). Furthermore, blood lactate response has been found to be influenced by menstrual cycle phase, although menstrual cycle phase was not recorded in the current studies and therefore any influence cannot be accounted for.

6) No wholly comprehensive measure of homeostatic recovery currently exists. Although cardiac parasympathetic activity represents the return of many physiological systems it has not been found to account for glycogen repletion (Stanley et al., 2013), which was not assessed in the studies presented in this thesis. Furthermore, cardiac parasympathetic assessment was achieved indirectly via HRV, HRVex, HRex, and HRR, as direct assessment via nerve activity was deemed impractical for the studies presented.

7) Lastly, the studies presented in this thesis could be criticised for a lack of exercise intensity standardisation, as IT sessions were instructed to be performed at the highest maintainable pace rather than relative to VO_2peak or the ventilatory thresholds. Instead, interval durations were carefully selected to represent high-intensity exercise performed at 1) VT_2 (Threshold IT); 2) maximal anaerobic glycolytic power (Glycolytic IT); and 3) maximal oxygen uptake (VO_2 IT). Thus reflecting three types of typically programmed high-intensity training sessions within rowing, that—in a real world setting—would be performed as prescribed in the current studies. Nevertheless, retrospective quantification of mean IT power (W) revealed IT sessions were performed relative to target intensities (Threshold IT: 79.6 ±6.8 %; VO_2 IT: 97.0 ±5.4 %; Glycolytic IT: 156.4 ±15.6 % VO_2peak power) (Meyer et al., 1999).

5.3 Practical applications

The disparity observed between the recoveries of differing parameters in Chapter Three emphasises the need for a complimentary holistic approach to the assessment of recovery status in well-trained athletes. The recovery of performance parameters occurred substantially earlier following all IT sessions than that of autonomic balance in Chapter Three, indicating that the recovery of performance indices does not necessarily correspond to the return of pre-exercise homeostatic functioning. Furthermore, the lack of REC-Q recovery questions the validity of measuring perceived recovery from a specific session throughout a non-passive recovery period, due to the sensitivity of this measure to training load accumulation (Saw et al., 2015). Chapter Three revealed the assessment of autonomic balance to be the most comprehensive measure of recovery status, with its recovery occurring after that of all other parameters assessed. Specifically, from the results obtained in this thesis HRV is deemed the most appropriate measure for the assessment of post-exercise homeostatic return, given it proves the most practically applicable and reliable method for the assessment of cardiac parasympathetic activity (Al Haddad et al., 2011).

The findings in this thesis provide evidence that time to recover to baseline following a single training session in highly trained rowers during a typical training week is longest following Threshold IT, requiring 29.2 ±12.1 h (mean ±CL). Whereas, time to return to baseline following Glycolytic and VO_2 IT requires 17.8 ±9.6 h and 15.7 ±11.2 h respectively. These time periods should be considered in the programming of subsequent high-intensity sessions for athletes of similar cardiovascular fitness. Furthermore, the findings of this thesis differ to that previously reported, with Glycolytic and VO_2 IT demonstrating more rapid recoveries of parasympathetic activity than that previously reported for high-intensity exercise (Stanley et al., 2013). This is likely due to the non-passive recovery period employed in the current studies, with participants continuing their normal squad training sessions, as would occur in a real-world setting. In light of these differences, the current findings indicate low-intensity exercise programmed in subsequent sessions reduce the time taken for recovery to baseline, and thus may aid in minimising fatigue accumulation and maximising the adaptive response when applied in the programming of training sessions.

Lastly, there appears to be little practical benefit in the use of energetic contribution to guide training programming with regard to the time period required for recovery between

high-intensity interval sessions. As although AeR demonstrated greater acute suppression of parasympathetic activity in Chapter Four, this did not translate to a difference in recovery time-course between AeR and AnT. Furthermore, Chapter Three revealed the relationship between energetic contribution and recovery time-course across a range of recovery variables to be limited and inconclusive. Rather, athletes should be monitored on an individual basis to inform training programming—although time consuming—this approach appears to be the most beneficial for maximising the adaptive response (Kiviniemi et al., 2007; Plews et al., 2013; Vesterinen et al., 2016).

5.4 Future research

No other research appears to exist investigating recovery time-course following a key exercise stimulus throughout a non-passive recovery period. Given the high training frequencies of successful athletes (Fiskerstrand & Seiler, 2004; Seiler et al., 2007) such information proves valuable for the optimisation of training programming, therefore further research investigating recovery time-course throughout a non-passive recovery period would be beneficial in substantiating the findings of this thesis. Furthermore, it is suggested that the performance of low-intensity exercise following a strenuous exercise stimulus enhances mechanisms involved in recovery and therefore hastens recovery time-course; however, evidence supporting this claim is limited to that presented in this thesis. Thus prospective studies examining the influence of subsequent exercise and its intensity on the time-course of homeostatic return is required to confirm the presented findings.

Additionally, although energetic contribution appeared promising for the individualisation of training programming, the studies presented in this thesis did not reveal any definitive relationship between energetic contribution and recovery time-course in athletes matched for performance ability. Furthermore, no other studies appear to have examined the relationship between recovery time-course and energetic contribution. Therefore, although the studies presented in this thesis disregard any practically applicable influence of energetic contribution on recovery time-course, additional research employing a wider sample size of participants matched for alternative variables of performance ability is needed to validate the current findings.

5.4 Conclusion

The aim of this thesis was to extend the current knowledge regarding the optimal recovery period preceding subsequent high-intensity training sessions, and to gain insight into factors influencing recovery time-course. The studies presented in this thesis are the first to examine recovery time-course throughout a non-passive recovery period, whereby subsequent programmed training sessions were reflective of that typically performed in a real-world setting by highly performing rowers, and therefore provided a real-world assessment of recovery. Additionally, no other research has investigated the influence of energetic contribution on homeostatic recovery time-course.

The key findings of this thesis were that highly trained rowers during a normal training week were found to require 29.2 ±12.1 h (mean ±CL) post-Threshold, 17.8 ±9.6 h post-Glycolytic and 15.7 ±11.2 h post-VO_2 IT for the recovery of parasympathetic activity. Additionally, a relationship between the acute deviation and recovery time-course to baseline was observed, with greater indices of acute deviation corresponding to longer recovery time-courses. While time spent close to HRmax reflected recovery time-course, indicating the existence of a durational effect on time to recover following exercise performed at HR intensities reflective of $\geq VT_2$. Finally, limited and inconclusive evidence supporting the influence of energetic contribution on recovery time-course was observed, with AeR demonstrating greater acute parasympathetic suppression but no difference in recovery time-course compared to their AnT counterparts. Suggesting AeR possess a greater ability to induce acute parasympathetic suppression and enhanced capacity for parasympathetic recovery. However, this lack of difference in recovery time-course revealed energetic contribution to have limited practical influence on individualising the programming of high-intensity interval sessions with regards to the time-course of recovery between acute sessions. Nevertheless, these findings contribute to the literature regarding the time-course for homeostatic recovery following typically performed training sessions, with the objective to inform optimal training programming strategies for highly-trained endurance athletes.

Chapter Six: References

Acevedo, E. O., Kraemer, R. R., Kamimori, G. H., Durand, R. J., Johnson, L. G., & Castracane, V. D. (2007). Stress hormones, effort sense, and perceptions of stress during incremental exercise: an exploratory investigation. *The Journal of Strength & Conditioning Research, 21*(1), 283-288.

Adlercreutz, H., Härkönen, M., Kuoppasalmi, K., Näveri, H., Huhtaniemi, I., Tikkanen, H., . . . Karvonen, J. (1986). Effect of training on plasma anabolic and catabolic steroid hormones and their response during physical exercise. *International Journal of Sports Medicine, 7*, 27-28.

Ahtiainen, J. P., Pakarinen, A., Kraemer, W. J., & Häkkinen, K. (2003). Acute hormonal and neuromuscular responses and recovery to forced vs maximum repetitions multiple resistance exercises. *International Journal of Sports Medicine, 24*(6), 410-418.

Al Haddad, H., Laursen, P., Chollet, D., Ahmaidi, S., & Buchheit, M. (2011). Reliability of resting and postexercise heart rate measures. *International Journal of Sports Medicine, 32*(8), 598.

Allemeier, C. A., Fry, A. C., Johnson, P., Hikida, R. S., Hagerman, F. C., & Staron, R. S. (1994). Effects of sprint cycle training on human skeletal muscle. *Journal of Applied Physiology, 77*(5), 2385-2390.

Andersson, H. M., Raastad, T., Nilsson, J., Paulsen, G., Garthe, I., & Kadi, F. (2008). Neuromuscular fatigue and recovery in elite female soccer: effects of active recovery. *Medicine and Science in Sports and Exercise, 40*(2), 372-380.

Andrew, G. M., Guzman, C. A., & Becklake, M. R. (1966). Effect of athletic training on exercise cardiac output. *Journal of Applied Physiology, 21*(2), 603-608.

Apple, F. S., Rogers, M. A., Casal, D. C., Lewis, L., Ivy, J. L., & Lampe, J. W. (1987). Skeletal muscle creatine kinase MB alterations in women marathon runners. *European journal of applied physiology and occupational physiology, 56*(1), 49-52.

Armstrong, R., Warren, G., & Warren, J. (1991). Mechanisms of exercise-induced muscle fibre injury. *Sports Medicine, 12*(3), 184-207.

Artioli, G. G., Bertuzzi, R. C., Roschel, H., Mendes, S. H., Lancha Jr, A. H., & Franchini, E. (2012). Determining the contribution of the energy systems during exercise. *JoVE (Journal of Visualized Experiments)*(61), e3413-e3413.

Aubert, A. E., Seps, B., & Beckers, F. (2003). Heart rate variability in athletes. *Sports Medicine, 33*(12), 889-919.

Ba, A., Delliaux, S., Bregeon, F., Levy, S., & Jammes, Y. (2009). Post-exercise heart rate recovery in healthy, obeses, and COPD subjects: relationships with blood lactic acid and PaO2 levels. *Clinical Research in Cardiology, 98*(1), 52-58.

Bangsbo, J. (1998). Quantification of anaerobic energy production during intense exercise. *Medicine and science in sports and exercise, 30*(1), 47-52.

Bartholomew, J. B., Stults-Kolehmainen, M. A., Elrod, C. C., & Todd, J. S. (2008). Strength gains after resistance training: the effect of stressful, negative life events. *The Journal of Strength & Conditioning Research, 22*(4), 1215-1221.

Bergstrom, J. (1962). Muscle electrolytes in man determined by neutron activation analysis on needle biopsy specimens. *Scandinavian Journal of Clinical and Laboratory Investigation, 14*(68).

Bernardi, L., Passino, C., Robergs, R., & Appenzeller, O. (1997). Acute and persistent effects of a 46-kilometre wilderness trail run at altitude: cardiovascular autonomic modulation and baroreflexes. *Cardiovascular Research, 34*(2), 273-280.

Bompa, T. O., & Haff, G. (2009). *Periodization: Theory and methodology of training*: Human Kinetics.

Borresen, J., & Lambert, M. I. (2008). Autonomic control of heart rate during and after exercise. *Sports Medicine, 38*(8), 633-646.

Bouchard, C., An, P., Rice, T., Skinner, J. S., Wilmore, J. H., Gagnon, J., . . . Rao, D. (1999). Familial aggregation of VO2 max response to exercise training: results from the HERITAGE Family Study. *Journal of Applied Physiology, 87*(3), 1003-1008.

Bouchard, C., & Rankinen, T. (2001). Individual differences in response to regular physical activity. *Medicine and Science in Sports and Exercise, 33*(6 Suppl), 446-453.

Bouchard, C., Sarzynski, M. A., Rice, T. K., Kraus, W. E., Church, T. S., Sung, Y. J., . . . Rankinen, T. (2011). Genomic predictors of the maximal O2 uptake response to standardized exercise training programs. *Journal of Applied Physiology, 110*(5), 1160-1170.

Bray, M., Hagberg, J., Perusse, L., Rankinen, T., Roth, S., Wolfarth, B., & Bouchard, C. (2009). The human gene map for performance and health-related fitness phenotypes: the 2006-2007 update. *Medicine and Science in Sports and Exercise, 41*(1), 35.

Brooks, B. P., Merry, D. E., Paulson, H. L., Lieberman, A. P., Kolson, D. L., & Fischbeck, K. H. (1998). A cell culture model for androgen effects in motor neurons. *Journal of Neurochemistry, 70*(3), 1054-1060.

Brooks, G. A., Fahey, T. D., & White, T. P. (1996). *Exercise physiology: Human bioenergetics and its applications*: Mayfield publishing company.

Buchheit, M. (2014). Monitoring training status with HR measures: do all roads lead to Rome. *Frontiers in Physiology, 27*(5), 73.

Buchheit, M., Al Haddad, H., Laursen, P., & Ahmaidi, S. (2009). Effect of body posture on postexercise parasympathetic reactivation in men. *Experimental Physiology, 94*(7), 795-804.

Buchheit, M., Al Haddad, H., Mendez-Villanueva, A., Quod, M. J., & Bourdon, P. C. (2011). Effect of maturation on hemodynamic and autonomic control recovery following maximal running exercise in highly trained young soccer players. *Frontiers in Physiology, 2*, 69.

Buchheit, M., Chivot, A., Parouty, J., Mercier, D., Al Haddad, H., Laursen, P., & Ahmaidi, S. (2010). Monitoring endurance running performance using cardiac parasympathetic function. *European Journal of Applied Physiology, 108*(6), 1153-1167.

Buchheit, M., & Gindre, C. (2006). Cardiac parasympathetic regulation: respective associations with cardiorespiratory fitness and training load. *American Journal of Physiology-Heart and Circulatory Physiology, 291*(1), H451-H458.

Buchheit, M., Hader, K., & Mendez-Villanueva, A. (2012). Tolerance to high-intensity intermittent running exercise: do oxygen uptake kinetics really matter? *Frontiers in Physiology, 3*(406), 1-13.

Buchheit, M., Laursen, P., Al Haddad, H., & Ahmaidi, S. (2009). Exercise-induced plasma volume expansion and post-exercise parasympathetic reactivation. *European Journal of Applied Physiology, 105*(3), 471-481.

Buchheit, M., Mendez-Villanueva, A., Quod, M. J., Poulos, N., & Bourdon, P. (2010). Determinants of the variability of heart rate measures during a competitive period in young soccer players. *European Journal of Applied Physiology, 109*(5), 869-878.

Buchheit, M., Millet, G., Parisy, A., Pourchez, S., Laursen, P., & Ahmaidi, S. (2008). Supramaximal training and postexercise parasympathetic reactivation in adolescents. *Medicine and Science in Sports and Exercise, 40*(2), 362.

Buchheit, M., Papelier, Y., Laursen, P. B., & Ahmaidi, S. (2007). Noninvasive assessment of cardiac parasympathetic function: postexercise heart rate recovery or heart rate variability? *American Journal of Physiology-Heart and Circulatory Physiology, 293*(1), H8-H10.

Buchheit, M., Racinais, S., Bilsborough, J., Bourdon, P., Voss, S., Hocking, J., . . . Coutts, A. (2013). Monitoring fitness, fatigue and running performance during a pre-season training camp in elite football players. *Journal of Science and Medicine in Sport, 16*(6), 550-555.

Buchheit, M., Voss, S., Nybo, L., Mohr, M., & Racinais, S. (2011a). Physiological and performance adaptations to an in-season soccer camp in the heat: Associations with heart rate and heart rate variability. *Scandinavian Journal of Medicine and Science in Sports, 21*(6), 477-485.

Buchheit, M., Voss, S., Nybo, L., Mohr, M., & Racinais, S. (2011b). Physiological and performance adaptations to an in-season soccer camp in the heat: Associations with heart rate and heart rate variability. *Scandinavian Journal of Medicine and Science in Sports, 21*(6), e477-e485.

Bundle, M. W., Hoyt, R. W., & Weyand, P. G. (2003). High-speed running performance: a new approach to assessment and prediction. *Journal of Applied Physiology, 95*(5), 1955-1962.

Burke, L. M., Kiens, B., & Ivy, J. L. (2004). Carbohydrates and fat for training and recovery. *Journal of Sports Sciences, 22*(1), 15-30.

Cairns, S. P. (2006). Lactic acid and exercise performance. *Sports Medicine, 36*(4), 279-291.

Carter, R., Watenpaugh, D. E., & Smith, M. L. (2001). Selected Contribution: Gender differences in cardiovascular regulation during recovery from exercise. *Journal of Applied Physiology, 91*(4), 1902-1907.

Carter, R., Wilson, T. E., Watenpaugh, D. E., Smith, M. L., & Crandall, C. G. (2002). Effects of mode of exercise recovery on thermoregulatory and cardiovascular responses. *Journal of Applied Physiology, 93*(6), 1918-1924.

Casey, A., Constantin-Teodosiu, D., Howell, S., Hultman, E., & Greenhaff, P. (1996). Metabolic response of type I and II muscle fibers during repeated bouts of maximal exercise in humans. *American Journal of Physiology-Endocrinology And Metabolism, 271*(1), E38-E43.

Cerretelli, P., & Di Prampero, P. (1971). Kinetics of respiratory gas exchange and cardiac output at the onset of exercise. *Scandinavian Journal of Respiratory Diseases. Supplementum, 77*, 35a.

Charkoudian, N., & Joyner, M. J. (2004). Physiologic considerations for exercise performance in women. *Clinics in chest medicine, 25*(2), 247-255.

Chen, H. (1991). Effects of 30-h sleep loss on cardiorespiratory functions at rest and in exercise. *Medicine and Science in Sports and Exercise, 23*(2), 193-198.

Chen, M. J., Fan, X., & Moe, S. T. (2002). Criterion-related validity of the Borg ratings of perceived exertion scale in healthy individuals: a meta-analysis. *Journal of Sports Sciences, 20*(11), 873-899.

Cheung, K., Hume, P. A., & Maxwell, L. (2003). Delayed onset muscle soreness. *Sports Medicine, 33*(2), 145-164.

Christen, J., Foster, C., Porcari, J., & Mikat, R. (2016). Temporal Robustness of the Session RPE. *International Journal of Sports Physiology and Performance*.

Christensen, N. J., & Galbo, H. (1983). Sympathetic nervous activity during exercise. *Annual Review of Physiology, 45*(1), 139-153.

Clarkson, P. M., & Hubal, M. J. (2002). Exercise-induced muscle damage in humans. *American Journal of Physical Medicine and Rehabilitation, 81*(11), S52-S69.

Convertino, V. A. (2003). Baroreflex-mediated heart rate and vascular resistance responses 24 h after maximal exercise. *Medicine and Science in Sports and Exercise, 35*(6), 970-977.

Costill, D. L., Daniels, J., Evans, W., Fink, W., Krahenbuhl, G., & Saltin, B. (1976). Skeletal muscle enzymes and fiber composition in male and female track athletes. *Journal of Applied Physiology, 40*(2), 149-154.

Coutts, A., Wallace, L., & Slattery, K. (2007). Monitoring changes in performance, physiology, biochemistry, and psychology during overreaching and recovery in triathletes. *International Journal of Sports Medicine, 28*(2), 125-134.

Craig, N. P., & Norton, K. I. (2001). Characteristics of track cycling. *Sports Medicine, 31*(7), 457-468.

Crewther, B., Cronin, J., Keogh, J., & Cook, C. (2008). The salivary testosterone and cortisol response to three loading schemes. *The Journal of Strength & Conditioning Research, 22*(1), 250-255.

Davy, K. P., Miniclier, N. L., Taylor, J. A., Stevenson, E. T., & Seals, D. R. (1996). Elevated heart rate variability in physically active postmenopausal women: a cardioprotective effect? *American Journal of Physiology-Heart and Circulatory Physiology, 271*(2), H455-H460.

de Campos Mello, F., de Moraes Bertuzzi, R. C., Grangeiro, P. M., & Franchini, E. (2009). Energy systems contributions in 2,000 m race simulation: a comparison among rowing ergometers and water. *European Journal of Applied Physiology, 107*(5), 615-619.

Del Rosso, S., Nakamura, F. Y., & Boullosa, D. A. (2016). Heart rate recovery after aerobic and anaerobic tests: is there an influence of anaerobic speed reserve? *Journal of Sports Sciences*, 1-8.

Delaney, J., & Brodie, D. (2000). Effects of short-term psychological stress on the time and frequency domains of heart-rate variability. *Perceptual and Motor Skills, 91*(2), 515-524.

Delp, M. D., & O'Leary, D. S. (2004). Integrative control of the skeletal muscle microcirculation in the maintenance of arterial pressure during exercise. *Journal of Applied Physiology, 97*(3), 1112-1118.

Dixon, E. M., Kamath, M. V., McCartney, N., & Fallen, E. L. (1992). Neural regulation of heart rate variability in endurance athletes and sedentary controls. *Cardiovascular Research, 26*(7), 713-719.

Doherty, M., Smith, P. M., & Schroder, K. (2000). Reproducibility of the maximum accumulated oxygen deficit and run time to exhaustion during short-distance running. *Journal of Sports Sciences, 18*(5), 331-338.

Douglas, P. S., O'Toole, M. L., Hiller, W., Hackney, K., & Reichek, N. (1987). Cardiac fatigue after prolonged exercise. *Circulation, 76*(6), 1206-1213.

Doyle, J. A., Sherman, W. M., & Strauss, R. L. (1993). Effects of eccentric and concentric exercise on muscle glycogen replenishment. *Journal of Applied Physiology, 74*(4), 1848-1855.

Edis, A. J., & Shepherd, J. T. (1970). Autonomic control of the peripheral vascular system. *Archives of Internal Medicine, 125*(4), 716-724.

Ehsani, A. A., Hagberg, J. M., & Hickson, R. C. (1978). Rapid changes in left ventricular dimensions and mass in response to physical conditioning and deconditioning. *The American journal of cardiology, 42*(1), 52-56.

Eisenstein, A. (1973). Effects of adrenal cortical hormones on carbohydrate, protein, and fat metabolism. *The American Journal of Clinical Nutrition, 26*(1), 113-120.

Fiskerstrand, Å., & Seiler, K. (2004). Training and performance characteristics among Norwegian international rowers 1970–2001. *Scandinavian Journal of Medicine and Science in Sports, 14*(5), 303-310.

Foster, C., Florhaug, J. A., Franklin, J., Gottschall, L., Hrovatin, L. A., Parker, S., . . . Dodge, C. (2001). A new approach to monitoring exercise training. *The Journal of Strength & Conditioning Research, 15*(1), 109-115.

Fry, A., Kraemer, W. J., van Borselen, F., Lynch, J. M., Marsit, J. L., Roy, E. P., . . . Knuttgen, H. G. (1994). Performance decrements with high-intensity. *Training, 26*(9), 1165-1173.

Fry, A., Schilling, B. K., Weiss, L. W., & Chiu, L. Z. (2006). β2-Adrenergic receptor downregulation and performance decrements during high-intensity resistance exercise overtraining. *Journal of Applied Physiology, 101*(6), 1664-1672.

Fry, R., Morton, A. R., Garcia-Webb, P., & Keast, D. (1991). Monitoring exercise stress by changes in metabolic and hormonal responses over a 24-h period. *European Journal of Applied Physiology and Occupational Physiology, 63*(3-4), 228-234.

Fukuda, S., & Morimoto, K. (2001). Lifestyle, stress and cortisol response: Review I. *Environmental Health and Preventive Medicine, 6*(1), 9-14.

Furlan, R., Piazza, S., Dell'Orto, S., Gentile, E., Cerutti, S., Pagani, M., & Malliani, A. (1993). Early and late effects of exercise and athletic training on neural mechanisms controlling heart rate. *Cardiovascular Research, 27*(3), 482-488.

Garet, M., Tournaire, N., Roche, F., Laurent, R., Lacour, J. R., Barthélémy, J. C., & Pichot, V. (2004). Individual interdependence between nocturnal ANS activity and performance in swimmers. *Medicine and Science in Sports and Exercise, 36*(12), 2112-2118.

Garrandes, F., Colson, S. S., Pensini, M., Seynnes, O., & Legros, P. (2007). Neuromuscular fatigue profile in endurance-trained and power-trained athletes. *Medicine and Science in Sports and Exercise, 39*(1), 149-158.

Gaskill, S. E., Rice, T., Bouchard, C., Gagnon, J., Rao, D., Skinner, J. S., . . . Leon, A. S. (2001). Familial resemblance in ventilatory threshold: the HERITAGE Family Study. *Medicine and Science in Sports and Exercise, 33*(11), 1832-1840.

Gastin, P. (1994). Quantification of anaerobic capacity. *Scandinavian journal of medicine & science in sports, 4*(2), 91-112.

Giustina, A., & Veldhuis, J. D. (1998). Pathophysiology of the neuroregulation of growth hormone secretion in experimental animals and the human 1. *Endocrine Reviews, 19*(6), 717-797.

Gratze, G., Rudnicki, R., Urban, W., Mayer, H., Schlögl, A., & Skrabal, F. (2005). Hemodynamic and autonomic changes induced by Ironman: prediction of competition time by blood pressure variability. *Journal of Applied Physiology, 99*(5), 1728-1735.

Greenhaff, P. (1989). Cardiovascular fitness and thermoregulation during prolonged exercise in man. *British Journal of Sports Medicine, 23*(2), 109-114.

Greenhaff, P., Nevill, M., Soderlund, K., Bodin, K., Boobis, L., Williams, C., & Hultman, E. (1994). The metabolic responses of human type I and II muscle fibres during maximal treadmill sprinting. *The Journal of physiology, 478*(Pt 1), 149.

Greiwe, J. S., Hickner, R. C., Hansen, P. A., Racette, S. B., Chen, M. M., & Holloszy, J. O. (1999). Effects of endurance exercise training on muscle glycogen accumulation in humans. *Journal of Applied Physiology, 87*(1), 222-226.

Grove, J., Main, L., Partridge, K., Bishop, D., Russell, S., Shepherdson, A., & Ferguson, L. (2014). Training distress and performance readiness: Laboratory and field validation of a brief self-report measure. *Scandinavian Journal of Medicine and Science in Sports, 24*(6), e483-490.

Haff, G. G., Lehmkuhl, M. J., McCoy, L. B., & Stone, M. H. (2003). Carbohydrate supplementation and resistance training. *The Journal of Strength & Conditioning Research, 17*(1), 187-196.

Hagerman, F. (1994). Physiology and nutrition for rowing. *Perspectives in Exercise Science and Sports Medicine, 7*, 221-302.

Hagerman, F., Connors, M., Gault, J., Hagerman, G., & Polinski, W. (1978). Energy expenditure during simulated rowing. *Journal of Applied Physiology, 45*(1), 87-93.

Halson, S. L., Bridge, M. W., Meeusen, R., Busschaert, B., Gleeson, M., Jones, D. A., & Jeukendrup, A. E. (2002). Time course of performance changes and fatigue markers during intensified training in trained cyclists. *Journal of Applied Physiology, 93*(3), 947-956.

Halson, S. L., Quod, M. J., Martin, D. T., Gardner, A. S., Ebert, T. R., & Laursen, P. B. (2008). Physiological responses to cold water immersion following cycling in the heat. *International Journal of Sports Physiology and Performance, 3*(3), 331-346.

Hamada, T., Sale, D., MacDougall, J., & Tarnopolsky, M. (2003). Interaction of fibre type, potentiation and fatigue in human knee extensor muscles. *Acta Physiologica Scandinavica, 178*(2), 165-173.

Hartwell, M. L., Volberding, J. L., & Brennan, D. K. (2015). Cardiovascular Drift while Rowing on an Ergometer. *Journal of Exercise Physiology Online, 18*(2).

Hautala, A. J., Kiviniemi, A. M., Mäkikallio, T. H., Kinnunen, H., Nissilä, S., Huikuri, H. V., & Tulppo, M. P. (2006). Individual differences in the responses to endurance and resistance training. *European Journal of Applied Physiology, 96*(5), 535-542.

Hautala, A. J., Mäkikallio, T. H., Kiviniemi, A. M., Laukkanen, R. T., Nissilä, S., Huikuri, H. V., & Tulppo, M. P. (2003). Cardiovascular autonomic function correlates with the response to aerobic training in healthy sedentary subjects. *American Journal of Physiology-Heart and Circulatory Physiology, 285*(4), H1747-H1752.

Hautala, A. J., Tulppo, M. P., Mäkikallio, T. H., Laukkanen, R., Nissilä, S., & Huikuri, H. V. (2001). Changes in cardiac autonomic regulation after prolonged maximal exercise. *Clinical Physiology, 21*(2), 238-245.

Hawley, J. A., Burke, L. M., Phillips, S. M., & Spriet, L. L. (2011). Nutritional modulation of training-induced skeletal muscle adaptations. *Journal of Applied Physiology, 110*(3), 834-845.

Hedelin, R., Kenttä, G., Wiklund, U., Bjerle, P., & Henriksson-Larsén, K. (2000). Short-term overtraining: effects on performance, circulatory responses, and heart rate variability. *Medicine and science in sports and exercise, 32*(8), 1480-1484.

Heffernan, K. S., Fahs, C. A., Shinsako, K. K., Jae, S. Y., & Fernhall, B. (2007). Heart rate recovery and heart rate complexity following resistance exercise training and detraining in young men. *American Journal of Physiology-Heart and Circulatory Physiology, 293*(5), H3180-H3186.

Hetlelid, K. J., Plews, D. J., Herold, E., Laursen, P. B., & Seiler, S. (2015). Rethinking the role of fat oxidation: substrate utilisation during high-intensity interval training in well-trained and recreationally trained runners. *BMJ Open Sport & Exercise Medicine, 1*(1), e000047.

Hickner, R. C., Fisher, J., Hansen, P., Racette, S., Mier, C., Turner, M. J., & Holloszy, J. (1997). Muscle glycogen accumulation after endurance exercise in trained and untrained individuals. *Journal of Applied Physiology, 83*(3), 897-903.

Hill, D. W. (1999). Energy system contributions in middle-distance running events. *Journal of Sports Sciences, 17*(6), 477-483.

Hill, E., Zack, E., Battaglini, C., Viru, M., Viru, A., & Hackney, A. (2008). Exercise and circulating cortisol levels: the intensity threshold effect. *Journal of Endocrinological Investigation, 31*(7), 587-591.

Hirvonen, J., Rehunen, S., Rusko, H., & Härkönen, M. (1987). Breakdown of high-energy phosphate compounds and lactate accumulation during short supramaximal exercise. *European Journal of Applied Physiology and Occupational Physiology, 56*(3), 253-259.

Holloszy, J. O., & Coyle, E. F. (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *Journal of Applied Physiology, 56*(4), 831-838.

Hooper, S., Mackinnon, L. T., Gordon, R. D., & Bachmann, A. W. (1993). Hormonal responses of elite swimmers to overtraining. *Medicine and Science in Sports and Exercise, 25*(6), 741-747.

Hopkins, W. G. (2006). Spreadsheets for analysis of controlled trials, with adjustment for a subject characteristic. (10), 46-50.

Hopkins, W. G. (2007). A spreadsheet for deriving a confidence interval, mechanistic inference and clinical inference from a p value. *Sportscience, 11*, 16-20.

Hopkins, W. G., Marshall, S., Batterham, A., & Hanin, J. (2009). Progressive statistics for studies in sports medicine and exercise science. *Medicine and Science in Sports and Exercise, 41*(1), 3.

Huang, C.-J., Webb, H. E., Zourdos, M. C., & Acevedo, E. O. (2007). Cardiovascular reactivity, stress, and physical activity. *The Role of Physical Fitness on Cardiovascular Responses to Stress, 4*, 6.

Hynynen, E., Vesterinen, V., Rusko, H., & Nummela, A. (2010). Effects of moderate and heavy endurance exercise on nocturnal HRV. *International Journal of Sports Medicine, 31*(6), 428-432.

Ingjer, F. (1979). Effects of endurance training on muscle fibre ATP-ase activity, capillary supply and mitochondrial content in man. *The Journal of Physiology, 294*(1), 419-432.

Ivy, J. L. (1998). Glycogen resynthesis after exercise: effect of carbohydrate intake. *International Journal of Sports Medicine, 19*, S142-145.

Ivy, J. L., Goforth, H. W., Damon, B. M., McCauley, T. R., Parsons, E. C., & Price, T. B. (2002). Early postexercise muscle glycogen recovery is enhanced with a carbohydrate-protein supplement. *Journal of Applied Physiology, 93*(4), 1337-1344.

Ivy, J. L., & Holloszy, J. (1981). Persistent increase in glucose uptake by rat skeletal muscle following exercise. *American Journal of Physiology-Cell Physiology, 241*(5), C200-C203.

Ivy, J. L., Lee, M., Brozinick, J., & Reed, M. (1988). Muscle glycogen storage after different amounts of carbohydrate ingestion. *Journal of Applied Physiology, 65*(5), 2018-2023.

Jacks, D. E., Sowash, J., Anning, J., McGloughlin, T., & Andres, F. (2002). Effect of exercise at three exercise intensities on salivary cortisol. *The Journal of Strength & Conditioning Research, 16*(2), 286-289.

James, D. V., Barnes, A., Lopes, P., & Wood, D. (2002). Heart rate variability: response following a single bout of interval training. *International Journal of Sports Medicine, 23*(4), 247-251.

Jentjens, R., & Jeukendrup, A. E. (2003). Determinants of post-exercise glycogen synthesis during short-term recovery. *Sports Medicine, 33*(2), 117-144.

Jeukendrup, A., Hesselink, M., Snyder, A., Kuipers, H., & Keizer, H. (1992). Physiological changes in male competitive cyclists after two weeks of intensified training. *International Journal of Sports Medicine, 13*(7), 534-541.

Jokela, M., & Hanin, Y. L. (1999). Does the individual zones of optimal functioning model discriminate between successful and less successful athletes? A meta-analysis. *Journal of Sports Sciences, 17*(11), 873-887.

Jurkowski, J., Jones, N. L., Toews, C. J., & Sutton, J. R. (1981). Effects of menstrual cycle on blood lactate, O2 delivery, and performance during exercise. *Journal of Applied Physiology, 51*(6), 1493-1499.

Kaikkonen, P., Hynynen, E., Mann, T., Rusko, H., & Nummela, A. (2010). Can HRV be used to evaluate training load in constant load exercises? *European Journal of Applied Physiology, 108*(3), 435-442.

Kaikkonen, P., Nummela, A., & Rusko, H. (2007). Heart rate variability dynamics during early recovery after different endurance exercises. *European Journal of Applied Physiology, 102*(1), 79-86.

Kaikkonen, P., Rusko, H., & Martinmäki, K. (2008). Post-exercise heart rate variability of endurance athletes after different high-intensity exercise interventions. *Scandinavian Journal of Medicine and Science in Sports, 18*(4), 511-519.

Kannankeril, P. J., Le, F. K., Kadish, A. H., & Goldberger, J. J. (2004). Parasympathetic effects on heart rate recovery after exercise. *Journal of Investigative Medicine, 52*(6), 394-401.

Karatzaferi, C., De Haan, A., Ferguson, R., Van Mechelen, W., & Sargeant, A. (2001). Phosphocreatine and ATP content in human single muscle fibres before and after maximum dynamic exercise. *Pflügers Archiv, 442*(3), 467-474.

Karlsson, J., & Saltin, B. (1971). Oxygen deficit and muscle metabolites in intermittent exercise. *Acta Physiologica Scandinavica, 82*(1), 115-122.

Kellmann, M. (2010). Preventing overtraining in athletes in high-intensity sports and stress/recovery monitoring. *Scandinavian Journal of Medicine and Science in Sports, 20*(s2), 95-102.

Kellmann, M., Altenburg, D., Lormes, W., & Steinacker, J. (2001). Assessing stress and recovery during preparation for the world championships in rowing. *Sport Psychologist, 15*(2), 151-167.

Kenny, G. P., & Jay, O. (2007). Evidence of a greater onset threshold for sweating in females following intense exercise. *European journal of applied physiology, 101*(4), 487-493.

Kindermann, W., Schnabel, A., Schmitt, W., Biro, G., Cassens, J., & Weber, F. (1982). Catecholamines, growth hormone, cortisol, insulin, and sex hormones in anaerobic and aerobic exercise. *European Journal of Applied Physiology and Occupational Physiology, 49*(3), 389-399.

Kingsley, J., Hochgesang, S., Brewer, A., Buxton, E., Martinson, M., & Heidner, G. (2014). Autonomic modulation in resistance-trained individuals after acute resistance exercise. *International Journal of Sports Medicine, 35*(10), 851-856.

Kirwan, J. P., del Aguila, L. F., Hernandez, J. M., Williamson, D. L., O'Gorman, D. J., Lewis, R., & Krishnan, R. K. (2000). Regular exercise enhances insulin activation of IRS-1-associated PI3-kinase in human skeletal muscle. *Journal of Applied Physiology, 88*(2), 797-803.

Kiviniemi, A. M., Hautala, A. J., Kinnunen, H., Nissila, J., Virtanen, P., Karjalainen, J., & Tulppo, M. P. (2010). Daily exercise prescription on the basis of HR variability among men and women. *Medicine and Science in Sports and Exercise, 42*(7), 1355-1363.

Kiviniemi, A. M., Hautala, A. J., Kinnunen, H., & Tulppo, M. P. (2007). Endurance training guided individually by daily heart rate variability measurements. *European Journal of Applied Physiology, 101*(6), 743-751.

Kjellberg, S. R., Rudhe, U., & Sjostrand, T. (1949). Increase of the amount of hemoglobin and blood volume in connection with physical training. *Acta Physiologica Scandinavica, 19*(2-3), 146-151.

Kohrt, W. M., Malley, M. T., Coggan, A. R., Spina, R. J., Ogawa, T., Ehsani, A., . . . Holloszy, J. (1991). Effects of gender, age, and fitness level on response of VO2max to training in 60-71 yr olds. *Journal of Applied Physiology, 71*(5), 2004-2011.

Krustrup, P., Mohr, M., Steensberg, A., Bencke, J., Kjær, M., & Bangsbo, J. (2006). Muscle and blood metabolites during a soccer game: implications for sprint performance. *Medicine and Science in Sports and Exercise, 38*(6), 1165-1174.

Krustrup, P., Ørtenblad, N., Nielsen, J., Nybo, L., Gunnarsson, T. P., Iaia, F. M., . . . Bangsbo, J. (2011). Maximal voluntary contraction force, SR function and glycogen resynthesis during the first 72 h after a high-level competitive soccer game. *European Journal of Applied Physiology, 111*(12), 2987-2995.

Lamberts, R. (2013). Predicting cycling performance in trained to elite male and female cyclists. *Int. J. Sports Physiol. Perform*.

Lamberts, R., Swart, J., Capostagno, B., Noakes, T., & Lambert, M. (2010). Heart rate recovery as a guide to monitor fatigue and predict changes in performance parameters. *Scandinavian Journal of Medicine and Science in Sports, 20*(3), 449-457.

Lippi, G., De Vita, F., Salvagno, G. L., Gelati, M., Montagnana, M., & Guidi, G. C. (2009). Measurement of morning saliva cortisol in athletes. *Clinical Biochemistry, 42*(9), 904-906.

Loebel, C. C., & Kraemer, W. J. (1998). Testosterone and Resistance Exercise in Men. *The Journal of Strength & Conditioning Research, 12*(1), 57-63.

Lortie, G., Simoneau, J., Hamel, P., Boulay, M., Landry, F., & Bouchard, C. (1984). Responses of maximal aerobic power and capacity to aerobic training. *International Journal of Sports Medicine, 5*(5), 232-236.

Mac Nair, D., Lorr, M., & Droppleman, L. (1971). Profile of mood States manual. *San Diego: Educational and industrial testing service*.

MacRae, H., Dennis, S. C., Bosch, A. N., & Noakes, T. D. (1992). Effects of training on lactate production and removal during progressive exercise in humans. *Journal of Applied Physiology, 72*(5), 1649-1656.

Manetta, J., Brun, J.-F., Prefaut, C., & Mercier, J. (2005). Substrate oxidation during exercise at moderate and hard intensity in middle-aged and young athletes vs sedentary men. *Metabolism: Clinical and Experimental, 54*(11), 1411-1419.

Mann, T., Lamberts, R. P., & Lambert, M. I. (2013). Methods of prescribing relative exercise intensity: physiological and practical considerations. *Sports Medicine, 43*(7), 613-625.

Mann, T., Webster, C., Lamberts, R. P., & Lambert, M. I. (2014). Effect of exercise intensity on post-exercise oxygen consumption and heart rate recovery. *European Journal of Applied Physiology, 114*(9), 1809-1820.

Marshall, G. J., & Turner, A. N. (2016). The importance of sleep for athletic performance. *Strength & Conditioning Journal, 38*(1), 61-67.

Marucha, P. T., Kiecolt-Glaser, J. K., & Favagehi, M. (1998). Mucosal wound healing is impaired by examination stress. *Psychosomatic Medicine, 60*(3), 362-365.

McCartney, N., Spriet, L. L., Heigenhauser, G., Kowalchuk, J. M., Sutton, J. R., & Jones, N. L. (1986). Muscle power and metabolism in maximal intermittent exercise. *Journal of Applied Physiology, 60*(4), 1164-1169.

McDonald, K. G., Grote, S., & Shoepe, T. C. (2014). Effect of Training Mode on Post-Exercise Heart Rate Recovery of Trained Cyclists. *Journal of Human Kinetics, 41*(1), 43-49.

McPhee, J. S., Williams, A. G., Degens, H., & Jones, D. A. (2010). Inter-individual variability in adaptation of the leg muscles following a standardised endurance training programme in young women. *European Journal of Applied Physiology, 109*(6), 1111-1118.

McPhee, J. S., Williams, A. G., Perez-Schindler, J., Degens, H., Baar, K., & Jones, D. A. (2011). Variability in the magnitude of response of metabolic enzymes reveals patterns of co-ordinated expression following endurance training in women. *Experimental Physiology, 96*(7), 699-707.

Medbo, J. I., Mohn, A.-C., Tabata, I., Bahr, R., Vaage, O., & Sejersted, O. M. (1988). Anaerobic capacity determined by maximal accumulated O2 deficit. *Journal of Applied Physiology, 64*(1), 50-60.

Meeusen, R., Duclos, M., Foster, C., Fry, A., Gleeson, M., Nieman, D., . . . Urhausen, A. (2013). Prevention, diagnosis, and treatment of the overtraining syndrome: joint consensus statement of the European College of Sport Science and the American College of Sports Medicine. *Medicine and Science in Sports and Exercise, 45*(1), 186-205.

Mendonca, G. V., Heffernan, K. S., Rossow, L., Guerra, M., Pereira, F. D., & Fernhall, B. (2010). Sex differences in linear and nonlinear heart rate variability during early recovery from supramaximal exercise. *Applied Physiology, Nutrition, and Metabolism, 35*(S1), 439-446.

Meyer, T., Gabriel, H., & Kindermann, W. (1999). Is determination of exercise intensities as percentages of VO2max or HRmax adequate? *Medicine and Science in Sports and Exercise, 31*(9), 1342-1345.

Mickelson, T. C., & Hagerman, F. (1981). Anaerobic threshold measurements of elite oarsmen. *Medicine and science in sports and exercise, 14*(6), 440-444.

Mikulić, P., Smoljanović, T., Bojanić, I., Hannafin, J. A., & Matković, B. R. (2009). Relationship between 2000-m rowing ergometer performance times and World Rowing Championships rankings in elite-standard rowers. *Journal of sports sciences, 27*(9), 907-913.

Mohr, M., Mujika, I., Santisteban, J., Randers, M. B., Bischoff, R., Solano, R., . . . Krustrup, P. (2010). Examination of fatigue development in elite soccer in a hot environment: a multi-experimental approach. *Scandinavian Journal of Medicine and Science in Sports, 20*(s3), 125-132.

Morley, J. E., Perry, I., Mitchell, H., Patrick, P., Dollbaum, C. M., & Kells, J. M. (2006). Validation of salivary testosterone as a screening test for male hypogonadism. *The Aging Male, 9*(3), 165-169.

Mourot, L., Bouhaddi, M., Tordi, N., Rouillon, J.-D., & Regnard, J. (2004). Short-and long-term effects of a single bout of exercise on heart rate variability: comparison between constant and interval training exercises. *European Journal of Applied Physiology, 92*(4-5), 508-517.

Munck, A. (1971). Glucocorticoid inhibition of glucose uptake by peripheral tissues: old and new evidence, molecular mechanisms, and physiological significance. *Perspectives in Biology and Medicine, 14*(2), 265-289.

Nagaya, N., & Herrera, A. A. (1995). Effects of testosterone on synaptic efficacy at neuromuscular junctions in a sexually dimorphic muscle of male frogs. *Journal of Physiology, 483*(Pt 1), 141.

Nevill, A. M., Beech, C., Holder, R. L., & Wyon, M. (2010). Scaling concept II rowing ergometer performance for differences in body mass to better reflect rowing in water. *Scandinavian journal of medicine & science in sports, 20*(1), 122-127.

Nieman, D., Nehlsen-Cannarella, S., Fagoaga, O., Henson, D., Shannon, M., Davis, J., . . . Hjertman, J. (1999). Immune response to two hours of rowing in elite female rowers. *International Journal of Sports Medicine, 20*(7), 476-481.

Niewiadomski, W., Gąsiorowska, A., Krauss, B., Mroz, A., & Cybulski, G. (2007). Suppression of heart rate variability after supramaximal exertion. *Clinical Physiology and Functional Imaging, 27*(5), 309-319.

Noordhof, D. A., De Koning, J. J., & Foster, C. (2010). The maximal accumulated oxygen deficit method. *Sports Medicine, 40*(4), 285-302.

Nybo, L. (2008). Hyperthermia and fatigue. *Journal of Applied Physiology, 104*(3), 871-878.

O'Connor, P. J., & Corrigan, D. L. (1987). Influence of short-term cycling on salivary cortisol levels. *Medicine and Science in Sports and Exercise, 19*(3), 224-228.

Orizio, C., Perini, R., Comandè, A., Castellano, M., Beschi, M., & Veicsteinas, A. (1988). Plasma catecholamines and heart rate at the beginning of muscular exercise in man. *European Journal of Applied Physiology and Occupational Physiology, 57*(5), 644-651.

Otsuki, T., Maeda, S., Iemitsu, M., Saito, Y., Tanimura, Y., Sugawara, J., . . . Miyauchi, T. (2007). Postexercise heart rate recovery accelerates in strength-trained athletes. *Medicine and Science in Sports and Exercise, 39*(2), 365.

Paavolainen, L., Häkkinen, K., Nummela, A., & Ruskol, H. (1994). Neuromuscular characteristics and fatigue in endurance and sprint athletes during a new anaerobic power test. *European Journal of Applied Physiology and Occupational Physiology, 69*(2), 119-126.

Papacosta, E., & Nassis, G. P. (2011). Saliva as a tool for monitoring steroid, peptide and immune markers in sport and exercise science. *Journal of Science and Medicine in Sport, 14*(5), 424-434.

Parekh, A., & Lee, C. M. (2005). Heart rate variability after isocaloric exercise bouts of different intensities. *Medicine and Science in Sports and Exercise, 37*(4), 599-605.

Penttilä, J., Helminen, A., Jartti, T., Kuusela, T., Huikuri, H. V., Tulppo, M. P., . . . Scheinin, H. (2001). Time domain, geometrical and frequency domain analysis of cardiac vagal outflow: effects of various respiratory patterns. *Clinical Physiology, 21*(3), 365-376.

Perini, R., Orizio, C., Comandè, A., Castellano, M., Beschi, M., & Veicsteinas, A. (1989). Plasma norepinephrine and heart rate dynamics during recovery from submaximal exercise in man. *European Journal of Applied Physiology and Occupational Physiology, 58*(8), 879-883.

Perna, F. M., & McDowell, S. L. (1995). Role of psychological stress in cortisol recovery from exhaustive exercise among elite athletes. *International Journal of Behavioral Medicine, 2*(1), 13-26.

Petersen, K., Hansen, C. B., Aagaard, P., & Madsen, K. (2007). Muscle mechanical characteristics in fatigue and recovery from a marathon race in highly trained runners. *European Journal of Applied Physiology, 101*(3), 385-396.

Phillips, S. M. (2004). Protein requirements and supplementation in strength sports. *Nutrition, 20*(7), 689-695.

Plews, D. J., Laursen, P. B., Kilding, A. E., & Buchheit, M. (2014). Heart rate variability and training intensity distribution in elite rowers. *International Journal of Sports Physiology and Performance*.

Plews, D. J., Laursen, P. B., Stanley, J., Kilding, A. E., & Buchheit, M. (2013). Training adaptation and heart rate variability in elite endurance athletes: opening the door to effective monitoring. *Sports Medicine, 43*(9), 773-781.

Pringle, J. S., Doust, J. H., Carter, H., Tolfrey, K., Campbell, I. T., & Jones, A. M. (2003). Oxygen uptake kinetics during moderate, heavy and severe intensity'submaximal'exercise in humans: the influence of muscle fibre type and capillarisation. *European Journal of Applied Physiology, 89*(3-4), 289-300.

Pripstein, L., Rhodes, E., McKenzie, D., & Coutts, K. (1999). Aerobic and anaerobic energy during a 2-km race simulation in female rowers. *European journal of applied physiology and occupational physiology, 79*(6), 491-494.

Rankinen, T., Sung, Y. J., Sarzynski, M. A., Rice, T. K., Rao, D., & Bouchard, C. (2012). Heritability of submaximal exercise heart rate response to exercise training is accounted for by nine SNPs. *Journal of Applied Physiology, 112*(5), 892-897.

Rehm, K. E., Elci, O. U., Hahn, K., & Marshall, G. D. (2013). The impact of self-reported psychological stress levels on changes to peripheral blood immune biomarkers in recreational marathon runners during training and recovery. *Neuroimmunomodulation, 20*(3), 164-176.

Reilly, T., Secher, N., Snell, P., & Williams, C. (1990). Physiology of sports: An overview. *Physiology of Sports*, 465-485.

Rennie, K. L., Hemingway, H., Kumari, M., Brunner, E., Malik, M., & Marmot, M. (2003). Effects of moderate and vigorous physical activity on heart rate variability in a British study of civil servants. *American Journal of Epidemiology, 158*(2), 135-143.

Riechman, S. E., Zoeller, R. F., Balasekaran, G., Goss, F. L., & Robertson, R. J. (2002). Prediction of 2000 m indoor rowing performance using a 30 s sprint and maximal oxygen uptake. *Journal of sports sciences, 20*(9), 681-687.

Rost, R., & Hollmann, W. (1983). Athlete's heart--a review of its historical assessment and new aspects. *International Journal of Sports Medicine, 4*(3), 147.

Roth, W., Hasart, E., Wolf, W., & Pansold, B. (1983). Untersuchungen zur Dynamik der Energiebereitstellung während maximaler Mittelzeitausdauerbelastung. *Med Sport, 23*(4), 107.

Rushall, B. S. (1990). A tool for measuring stress tolerance in elite athletes. *Journal of Applied Sport Psychology, 2*(1), 51-66.

Russell, A., Rossignol, P. L., & Sparrow, W. (1998). Prediction of elite schoolboy 2000-m rowing ergometer performance from metabolic, anthropometric and strength variables. *Journal of sports sciences, 16*(8), 749-754.

Ruuska, P. S., Hautala, A. J., Kiviniemi, A. M., Mäkikallio, T. H., & Tulppo, M. P. (2012). Self-rated mental stress and exercise training response in healthy subjects. *Frontiers in Physiology, 3*, 51.

Saeki, Y., Atogami, F., Takahashi, K., & Yoshizawa, T. (1997). Reflex control of autonomic function induced by posture change during the menstrual cycle. *Journal of the autonomic nervous system, 66*(1), 69-74.

Saltin, B., & Rowell, L. B. (1980). Functional adaptations to physical activity and inactivity Symposium conducted at the meeting of the Federation Proceedings

Samuels, C. (2009). Sleep, recovery, and performance: the new frontier in high-performance athletics. *Physical Medicine and Rehabilitation Clinics of North America, 20*(1), 149-159.

Sato, N., Miyake, S., Akatsu, J. i., & Kumashiro, M. (1995). Power spectral analysis of heart rate variability in healthy young women during the normal menstrual cycle. *Psychosomatic medicine, 57*(4), 331-335.

Saw, A. E., Main, L. C., & Gastin, P. B. (2015). Monitoring the athlete training response: subjective self-reported measures trump commonly used objective measures: a systematic review. *British Journal of Sports Medicine*, bjsports-2015-094758.

Scharhag-Rosenberger, F., Walitzek, S., Kindermann, W., & Meyer, T. (2012). Differences in adaptations to one year of aerobic endurance training: individual patterns of nonresponse. *Scandinavian Journal of Medicine and Science in Sports, 22*(1), 113-118.

Schumacher, Y. O., & Mueller, P. (2002). The 4000-m team pursuit cycling world record: theoretical and practical aspects. *Medicine and Science in Sports and Exercise, 34*(6), 1029-1036.

Secher, N. H., Vaage, O., & Jackson, R. C. (1982). *Rowing performance and maximal aerobic power of oarsmen*

Seiler, S. (2010). What is best practice for training intensity and duration distribution in endurance athletes. *International Journal of Sports Physiology and Performance, 5*(3), 276-291.

Seiler, S., Haugen, O., & Kuffel, E. (2007). Autonomic recovery after exercise in trained athletes: intensity and duration effects. *Medicine and Science in Sports and Exercise, 39*(8), 1366.

Seiler, S., & Tønnessen, E. (2009). Intervals, thresholds, and long slow distance: the role of intensity and duration in endurance training. *Sportscience, 13*, 32-53.

Sherman, W. M., Costill, D. L., Fink, W., Hagerman, F., Armstrong, L., & Murray, T. (1983). Effect of a 42.2-km footrace and subsequent rest or exercise on muscle glycogen and enzymes. *Journal of Applied Physiology, 55*(4), 1219-1224.

Shumate, J. B., Brooke, M. H., Carroll, J. E., & Davis, J. E. (1979). Increased serum creatine kinase after exercise A sex-linked phenomenon. *Neurology, 29*(6), 902-902.

Sikorski, E. M., Wilson, J. M., Lowery, R. P., Joy, J. M., Laurent, C. M., Wilson, S. M., . . . Gilchrist, P. (2013). Changes in perceived recovery status scale following high-volume muscle damaging resistance exercise. *The Journal of Strength & Conditioning Research, 27*(8), 2079-2085.

Simoneau, J.-A., & Bouchard, C. (1995). Genetic determinism of fiber type proportion in human skeletal muscle. *The FASEB Journal, 9*(11), 1091-1095.

Sisson, S. B., Katzmarzyk, P. T., Earnest, C. P., Bouchard, C., Blair, S. N., & Church, T. S. (2009). Volume of exercise and fitness non-response in sedentary, post-menopausal women. *Medicine and Science in Sports and Exercise, 41*(3), 539.

Skein, M., Duffield, R., Edge, J., Short, M. J., & Mundel, T. (2011). Intermittent-sprint performance and muscle glycogen after 30 h of sleep deprivation. *Medicine and Science in Sports and Exercise, 43*(7), 1301-1311.

Skein, M., Duffield, R., Minett, G. M., Snape, A., & Murphy, A. (2013). The effect of overnight sleep deprivation after competitive rugby league matches on postmatch physiological and perceptual recovery. *International Journal of Sports Physiology and Performance, 8*(5), 556-564.

Smith, T. B., & Hopkins, W. G. (2012). Measures of rowing performance. *Sports Medicine, 42*(4), 343-358.

Snegovskaya, V., & Viru, A. (1993). Steroid and pituitary hormone responses to rowing: relative significance of exercise intensity and duration and performance level. *European Journal of Applied Physiology and Occupational Physiology, 67*(1), 59-65.

Spiegel, K., Leproult, R., & Van Cauter, E. (1999). Impact of sleep debt on metabolic and endocrine function. *The Lancet, 354*(9188), 1435-1439.

Stanley, J., Peake, J. M., & Buchheit, M. (2013). Cardiac parasympathetic reactivation following exercise: implications for training prescription. *Sports Medicine, 43*(12), 1259-1277.

Steinacker, J. M., Lormes, W., Lehmann, M., & Altenburg, D. (1998). Training of rowers before world championships. *Medicine and Science in Sports and Exercise, 30*(7), 1158-1163.

Stults-Kolehmainen, M. A., Bartholomew, J. B., & Sinha, R. (2014). Chronic psychological stress impairs recovery of muscular function and somatic

sensations over a 96-hour period. *The Journal of Strength & Conditioning Research, 28*(7), 2007-2017.

Suay, F., Salvador, A., González-Bono, E., Sanchıs, C., Martınez, M., Martınez-Sanchis, S., . . . Montoro, J.-B. (1999). Effects of competition and its outcome on serum testosterone, cortisol and prolactin. *Psychoneuroendocrinology, 24*(5), 551-566.

Tanner, R., & Gore, C. (2013). *Physiological tests for elite athletes*: Human Kinetics.

Task-Force. (1996). Heart rate variability: standards of measurement, physiological interpretation, and clinical use. *European Heart Journal, 17*, 354-381.

Teixeira, L., Ritti-Dias, R. M., Tinucci, T., Júnior, D. M., & de Moraes Forjaz, C. L. (2011). Post-concurrent exercise hemodynamics and cardiac autonomic modulation. *European Journal of Applied Physiology, 111*(9), 2069-2078.

Terziotti, P., Schena, F., Gulli, G., & Cevese, A. (2001). Post-exercise recovery of autonomic cardiovascular control: a study by spectrum and cross-spectrum analysis in humans. *European Journal of Applied Physiology, 84*(3), 187-194.

Tesch, P. (1979). Muscle fatigue in man. With special reference to lactate accumulation during short term intense exercise. *Acta Physiologica Scandinavica. Supplementum, 480*, 1-40.

Tipton, K. D., Ferrando, A. A., Phillips, S. M., Doyle, D., & Wolfe, R. R. (1999). Postexercise net protein synthesis in human muscle from orally administered amino acids. *American Journal of Physiology-Endocrinology And Metabolism, 276*(4), E628-E634.

Tipton, K. D., & Wolfe, R. R. (2004). Protein and amino acids for athletes. *Journal of Sports Sciences, 22*(1), 65-79.

Tomlin, D. (1998). *The aerobic response to intense intermittent exercise*

Tomlin, D., & Wenger, H. A. (2001). The relationship between aerobic fitness and recovery from high intensity intermittent exercise. *Sports Medicine, 31*(1), 1-11.

Tsigos, C., & Chrousos, G. P. (2002). Hypothalamic–pituitary–adrenal axis, neuroendocrine factors and stress. *Journal of Psychosomatic Research, 53*(4), 865-871.

Tucker, R., & Collins, M. (2012). What makes champions? A review of the relative contribution of genes and training to sporting success. *British Journal of Sports Medicine*, bjsports-2011-090548.

Urban, R. J., Bodenburg, Y. H., Gilkison, C., Foxworth, J., Coggan, A. R., Wolfe, R. R., & Ferrando, A. (1995). Testosterone administration to elderly men increases skeletal muscle strength and protein synthesis. *American Journal of Physiology-Endocrinology And Metabolism, 269*(5), E820-E826.

Urhausen, A., Weiler, B., Coen, B., & Kindermann, W. (1994). Plasma catecholamines during endurance exercise of different intensities as related to the individual anaerobic threshold. *European Journal of Applied Physiology and Occupational Physiology, 69*(1), 16-20.

Van Dongen, H., Baynard, M. D., Maislin, G., & Dinges, D. F. (2004). Systematic interindividual differences in neurobehavioral impairment from sleep loss: evidence of trait-like differential vulnerability. *Sleep, 27*(3), 423-433.

van Loon, L. J., Saris, W. H., Kruijshoop, M., & Wagenmakers, A. J. (2000). Maximizing postexercise muscle glycogen synthesis: carbohydrate supplementation and the application of amino acid or protein hydrolysate mixtures. *The American Journal of Clinical Nutrition, 72*(1), 106-111.

Vesterinen, V., Nummela, A., Heikura, I., Laine, T., Hynynen, E., Botella, J., & Häkkinen, K. (2016). Individual Endurance Training Prescription with Heart Rate Variability. *Medicine and Science in Sports and Exercise*.

Volek, J. S., Freidenreich, D. J., Saenz, C., Kunces, L. J., Creighton, B. C., Bartley, J. M., . . . Maresh, C. M. (2016). Metabolic characteristics of keto-adapted ultra-endurance runners. *Metabolism: Clinical and Experimental, 65*(3), 100-110.

Vollaard, N. B., Constantin-Teodosiu, D., Fredriksson, K., Rooyackers, O., Jansson, E., Greenhaff, P. L., . . . Sundberg, C. J. (2009). Systematic analysis of adaptations in aerobic capacity and submaximal energy metabolism provides a unique insight into determinants of human aerobic performance. *Journal of Applied Physiology, 106*(5), 1479-1486.

Walburn, J., Vedhara, K., Hankins, M., Rixon, L., & Weinman, J. (2009). Psychological stress and wound healing in humans: a systematic review and meta-analysis. *Journal of Psychosomatic Research, 67*(3), 253-271.

Wilmore, J. H., Stanforth, P. R., Gagnon, J., Rice, T., Mandel, S., Leon, A. S., . . . Bouchard, C. (2001). Cardiac output and stroke volume changes with endurance training: the HERITAGE Family Study. *Medicine and Science in Sports and Exercise, 33*(1), 99-106.

Yamamoto, K., Miyachi, M., Saitoh, T., Yoshioka, A., & Onodera, S. (2001). Effects of endurance training on resting and post-exercise cardiac autonomic control. *Medicine and Science in Sports and Exercise, 33*(9), 1496-1502.

Yamasaki, Y., Kodama, M., Matsuhisa, M., Kishimoto, M., Ozaki, H., Tani, A., . . . Kamada, T. (1996). Diurnal heart rate variability in healthy subjects: effects of aging and sex difference. *American Journal of Physiology-Heart and Circulatory Physiology, 271*(1), H303-H310.

Zouhal, H., Lahaye, S. L. D., Abderrahaman, A. B., Minter, G., Herbez, R., & Castagna, C. (2012). Energy system contribution to Olympic distances in flat water kayaking (500 and 1,000 m) in highly trained subjects. *The Journal of Strength & Conditioning Research, 26*(3), 825-831.

Appendices

Appendix 1: Participant information sheet

Participant Information Sheet

January 10, 2015

Project title

Variation in response and recovery to training intensity in highly trained rowers

An invitation

Hi, my name is Ana Holt, I am a Masters student at AUT and along with Assoc. Prof Andrew Kilding and Dr Daniel Plews, I invite you to participate in a research project that examines differences in the response to various intensities of training. More specifically, we will assess whether there is a difference in the degree of response to training and amount of recovery time required after training sessions of varying intensities, between rowers who demonstrate different energy system contributions to a rowing race. The information obtained from this study will guide the individualisation of training programs to help athletes achieve optimal performance gains from the training they are performing.

Purpose of this research

Previous research has shown that individual's responsiveness to exercise training can vary greatly and is responsible for differences in performance gains following training. An individual's responsiveness to training can be measured as the extent of change of several factors following exercise; these factors include heart rate variability (the beat-to-beat variation in heart rate), the hormone cortisol, oxygen uptake, and the amount of lactate present in the blood. The measurement of these factors can be used to determine the period of time following exercise required for rest and recovery before performing a subsequent training session. This recovery period is crucial for enhancing performance, as it allows the most adaptive changes to occur in the body following a training session.

Knowing an athlete's individual recovery period to various exercise intensities is valuable for optimising training gains, therefore we propose to answer the following questions through this research project:

1) Do rowers require different recovery durations following different types of high-intensity rowing?
2) Do rowers who demonstrate different energy system contributions to a 6-minute rowing race have different requirements for recovery duration following training of various intensities?

Furthermore, the findings of this research will contribute to a Masters thesis, and may be used for submission to peer review journals.

How was I identified and why am I being invited to participate in this research?

As a member of one of Rowing New Zealand's Regional Performance Centres (RPCs) you have been invited to take part in this study.

However, you will not be able to partake in this study if you identify with any of the following:

- You currently have an illness or injury that would inhibit your performance on the rowing machine or put you at risk of further injury.
- You have cultural or religious sensitivities regarding human body measurements.
- You have any reason, medical or otherwise, to consider that you are not in good health and of average, or above average fitness.

What will happen in this research?

Before participating in this research, you will need to read through this information sheet and provide your informed consent by returning the signed consent form to Ana. After which your involvement in this research will require you to perform: 1) Preliminary testing; 2) Interval training sessions and 3) Post training measurements.

Preliminary testing:

- This will take place at the Cambridge Avantidrome, and will consist of 10 minutes of resting heart rate recording, followed by a 30-minute submaximal step test

whereby your expired air will be measured as you breathe through a snorkel-like mouthpiece.

- After a 30-minute rest period, you will then perform a maximal effort 6-minute ergometer test. Expired air will also be collected throughout this test, as will three blood samples from a small needle prick in your finger within 10 minutes following this test.

Interval training sessions:

- You will perform four ergometer interval-training sessions at your usual erg session location; each session will be separated by seven days.
- The day before and the day of each session you will be asked to keep your food and drink consumption the same by keeping a food diary before the first training session and replicating this for the remaining sessions. You will also be asked to refrain from consuming caffeine 12 hours prior to and 48 hours following each training session.
- You will perform a standardized warm up on the rowing ergometer prior to each training session
- Each training session will be performed on the rowing ergometer and will be between 30-60 min duration, containing 10 min, 30 sec, or 3.5 min work intervals separated by rest periods of 2 to 5 min.
- Before and within the 30-minutes following each session a blood sample from one finger prick, salivary cortisol from passive drool into a collection tube, heart rate variability from a heart rate strap worn around the chest, expired air collected from breathing through a snorkel-like mouthpiece, session rating of perceived exertion (sRPE) from a scale used to identify your view of each training session's intensity, and recovery status from your completion of a questionnaire will be measured.

Post-training measurements

- Prior to your subsequent afternoon and morning training sessions at 10, 24, 34, 48, 58, and 72 hours following each of the training sessions your heart rate variability, sRPE, and perceived recovery status will be measured.

- You will also be asked to perform a 5-minute warm up followed by a 30-second maximal sprint on the ergometer. After which you will perform your regular squad training session.
- Following the post-training measurements at 72 hours, you will be asked to resume regular squad training for the remaining four days of the week. The interval training sessions and post-training measurements protocol will be repeated two more times with differing interval training sessions, commencing at the start of each week.
- The blood samples taken to measure your blood lactate will indicate how your body responds to each of the training sessions. Feedback regarding your results will be given after all participants have completed the final measurements. The results of this study will be submitted as a thesis and may be submitted to peer review journals.

What are the discomforts and risks?

The discomforts you may experience as a participant in this study involve those that you would usually experience during your normal high intensity training sessions, and 2000 m ergometer test (heavy breathing and a burning sensation in the legs). You may also experience some discomfort as a small sting from the needle during blood collection, however this is in the form of a minimally invasive fingertip prick.

What are the benefits?

The benefits you will gain from participating in this research include finding out how your energy systems perform in a race, how your body responds to the various intensities of training you normally perform, and the period of time required after completion of each of these intensities you require to achieve an optimally recovered state. This information will guide your coach (if you have granted permission for results to be shared with your coach) in individualising your training program to ensure you are recovering sufficiently between training sessions. This will aid your body to get the most out of each training session and allow you to perform at your best in subsequent training sessions, which is crucial for enhancing performance. The benefit I will receive from completing this research regards obtaining my Masters qualification.

What compensation is available for injury or negligence?

In the unlikely event of a physical injury as a result of your participation in this study, rehabilitation and compensation for injury by accident may be available from the Accident Compensation Corporation, providing the incident details satisfy the requirements of the law and the Corporation's regulations.

How will my privacy be protected?

All data collected in this study will be available only to the researchers involved. Your name will not be associated with any data published in the public domain, and the confidentiality of all participants' data collected in this study will be maintained after its conclusion.

What are the costs of participating in this research?

Costs to the participants involved in this study include the time required to complete preliminary testing (~60 min), interval training sessions (~60 min each), and post-training measures (~20 min each). Also, travel to and from the Cambridge Avantidrome for preliminary testing, and time away from regular training for the two days of preliminary testing and four interval-training sessions.

What opportunity do I have to consider this invitation?

You have until July 12 in which you can consider this invitation and respond to me if you are interested in participating. If you have any questions or require further information regarding the study, please contact me (Ana, contact details below). Please note that participation in this study is voluntary and you have the right to withdraw your participation at any point prior to the completion of data collection, for any reason, without issue.

Will I receive feedback on the results of this research?

After completion of your participation in the study, you will be provided with verbal feedback of your results, written feedback will also be provided upon request. Your results will only be shared with your coach if you have granted us permission to do so.

How do I join the study?

If you are interested in participating in this study the next step is to contact me (Ana, contact details below), whereby I can answer any further questions you may have, provide you with a consent form, and further details regarding the data collection process. Once you have signed and returned the consent form you may participate in the study, provided you meet the criteria listed above.

What do I do if I have concerns about this research?

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

Associate Professor Andrew Kilding, andrew.kilding@aut.ac.nz, 09 921 9999 ext 7056

Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTEC, Kate O'Connor, *ethics@aut.ac.nz*, 09 921 9999 ext 6038.

Whom do I contact for further information about this research?

Researcher Contact Details:

Name: Ana Holt
E-mail: scn3171@aut.ac.nz

Project Supervisor Contact Details:

Name: Associate Professor Andrew Kilding
E-mail: andrew.kilding@aut.ac.nz
Ph: 09 921 9999 ext 7056

Approved by the Auckland University of Technology Ethics Committee on *24/02/2015*.

AUTEC Reference number *15/36*

Appendix 2: Consent form

Consent Form

Project Title: *Variation in response and recovery to training intensity in highly trained rowers*

Project Supervisors: *Assoc Prof Andy Kilding, Dr Daniel Plews*

Researcher: *Ana Holt*

- I have read and understood the information provided about this research project (Information Sheet dated 10 January, 2015). **Yes/No**
- I have had an opportunity to ask questions and to have them answered. **Yes/No**
- I am in good health and am not currently suffering from any injury or illness which may impair my physical performance **Yes/No**
- I agree to provide saliva and blood samples from finger pricks, and will inform the researchers before participation if I require my samples to be returned after analysis. **Yes/No**
- I understand that I will be informed of all of my results after the completion of my participation in this study. **Yes/ No**
- I understand that I may withdraw myself, or any information that I have provided for this project at any time prior to completion of data collection, without being disadvantaged in any way. **Yes/No**
- I agree to take part in this research. **Yes/No**
- I agree to allow the use of my collected data to be used for research, including journal publications and post-graduate thesis **Yes/No**
- I agree to allow the use of my collected data to be shared with my coach. **Yes/No**

Participant signature :_____

Parent/Guardian signature if Under 18 :_____

Participant Name : _____

Date : _____

Project Supervisor Contact Details:

Assoc Prof Andrew Kilding

Sports Performance Research Institute New Zealand

AUT|Millennium

17 Antares Place, Mairangi Bay, 0632

Phone: 09 921 9999 x 7056

Email: andrew.kilding@aut.ac.nz

Approved by the Auckland University of Technology Ethics Committee on

24/02/2015

Appendix 3: Ethical approval letter

AUTEC
SECRETARIAT

24 June 2015

Andrew Kilding
Faculty of Health and Environmental Sciences

Dear Andrew
Re: Ethics Application: **15/36 Individual variation in response and recovery to training intensity in highly trained rowers.**

Thank you for your request for approval of an amendment to your ethics application. I have approved the minor amendment to your ethics application allowing changes to the inclusion criteria and the testing protocols.

I remind you that as part of the ethics approval process, you are required to submit the following to the Auckland University of Technology Ethics Committee (AUTEC):

- A brief annual progress report using form EA2, which is available online through http://www.aut.ac.nz/researchethics. When necessary this form may also be used to request an extension of the approval at least one month prior to its expiry on 23 February 2018;
- A brief report on the status of the project using form EA3, which is available online through http://www.aut.ac.nz/researchethics. This report is to be submitted either when the approval expires on 23 February 2018 or on completion of the project.

It is a condition of approval that AUTEC is notified of any adverse events or if the research does not commence. AUTEC approval needs to be sought for any alteration to the research, including any alteration of or addition to any documents that are provided to participants. You are responsible for ensuring that research undertaken under this approval occurs within the parameters outlined in the approved application.

AUTEC grants ethical approval only. If you require management approval from an institution or organisation for your research, then you will need to obtain this.

To enable us to provide you with efficient service, please use the application number and study title in all correspondence with us. If you have any enquiries about this application, or anything else, please do contact us at ethics@aut.ac.nz.

All the very best with your research,

Kate O'Connor
Executive Secretary
Auckland University of Technology Ethics Committee
Cc: Ana Holt scn3171@aut.ac.nz, Daniel Plews

Appendix 4: Participant recruitment advertisement

WANTED
Research Participants

Individual variation in response and recovery to training intensity in highly trained rowers

You will gain valuable information about how your body responds to various training intensities and what the best recovery period is for you following these training intensities

You may be eligible to take part in this study if you meet the following criteria:

- Are a current member of a Rowing NZ Regional Performance Centre (RPC)
- Are currently training towards RPC selection

If you are interested please see the contact details below to get further information:

Primary Researcher
Ana Holt
scn3171@aut.ac.nz

Primary Supervisor
Andrew Kilding, Ph 09 921 9999 ext. 7056
andrew.kilding@aut.ac.nz

Secondary Supervisor
Daniel Plews
daniel.plews@hpsnz.org.nz

AUT UNIVERSITY