



The Practical Application of Heart Rate  
Variability –  
Monitoring Training Adaptation  
in World Class Athletes  
2013

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## **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.

A handwritten signature in black ink, appearing to read 'D. J. Plews', with a large, sweeping flourish underneath.

Daniel J. Plews



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Study 2 (and part of Study 5) was approved in accordance with the Declaration of Helsinki (see original paper, Buchheit et al. 2010; Ethics Application Number not obtained).

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## CO-AUTHORED WORKS

### *Manuscripts Published (Chapters 3-7)*

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Plews DJ, Laursen, PB, Le Meur Y, Hausswirth C, Kilding AE, Buchheit M (2014) Monitoring training with heart rate variability: How much compliance is needed for valid assessment? *International Journal of Sports Physiology and Performance*, Epub ahead of Print. (Plews 82.5%, Laursen 5%, Le Meur 2.5%, Hausswirth 2.5%, Kilding 2.5%, Buchheit 5%)

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## ABSTRACT

Few will disagree that it is the preparatory exercise training completed that makes the most substantial impact on individual exercise performance. It is not surprising therefore that we often see elite athletes that chronically live between the borders of positive adaptation and maladaptation (overtraining/non-functional overreaching) to training, as they attempt to reach the greatest fitness level possible. The adaptive response to any number of training stimuli however, are individual, with genetic factors likely being a substantial determinant of how an athlete might respond. The ability to effectively track these individual responses (positive or negative) using quantitative physiological measures would be advantageous for sports practitioners and coaches alike. Heart rate variability (HRV) provides an estimate of a person's cardiac autonomic activity, and has shown promise as a potential tool to monitor individual adaptation to endurance training. However, effective methods for assessment are yet to be established. Therefore, the overarching aim of this doctoral thesis was to establish methods by which vagally-derived indices of HRV can be practically applied to monitor and assess an elite endurance athlete's adaptation to training in an every-day setting. In order to answer this question effectively, this thesis is made up of one case comparison, two methodological studies, one current opinion and an observational study.

In the first study of the thesis, a case comparison, the daily HRV and training ( $23 \text{ h} \pm 2 \text{ h}$  per week) were monitored over a 77-day period in two elite triathletes (one male: 22 yr,  $\dot{V}\text{O}_{2\text{max}}$   $72.5 \text{ ml.kg.min}^{-1}$ ; one female: 20 yr,  $\dot{V}\text{O}_{2\text{max}}$   $68.2 \text{ ml.kg.min}^{-1}$ ). During this period, one athlete performed poorly in a key triathlon event and was diagnosed as non-functionally over-reached (NFOR). The 7-day rolling average of the log-transformed square root of the mean sum of the squared differences between R-R

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intervals ( $\ln$  rMSSD) were compared to the individual smallest worthwhile change (SWC).  $\ln$  rMSSD values declined towards the day of the triathlon event (slope =  $-0.17$  ms/wk;  $r^2 = -0.88$ ) in the NFOR athlete, and remained stable in the control athlete (slope =  $0.01$  ms/wk;  $r^2 = 0.12$ ). Furthermore, in the NFOR athlete, the HRV coefficient of variation (CV of  $\ln$  rMSSD 7-day rolling average) revealed large linear reductions towards NFOR (i.e., linear regression of HRV variables vs. day number towards NFOR:  $-0.65$  %/wk and  $r^2 = -0.48$ ), while these variables remained stable for the control athlete (slope =  $0.04$  %/wk). These data suggest that trends in both absolute HRV values and day-to-day variations along with the individual SWC may be useful measurements indicative of the progression towards mal-adaptation or non-functional over-reaching.

Considering the findings of the case comparison, showing that weekly-averaged  $\ln$  rMSSD values provided superior representation of maladaptation compared with values taken on a single day, the aim of the second study of the thesis was to compare relationships between performance, positive adaptation and HRV measured on an isolated day or with values averaged over the week. The relative change in estimated maximum aerobic speed (MAS) and 10-km running performance was correlated to the relative change in  $\ln$  rMSSD on an isolated day ( $\ln$  rMSSD<sub>day</sub>) or when averaged over 1 week ( $\ln$  rMSSD<sub>week</sub>) in 10 runners who responded to a 9-week training intervention. A trivial correlation was observed for MAS vs.  $\ln$  rMSSD<sub>day</sub> ( $r = -0.06$  ( $-0.59$ ;  $0.51$ )), while a very-large correlation was shown between MAS and  $\ln$  rMSSD<sub>week</sub> ( $r = 0.72$  ( $0.28$ ;  $0.91$ )). Similarly, changes in 10-km running performance revealed a small correlation with  $\ln$  rMSSD<sub>day</sub> ( $r = -0.17$  ( $-0.66$ ;  $0.42$ )), versus a very-large correlation for  $\ln$  rMSSD<sub>week</sub> ( $r = -0.76$  ( $-0.92$ ;  $-0.36$ )). It was concluded that the averaging of HRV values over a 1-week period appeared to be a superior method for monitoring positive adaption to training compared with assessing its value on a single isolated day.

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The third study of the thesis was based on the findings from studies 1 and 2, and its primary aim was to establish the minimum number of days that Ln rMSSD data should be averaged before equivalent outcome results were attained. Standardised changes in Ln rMSSD between different phases of training (normal training, functional overreaching, overall training and taper) and the correlation coefficients were compared when averaging Ln rMSSD from 1 to 7 days, randomly selected within the week. Standardised Ln rMSSD changes (90% confidence intervals, CI) from baseline to overload (functional overreaching) were 0.20 (-0.08; 0.47); 0.33 (0.07; 0.59); 0.49 (0.17; 0.82); 0.48 (0.20; 0.76); 0.47 (0.21, 0.73); 0.45 (0.19; 0.71) and 0.43 (0.19; 0.72) using from 1 to 7 days, respectively. Correlations (90% CI) over the same time sequence and training phase were: -0.02 (-0.21; 0.25); -0.07 (-0.16; 0.3); -0.17 (-0.16; 0.3); -0.25 (-0.45; -0.02); -0.26 (-0.46; -0.03); -0.28 (-0.48; -0.5) and -0.25 (-0.45; -0.2) from 1 to 7 days, respectively. There were almost perfect quadratic relationships between standardised changes/r values vs. the number of days Ln rMSSD was averaged ( $r^2 = 0.92$  and  $0.97$ , respectively), indicating a plateau in the increase in the magnitude of the standardised changes/r values after 3 and 4 days, respectively, in trained triathletes. It was concluded that practitioners using HRV to monitor training adaptation in trained athletes should use a minimum of 3 (randomly selected) valid data points per week.

While assessing HRV in a number of elite athletes, it became clear to me that a shift in current opinion on various issues was required. Accordingly, the fourth study in this thesis, a current opinion review, outlines the changes in HRV in response to training loads and the likely positive and negative adaptations shown, along with some limitations to these reported findings. Solutions are offered to some of the



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methodological issues associated with using HRV as a day-to-day monitoring tool, including the use of appropriate averaging techniques, and the Ln rMSSD to R-R interval ratio to overcome the issue of HRV saturation in elite athletes (i.e. reductions in HRV despite decreases in resting heart rate). Finally, this work offers examples in Olympic and World Champion athletes, showing how these indices can be practically applied to assess training status and readiness to perform in the period leading up to a pinnacle event. The paper reveals how longitudinal HRV monitoring in elites is required to understand their unique individual HRV fingerprint. For the first time, it is demonstrated how increases and decreases in HRV relate to changes in fitness and freshness, respectively, in elite athletes.

In the final study of the thesis, the relationship between HRV and training intensity distribution in elite rowers (4 female, 5 male) were examined during a 26-week build-up to the 2012 Olympic Games. The weekly-averaged Ln rMSSD were reported, and compared to changes in total training time (TTT) and training time below the first lactate threshold ( $<LT_1$ ); above the second lactate threshold ( $LT_2$ ), and between  $LT_1$  and  $LT_2$  ( $LT_1-LT_2$ ). After substantial increases in training time in a particular training zone/load variable (average effect size = 1.47, 90% confidence limits (1.35; 1.59)), standardized changes in Ln rMSSD were +0.13 (trivial; unclear) for TTT, +0.20 (small; 51% chance of greater values) for time  $<LT_1$ , -0.02 (trivial and unclear) for time  $LT_1-LT_2$ , and -0.20 (small; 53% chance of lower values) for time  $>LT_2$ . Correlations for Ln rMSSD were small vs. TTT ( $r = 0.37$  (0.28; 0.45)), moderate vs. time  $<LT_1$  ( $r = 0.43$  (0.32; 0.53)), trivial vs.  $LT_1-LT_2$  ( $r = 0.01$  (-0.16; 0.17)) and small vs.  $>LT_2$  ( $r = -0.22$  (-0.27; -0.17)). These data suggest that training phases with increased time spent at high intensity suppress cardiac parasympathetic activity, whilst low-intensity training preserves and increases it. Practically, ~5% increase in high-intensity training should be

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accompanied by ~6% increase in low-intensity training, so that autonomic balance is preserved.

Collectively, the studies in this thesis demonstrate that vagally-derived indices of HRV can be used as an effective tool to individually monitor endurance training in elite athletes. The new findings included: 1) that optimal monitoring should be carried out using Ln rMSSD values averaged over a minimum of 3 days per week (or 1 micro-cycle) alongside the individual SWC, 2) that the Ln rMSSD to R-R interval ratio should also be measured when considering changes due to training as a result of HRV saturation present in elite athletes, and 3) that when monitoring training using HRV, changes should be considered in light of the training phase being completed; that is, that increases in HRV falling above the SWC during periods of overload are likely reflective of positive adaptation, and decreases in HRV below the SWC with reduced training loads (e.g. taper) are likely signs of increasing freshness and readiness to perform. HRV values falling below the SWC, coupled with substantial increases in the Ln rMSSD to R-R interval ratio during periods of high training loads may be indicative of maladaptation.

# **CHAPTER ONE: INTRODUCTION**

### 1.1 Background

After inherent talent or genetic predisposition, it is the preparatory training an athlete performs that unequivocally has the greatest impact on their performance (Tucker & Collins, 2012). It is not surprising therefore that elite athlete training programmes consist of numerous periods of high training loads, with limited time for rest and recovery (Fiskerstrand & Seiler, 2004; Laursen, 2010). These athletes seem to chronically live between the borders of positive adaptation and maladaptation (overtraining/non-functional overreaching) in an attempt to gain the greatest possible training stimulus and resulting fitness level.

The adaptive response to any number of training stimuli however, are individual, with genetic factors likely being the main contributing factor to one's unique response (Bouchard et al., 1999; Bouchard & Rankinen, 2001; Rankinen et al., 2003). As such, effective methods by which adaptive responses can be individually assessed are often sought. Examples of such attempts include the Profile of Mood States (POMS) (Pierce, 2002), the Daily Analysis of Life Demands of athletes (DALDA) questionnaire (Rushall, 1990), the Hooper scale (Hooper et al., 1995) and the Positive and Negative Affect Schedule (PANAS) (Watson, Clark, & Tellegen, 1988); all which work by measuring overall well-being of an athlete in response to a training load. However, such methods are considered 'qualitative' and 'subjective', and not necessarily an accurate measure a person's physiological state. To date, an effective and practically applicable quantitative and objective method of assessing physiological state has yet to be established.

A relatively new means of assessing physiological state and individual adaptation to training is through the daily measurement of heart rate variability (HRV),

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which is literally the variation in time between consecutive heart beats. This variation is thought to provide an estimate of a person's autonomic nervous system (ANS) activity via the heart (Hautala et al. 2009). With new technological advances in heart rate monitor devices, HRV can now be easily measured in athletes daily, by taking a 3-6 min morning heart rate reading immediately upon waking. With such methods, HRV has been used to monitor the adaptive response to training (Hedelin, Bjerle, & Henriksson-Larsen, 2001; Hedelin et al., 2000; Hynynen et al., 2006; Mourot, Bouhaddi, Perrey, Rouillon, & Regnard, 2004; Uusitalo, Uusitalo & Rusko, 1998a; Uusitalo et al., 2000; Yamamoto et al., 2001), and more recently even, to guide daily training in recreational athletes (Kiviniemi et al., 2010; Kiviniemi et al. 2007). While the outcomes from these studies are promising, the practicalities of monitoring HRV in real athletes, and whether or not any level of usefulness is gained from such an initiative, has yet to be established. Indeed, there remains conjecture within the literature, with both increases (Lee, Wood, & Welsch, 2003; Mourot et al., 2004; Yamamoto et al., 2001; Buchheit et al., 2010) and decreases (Iellamo et al., 2002; Manzi et al., 2009) in cardiac parasympathetic indices of HRV being shown to be associated with increases in fitness/performance. Similarly, increases (Hedelin, Wiklund, Bjerle, & Henriksson-Larsen, 2000), decreases (Hynynen et al., 2006; Uusitalo et al., 2000) and no change (Bosquet, Papelier, Leger, & Legros, 2003; Hedelin, Wiklund, et al., 2000; Uusitalo et al., 1998a) in parasympathetic activity have been shown to be associated with maladaptation. As such, it is clear that methods by which HRV can be used to track training responses are complicated and yet to be established.

There are a number of likely reasons that explain why practitioners and researchers have been unable to develop practically useful methods for assessing training adaptation using HRV, along with consistent outcomes. First, environmental

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factors influencing measurement noise and training-related acute changes in homeostasis can influence HRV recordings (TaskForce, 1996). Therefore, a more sensitive measure and improved methodological approach able to detect meaningful change needs to be established. Second, it is difficult to differentiate between functional and non-functional overreaching in real athletes (Halsen & Jeukendrup, 2004). For example, while HRV has been used to assess maladaptation in some studies, researchers have purposely induced athletes into this state (non-functional overreaching (NFOR) or overtraining (OT)) (Hedelin, Kentta, et al., 2000; Uusitalo, Uusitalo, & Rusko, 1998b; Uusitalo, Uusitalo, & Rusko, 2000). Thus, it is difficult to determine whether subjects in these studies were in a stage of functional or NFOR (Meeusen et al., 2013). Furthermore, in athletes that are measured in the condition where they are already in an overtrained state (Hedelin, Wiklund, et al., 2000; Hynynen, Uusitalo, Konttinen, & Rusko, 2008), authors have been unable to provide baseline HRV values prior to its occurrence. This is an important limitation of the research to date, as there is large intra-individuality within field-based HRV recordings (Al Haddad, Laursen, Chollet, Ahmaidi, & Buchheit, 2011). Second, the HRV to fitness/fatigue relationship is complicated by quadratic relationships often shown between HRV and resting heart rate (R-R interval). As such, at very high levels of vagal tone (low resting heart rate), HRV values are diminished (known as HRV saturation (Goldberger, Ahmed, Parker, & Kadish, 1994; Goldberger, Challapalli, Tung, Parker, & Kadish, 2001; Kiviniemi et al., 2004)). Accordingly, in elite athletes and athletes with extensive training histories, HRV values can be low (thought to be indicative of fatigue (Hynynen et al. 2006; Uusitalo et al. 2000) despite high levels of vagal tone and improved fitness/performance. For example, elite rowers showed very large reductions HRV (−19%) compared with pre-training values, despite moderate increases in  $\dot{V}O_{2\max}$  (+3.6%) (Iellamo et al., 2002). Furthermore, cross-sectional studies have shown lower HRV to be associated with

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superior  $\dot{V}O_{2\max}$  ( $r = -0.49$  (Buchheit, Al Haddad, Mendez-Villanueva, Quod, & Bourdon, 2011);  $r = -0.52$  (Bosquet, Gamelin, & Berthoin, 2007) in highly trained athletes. Last, there is a lack of HRV data collected in elite athletes during actual training programmes and competitive racing seasons using methods that are practically achievable each and every day. Thus, the HRV response to different training phases (e.g. recovery, overload and pre-competition taper) and its link to actual competitive performance have yet to be established. For example, although increases in HRV have been associated with positive performance outcomes (Garet et al., 2004), increases in HRV have also been found to be associated with decreases in performance, and decreases in HRV have been shown to be linked to supercompensation (i.e., period after a taper) in trained triathletes (Le Meur et al., 2013).

### 1.2 Rationale and Thesis Aims

In light of this background, the overarching aim of the thesis was to establish methods by which HRV could be practically applied to monitor elite endurance athlete's adaptation to training in an every-day setting. Such methods would allow for elite athletes to train more effectively whereby training loads can be individually monitored and therefore maximised. To achieve this objective, the thesis has been divided into five experimental chapters with the following specific aims:

1. To evaluate changes in HRV in an elite athlete who suffered maladaptation, and to establish a sound methodological approach to assess these changes.
2. To consider different methodological assessments when analysing the relationship between performance and HRV in order to evaluate positive adaptation to training.
3. To further develop methodological approaches based on parts 1 and 2, and understand how these might be applicable to sports practitioners collecting HRV values.

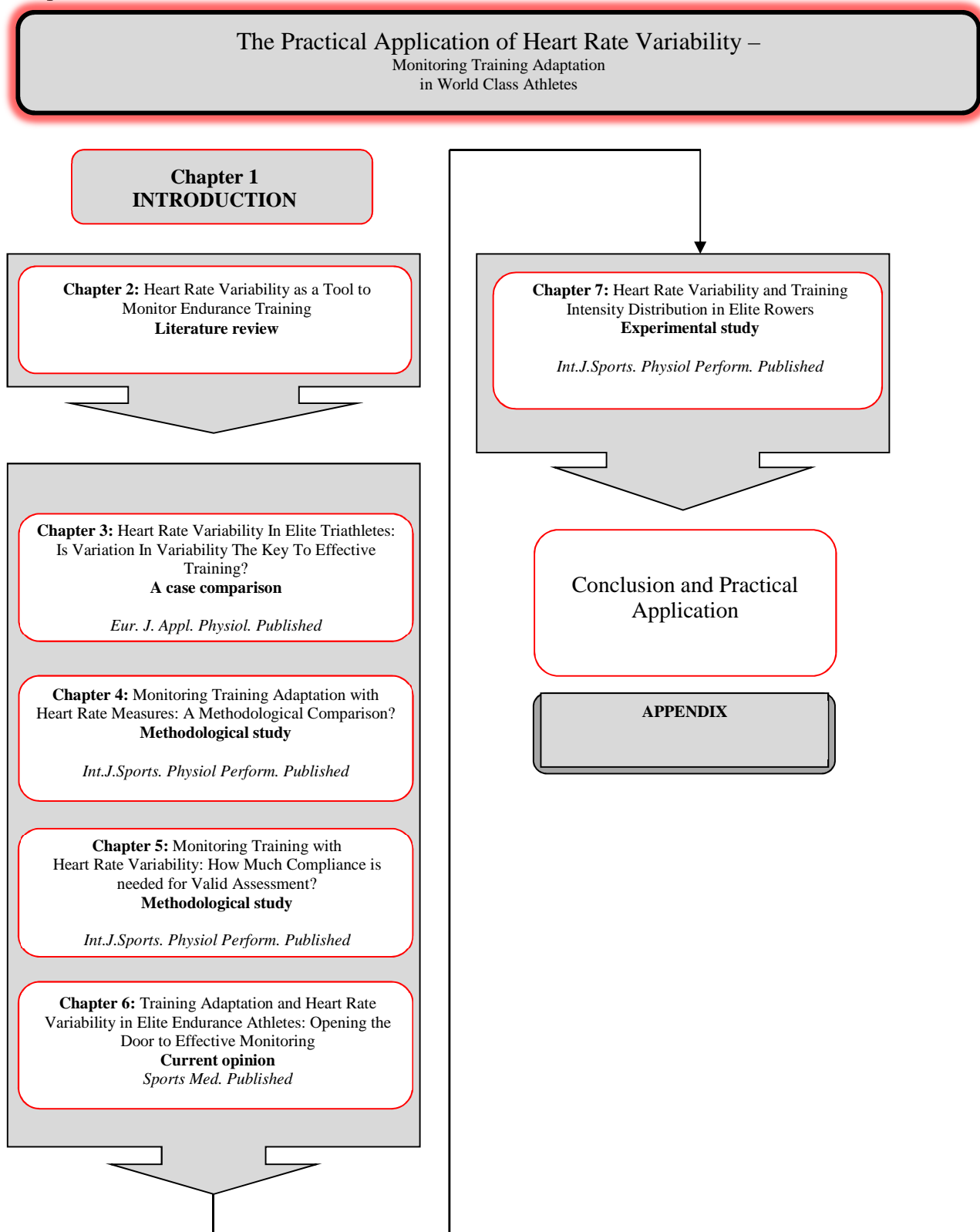
## Chapter One

4. To demonstrate solutions to some of the methodological issues associated with using HRV as a day-to-day monitoring tool. Data from Olympic and world champion athletes in the build-up to peak performance in pinnacle events will be used.
5. To determine the HRV changes in response to training intensity distribution and training load and establish ways by which these changes may influence decisions made around the use of HRV as a tool to monitor training.

### **1.3 Thesis Organisation**

This doctoral thesis is intended to inform how HRV can be used to monitoring training adaptation in elite endurance athletes. The thesis is presented in five main sections that have focused on HRV and its response longitudinally to different types of training (Figure 1). These sections include an introduction, literature review, studies (1-5), conclusion and appendix, and the studies have been written specifically for publication in peer-reviewed journals. Thus, the experimental chapters have been formatted, where necessary, to take into account word limits and publication guidelines for the respective journals for which they have been submitted to. However, for consistency and ease of reference, all citations have been presented in American Psychological Association (APA) referencing format using a single bibliography at the end of the thesis.





**Figure 1:** Overview of the structure of the thesis.

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The literature review in this thesis (Chapter 2) considers the application of HRV as a tool to monitor endurance training. First, the link between the autonomic nervous system and HRV, and various methods of HRV assessment are provided. Next the HRV literature is reviewed with specific reference to training intensity and load, positive adaption and performance, and maladaptation (i.e. overtraining). Other areas such as HRV-guided training and heart rate recovery are also explored. Based on these findings, the limitations and apparent problems when using HRV as a tool to monitor training adaptation are highlighted. Subsequent chapters of this thesis focus on the limitations and recommendations presented in the literature review, and offer a way forward for researchers and practitioners alike.

Chapters 3-7 comprise five separate published (or submitted) papers consisting of a case comparison, two methodological studies, one current opinion and one observational study. These chapters are carried out in a somewhat progressive manner, where the findings from one study naturally lead to the question raised in the subsequent one.

The final chapter of the thesis (Chapter 10) consists of a general discussion section, including a summary of key findings from each study, their limitations, along with areas for future research. Given the applied intention of the research for elite endurance sports, a practical section is also provided to enable coaches and sports scientists to use HRV as a tool to monitor training adaptation. Overall, the thesis addresses questions related to the use of HRV for monitoring training adaptation in elite athletes within a practically-applicable setting.

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### 1.4 Overview of Studies

A summary of each study is provided below:

#### *Chapter 3, Study 1*

This case comparison involves two elite triathletes in the build-up to the same key triathlon event; one of the athletes became non-functionally overreached, whilst the other achieved what was considered to be an optimal performance. The first aim of this study was to establish trends in vagally-derived indices of HRV in an athlete adapting negatively to a training stimulus. The second aim was to develop methods by which these changes could be practically monitored and assessed.

#### *Chapter 4, Study 2*

This methodological comparison is based on the findings in study 1, where HRV values averaged over a 1-week period provided a superior assessment of non-functional overreaching than HRV values taken on a single day. The aim of this study was to explore whether this same method of assessment would apply to evaluate positive adaptation to training.

#### *Chapter 5, Study 5*

The second methodological study in the thesis is based on the findings of studies 1 and 2, where weekly-averaged HRV values were shown to provide a superior method of assessment compared with values taken on an isolated day. The aim of this study was to establish the minimum number of daily HRV recordings required over a 7-day period to achieve correspondingly equivalent results to that of data averaged over an entire 1-week.

#### *Chapter 6, Study 3*

Having assessed the HRV trends of many elite athletes over a number of competitive seasons, and from my HRV assessment methods shown in studies 1 and 2, I realised that limitations existed with its interpretation. This is of particular consideration

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when observing HRV trends in key athletes building up to actual major competition. In this current opinion piece, practical methods to track vagally-derived indices of HRV effectively are offered using data from elite athletes achieving optimal performance, winning Olympic and World Championship rowing events.

## *Chapter 7, Study 4*

In this observational study, the response of vagally-derived indices of HRV to training load and training intensity in elite rowers are shown. Here, observed increases and decreases in HRV prior to competition were shown and thought to be due to changes in training load and intensity distribution. The aim of this study was to explore the relationships between these variables to assess how changes in these variables might be used to effectively monitor training.

## **1.5 Significance of Thesis**

Elite endurance athletes are always pushing the boundary between positive and negative adaptation to training in order to get as fit as possible before major events. As such, the ability to quantifiably measure an athlete's individual response to training, and instruct them to be able to do more or less training in accordance with their body's feedback, would be highly advantageous. This thesis contributes to the body of knowledge in the area of HRV by exploring practically applicable methods by which endurance athletes might be more objectively monitored during training, in order to determine whether they were positively or negatively adapting to their training programme.

**CHAPTER TWO:**  
**HEART RATE VARIABILITY AS A  
TOOL TO MONITOR  
ENDURANCE TRAINING  
LITERATURE REVIEW**

### 2.1 Introduction

It was Albert Einstein who once said *“Only one who devotes himself to a cause with his whole strength and soul can be a true master. For this reason mastery demands all of a person”*. And more often than not, success at the top levels of elite sport tends to follow a similar philosophy, where athletes with talent devote large amounts of time and deliberate practice to their sport. This desire to be the best often pushes the athlete into a very precarious position. On one side, completing such high training volumes and intensities may permit the maximal training stimulus leading to maximal adaptation. But lurking on the other side is chronic central nervous system fatigue syndrome or maladaptation (Seiler, 2010). This approach means that elite athletes are often at the ‘knives edge’ between the maximisation of effective training (achieved by duration, frequency and intensity of training) and ineffective training (e.g. maladaptation, non-functional overreaching and overtraining). Given the fact that the adaptive responses to a training load or stimulus are individual (Bouchard et al., 1999; Bouchard & Rankinen, 2001; Rankinen et al., 2003), the ability to independently assess training adaptation (positive or negative) would be advantageous to sport scientists and coaches alike.

A relatively new means of assessing physiological state and individual adaptation is via the monitoring of cardiac autonomic activity through the daily measurement of heart rate variability (HRV). Indeed, with new advances in heart rate monitors, HRV can be assessed daily and has grown in popularity over recent years. Research interest has paralleled this growth, and the HRV response associated with different training loads (Buchheit & Gindre, 2006; Buchheit, Simon, Piquard, Ehrhart, & Brandenberger, 2004; Hynynen, Vesterinen, Rusko, & Nummela, 2010; Kaikkonen, Hynynen, Mann, Rusko, & Nummela, 2010; Kaikkonen, Nummela, & Rusko, 2007; Kaikkonen, Rusko, & Martinmaki, 2008; Mouroto, Bouhaddi, Tordi, Rouillon, &

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Regnard, 2004; Parekh & Lee, 2005; Pichot et al., 2000; S. Seiler, Haugen, & Kuffel, 2007), performance (Atlaoui et al., 2007; Garet et al., 2004; Hedelin et al., 2001; Nummela, Hynynen, Kaikkonen, & Rusko, 2010), training adaptations (Atlaoui et al., 2007; Borresen & Lambert, 2008; Buchheit, Chivot, et al., 2009; Furlan et al., 1993; Hautala, Kiviniemi, & Tulppo, 2009; Hautala et al., 2003; Lamberts, Swart, Capostagno, Noakes, & Lambert, 2009; Pichot et al., 2002) and overtraining/fatigue (Hedelin, Kentta, et al., 2000; Hedelin, Wiklund, et al., 2000; Hynynen et al., 2006, 2008; Lehmann, Foster, Dickhuth, & Gastmann, 1998) have all been explored. Two studies by Kiviniemi et al. (2010 and 2007) even suggest that performance may be maximised by guiding daily training based on morning resting HRV recordings. However, to date, there remains conjecture within the literature around changes in HRV with regard to positive (Buchheit, Chivot, et al., 2010; Iellamo et al., 2002; Manzi et al., 2009; Vesterinen et al., 2011; Yamamoto et al., 2001), and negative adaptation to training (Hedelin, Kentta, et al., 2000; Hynynen et al., 2006; Uusitalo et al., 1998a; Uusitalo et al., 2000), as well as the responses of HRV to variations in training load (Atlaoui et al., 2007; Garet et al., 2004; Manzi et al., 2009). Furthermore, there is a lack of studies in elite athletes using data collected with practically-applicable methods.

The purpose of the literature review is to provide the background literature of the research to date that has used HRV as a tool to monitor training adaptation. The review begins by describing the autonomic nervous system (ANS) and its link to HRV, along with the various methods of HRV measurement used to date. The HRV response to 'training intensity and load', 'positive adaptation and performance' and 'negative adaptation' will then be discussed. Finally, although not a focus of my research, but still related to the topic, I will briefly describe the promising HRV-guided training and heart rate recovery research studies completed to date.

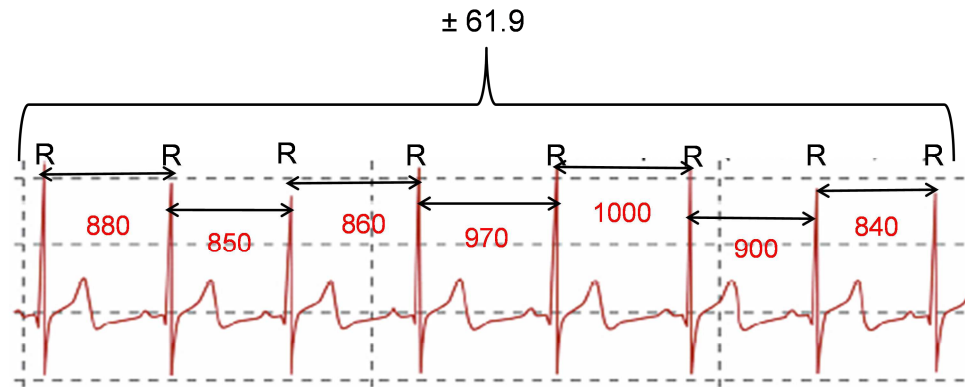
### **2.2 The autonomic nervous system**

The ANS describes those nerves that are concerned with the regulation of bodily functions. It comprises both sympathetic and parasympathetic branches, with the latter branch often referred to as ‘vagal’, due to the vagal parasympathetic nerves that supply the heart. The two branches of the ANS are complementary to one another. The activity of the sympathetic branch causes reactions, such as excitement of the heart (increased heart rate), constriction of blood vessels, along with reductions in gastrointestinal motility and constriction of sphincters. The parasympathetic branch has the opposite effect (e.g. constricts pupils, inhibits heart rate and stimulates digestive activity) (Aubert, Seps, & Beckers, 2003). As such, the ANS plays a vital role at multiple sites around the body, both during exercise and in recovery (Achten & Jeukendrup, 2003; Borresen & Lambert, 2008).

#### **2.2.1 Heart rate variability: A barometer for assessing autonomic nervous system status**

Since the ANS regulates homeostatic function of the body (Porges, 1992), it has been suggested that a non-invasive assessment of ANS status may offer an appropriate tool for quantifying training load during the transition from exercise to recovery as it returns to homeostasis (Friden, Lieber, Hragreaves, & Urhausen, 2003). One of the non-invasive tools used by clinicians to gain insight into ANS function is via the analysis of HRV. Heart rate variability is the variability in time between each successive heartbeat, and is performed by recording the R-R interval of a P-Q-R-S wave during a normal electrocardiograph (ECG) trace (Figure 2). As shown, the time between consecutive heart beats is never constant, even when heart rate seems stable (Achten & Jeukendrup, 2003). Greater variations in the R-R intervals has been linked to greater dominance of the parasympathetic system, and less variation more to a mix of sympathetic and parasympathetic dominance (TaskForce, 1996).





**Figure 2:** Analysis of heart rate variability: calculation of consecutive R-R intervals from an electrocardiography trace, with representative R-R intervals written in milliseconds. The standard deviation of all R-R intervals, reflective of variation in time between heart beats, is shown at the top ( $\pm 61.9$  ms).

### 2.2.2 Methods of assessing heart rate variability and autonomic nervous system status

Heart rate variability can be analysed in a variety of different ways, with each analysis having its own strengths and weaknesses. Methods of HRV analysis include time and frequency domain analyses as well as non-linear methods. Time-domain analysis is the simplest method of HRV analysis, and involves plotting the R-R intervals in milliseconds (ms) against time (TaskForce, 1996). Frequency domain analysis quantifies the magnitude of the periodic oscillations in R-R intervals as a function of time (measured in Hz). Indeed, both time and frequency domain analyses of HRV have contributed substantially to the understanding of ANS function (Saul, 1990; TaskForce, 1996). Non-linear methods calculate heart dynamics using quantification of the randomness or predictability of the beat-to-beat dynamics (e.g., Poincare plot; each R-R interval plotted as a function of the previous interval) (Tulppo, Makikallio, Takala, Seppanen, & Huikuri, 1996). An outline of time/frequency domain and non-linear

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methods of HRV analysis and their retrospective representation of parasympathetic and sympathetic branches of the ANS are shown in Table 1.

**Table 1:** Commonly used indices of heart rate variability (HRV). HRV indices highlighted in “bold” are indices of interest in this thesis.

HRV Index	Units	Definition	Physiological Significance
<i>Time Domain</i>			
Mean R-R	ms	Time between consecutive heart beats	Related to vagal tone
SDNN	ms	Standard deviation of all normal R-R intervals	Overall cardiac ANS activity (HRV)
<b>RMSSD</b>	<b>ms</b>	<b>The square root of the mean squared differences of successive normal R-R intervals</b>	<b>Parasympathetic modulation (short-term components of HRV)</b>
<b>Ln RMSSD</b>	<b>ms</b>	<b>The natural logarithm of the RMSSD</b>	<b>Parasympathetic modulation (short-term components of HRV)</b>
pNN50	%	Proportion of differences between adjacent normal R-R intervals that are >50 ms	Parasympathetic
<i>Frequency Domain</i>			
TP	ms <sup>2</sup>	Total power	Overall cardiac ANS activity (HRV)
LF	ms <sup>2</sup>	Low frequency power (0.04-0.15 Hz)	Parasympathetic and Sympathetic modulation
LFnu	n.u.	LF power in normalized units (LF/(TP – VLF) x 100)	Parasympathetic and Sympathetic modulation
HF	ms <sup>2</sup>	High frequency power (0.15-0.4 Hz)	Parasympathetic modulation
HFnu	n.u.	HF power in normalized units (HF/(TP – VLF) x 100)	Parasympathetic modulation
LF/HF	ratio	Ratio of LF/HF	Sympathovagal balance
<i>Non-Linear</i>			
SD1	Standard deviation of instantaneous R-R variability		Parasympathetic
SD2	Standard deviation of continuous R-R variability		Parasympathetic and Sympathetic modulation
SD1/SD2	Ratio of SD1/SD2		Sympathovagal balance

Measurement of HRV is not without its limitations. First, both the time and frequency domain analysis of the HRV is affected by body posture, such as supine and

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standing positions, and needs to be carefully controlled during consecutive recordings (Buchheit, Al Haddad, Laursen, & Ahmaidi, 2009). Second, in regards to the time domain analysis, the sympathetic and parasympathetic branches cannot be easily distinguished. While this is possible during frequency domain analysis, there is still no index that solely represents sympathetic activity (TaskForce, 1996). Third, HRV indices are highly influenced by respiration rate due to parasympathetic modulation of the heart occurring at the respiratory frequency (the respiratory sinus arrhythmia) (Bloomfield et al., 2001). However, many researchers choose not to control for breathing frequency when making HRV recordings, as HRV changes are referenced to their own previous recordings (Buchheit, Chivot, et al., 2009). Fourth, the analyses of beat-to-beat recordings (such as time and frequency domain analysis) are only possible when heart rate is stable. To achieve 'stationary' recordings, a period of stabilisation is necessary; here subjects must assume a standardised posture, prior to and during the HRV data collection period. In many situations, such as during or immediately after exercise, heart rate will not be stable. In such a scenario, HRV indices are analysed over consecutive shorter timeframes of the HRV recordings (Goldberger et al., 2006) or Poincare plots. Fifth, HRV recordings have high test-retest variability, and measurements are heavily influenced by environmental factors and prior events (e.g. exercise or other external excitations) (Al Haddad et al., 2011; TaskForce, 1996). Last, it is important to note that HRV represents vagal modulation, not vagal tone (Goldberger et al., 1994; Malik et al., 1993).

### **2.3 Heart rate variability in response to training intensity and training load**

Throughout the thesis, HRV refers to indices of cardiac parasympathetic activity (for clarification see Table 1). The fatigue associated with endurance training (single session or accumulative), or training load, is dependent on the exercise intensity, duration and frequency of the exercise bout (Friden et al., 2003). Training load can be

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quantified a number of different ways, but has historically been measured through heart rate, oxygen uptake, blood lactate concentration, rating of perceived exertion (Borresen et al, 2009) and power output (Allen & Coggan, 2006). As the ANS regulates homeostatic body functions (Porges, 1992), HRV may serve as a useful tool for quantifying training load during the transition from exercise to recovery (Friden et al., 2003). The next section provides a review of the HRV response observed with changes in exercise training intensity, volume (measured in total time or distance without taking into account intensity) and load (a combination of both intensity and volume) (Issurin, 2010).

### *2.3.1 HRV and training intensity*

Training intensity is normally measured as a fraction of an individual's maximal work capacity (e.g. maximum heart rate or  $\text{VO}_{2\text{max}}$ ), without taking into account time or distance. Conversely, training volume is typically measured as distance or time completed, without taking into account exercise intensity (Mujika, 2012). There are a number of studies that have focused on the effects of training intensity on the HRV response. The most important finding from these studies is that exercise intensity appears to suppress HRV more than training volume (Kaikkonen et al., 2007; Kaikkonen et al., 2008; Mourot, Bouhaddi, Tordi, et al., 2004; Parekh & Lee, 2005). More recently however, Kaikkonen et al. (2010) and Hynynen et al. (2010) demonstrated that increases in acute training volume can also significantly affect HRV. Kaikkonen et al. (2010) showed how increasing running distance in a single bout of exercise from 3 km to 14 km ( $60\% \text{ } \dot{V}\text{O}_{2\text{max}}$ ) substantially decreased HRV after exercise in trained male runners. Hynynen et al. (2010) revealed similar findings, showing a decrease in parasympathetic activity after a marathon run compared to a submaximal run ( $52 \pm 26$  min at  $72\%$  maximal heart rate;  $\text{HR}_{\text{max}}$ ). However, both of these studies used trained and not highly-trained subjects, a factor that likely plays a significant role

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in the post-exercise ANS status (Hautala et al., 2006). In more highly trained athletes, Seiler et al. (2007) showed no change in HRV when low intensity exercise duration was doubled from 60 min to 120 min. The authors also showed that highly-trained athletes had a significantly faster HRV recovery after high-intensity interval training (6 x 3 min at 95 % maximal oxygen uptake ( $\text{VO}_{2\text{max}}$ )) compared with trained athletes (Seiler et al. 2007). More recently, Stanley et al. (2013) quantitatively reviewed post-exercise cardiac parasympathetic reactivation in athletes and healthy individuals with respect to exercise intensity and duration, and fitness/training status. The authors showed that cardiac autonomic recovery after a low-intensity training session appeared to be recovered after 24 hr. Comparatively, threshold-intensity exercise and high-intensity exercise was recovered after 24-48 and 48 hr respectively. Furthermore, cardiac parasympathetic reactivation occurs quicker in athletes with greater aerobic fitness (Hautala et al., 2003). This suggests that both exercise intensity and aerobic fitness are the most important determinants of parasympathetic suppression following exercise.

### ***2.3.2 HRV and training volume and load***

Training load is an objective measure of biological stress imposed by a training session, and takes into account both training intensity and volume (Mujika, 2012). The influence of both intensified and reduced training loads on HRV has been thoroughly studied (Table 2). Moderate training loads increase HRV in well-trained and elite subjects (Iellamo et al., 2002; Manzi et al., 2009; Mourot, Bouhaddi, Perrey, Rouillon, et al., 2004; Pichot et al., 2000). However, when training loads approach higher levels (100% of an individual's maximal training load), HRV indices are reduced (Iellamo et al., 2002; Iwasaki, Zhang, Zuckerman, & Levine, 2003a; Manzi et al., 2009; Pichot et al., 2000) and are thought to rebound after periods of reduced training (e.g. taper) (Atlaoui et al., 2007; Garet et al., 2004; Pichot et al., 2002; Pichot et al., 2000). For

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example, after 3 weeks of overload training in swimmers and distance runners, HRV was reduced by 22% (Garet et al., 2004) and 38% (Pichot et al., 2000), respectively. Following 2 weeks of reduced training (69% reduction in training load compared with overload), HRV rebounded and increased by 7% in swimmers (Garet et al., 2004) and after 1 week (40% reduction in training load compared with overload) increased by 38% in distance runners (Pichot et al., 2000). Conversely however, Atlaoui et al. (2007) observed no change in vagal-related HRV in response to 4 weeks of overload or 3 weeks of taper (34% reduction in training load compared with overload).

Studies that have examined HRV responses to training loads over longer time periods (6, 9 and 12 months (Iellamo et al., 2002; Iwasaki et al., 2003a; Manzi et al., 2009)) have similarly found increases in HRV following moderate training loads, and decreases with high training loads (Iellamo et al., 2002; Manzi et al., 2009). However, in these studies low levels of HRV were also apparent prior to competition. For example, Iellamo et al (2002) showed reductions in HRV (−19%) during the highest training loads despite small increases in  $\dot{V}O_{2\max}$  (+3.6%) over a 9-month training period; and low levels of HRV were preceded by superior competitive rowing performance. Similarly, Iwasaki et al (2003a) showed over a 12-month training period that after 9 months (when training load was at its highest), HRV was at its lowest, despite very large (20.5%) improvements in  $VO_{2\max}$ . More recently, Le Meur et al. (2013) found small increases in cardiac parasympathetic activity (standardised difference = 0.38, 90% confidence limits  $\pm 0.23$ ) during 3 weeks of overload training in trained triathletes. Moreover, these values decreased back to baseline levels as a result of the taper and reduced training.

In summary, increases in training intensity suppress HRV (Stanley et al., 2013), while increases in training volume appear to do little or have no substantial effect on

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HRV, particularly with highly-trained and elite athletes (Seiler et al., 2007). Thus, HRV generally appears to increase during moderate training loads, and decrease during maximal training loads (Iellamo et al., 2002; Manzi et al., 2009; Mourot, Bouhaddi, Perrey, Rouillon, et al., 2004; Pichot et al., 2000). The reduction in HRV during high compared with moderate training loads possibly reflects the reductions in HRV in response to consecutive days of high intensity training (Kaikkonen et al., 2007; Kaikkonen et al., 2008; Seiler et al., 2007) and/or HRV saturation at low HR levels due to chronically high training loads (see more information regarding HRV saturation in chapter 5) (Buchheit et al., 2004). In some instances, increases and decreases in HRV have been associated with increases and decreases in training load respectively (Le Meur et al., 2013). Furthermore, there appears to be a disassociation between HRV, training load and fatigue, particularly in longitudinal studies or in athletes with extensive training histories; with some studies showing increases in training load and fitness, despite concomitant decreases in HRV (Iellamo et al., 2002; Iwasaki et al., 2003a; Manzi et al., 2009). Finally, out of all the studies cited above, only one involved elite athletes in their build-up to actual competition (Iellamo et al., 2002). As such, our knowledge surrounding the optimal HRV response to training overload and pre-competition tapers (in elites) is lacking.

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**Table 2:** Effects of changes in training load on vagal-related HRV, shown in chronological order by year and alphabetical order within years

Study	No. of participants; sex; fitness level	Exercise	HRV measurement timing	HRV analysis method	HRV recording method	HRV in response to moderate TL	HRV in response to high TL	HRV in response to low TL (e.g. taper, recovery)
(Pichot et al., 2000)	7; M; well-trained distance runners 8; M; healthy sedentary	3 wk progressive overload training + 1 wk recovery	2 × per week during 4 wk training cycle	TD, FD	24 h ‘Holter’ recording, 4 h nocturnal period analysed (spontaneous respiration)		↓HRV	↑HRV
(Portier, Louisy, Laude, Berthelot, & Guezennec, 2001)	6; M, 3; F; well-trained distance runners	3 wk of light training and 12 wk of intensive endurance training	Following the period of light training and following the period of intensive training	FD	Tilt-test (0.2 Hz respiration rate), no details provided	↑HRV	↓HRV	
(Iellamo et al., 2002)	7; M; elite junior rowers	9 months of progressively ↑ training load (from detrained to maximal training state)	Baseline, after 3 and 6 months (75% training load), after 9 months (100% training load)	FD	10 min supine (spontaneous respiration)	↑HRV	↔HRV	↓HRV
(Pichot et al., 2002)	6; M; sedentary middle aged adults	2 months of intensive training + 1 month of overload	Baseline, 2 × during wk 3, 5, 7, and 8 (intensive), 1 × during wk 9 (transition), 2 × during wk 10–14 (overload), 1 × during wk 21 (post detraining)	TD, FD	24 h ‘Holter’ recording, 4 h nocturnal period analysed (spontaneous respiration)	↑HRV	↔HRV	↑HRV
(Iwasaki et al., 2003a)	6; M, 5; F; healthy sedentary adults	12 month progressive endurance training program	Baseline, after 3, 6, 9, and 12 months of training	FD	6 min supine (spontaneous respiration), 6 min (0.2 Hz respiration rate)	↑HRV	↓HRV	
(Garet et al., 2004)	4; M, 3; F; recreational junior swimmers	1 wk relative rest, 3 wk intensive training, 2 wk taper; 400 m race at end of each phase	1 × at baseline, 2 × wk for during training and taper period	TD, FD	6 h nocturnal period analysed (spontaneous respiration)		↓HRV	↑HRV



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(Atlaoui et al., 2007)	9; M, 4; F; elite swimmers	4 wk of overload, 3 wk of taper	Following 27 wk of normal training (pre-overload), following overload, following taper	TD, FD	5 min supine on waking (spontaneous respiration)		↔HRV	↔HRV
(Manzi et al., 2009)	8; M; recreational endurance athletes	6 months of individualized training culminating with a marathon race	Pre- training (detrained state), after 8, 16, and 24 wk of training	FD	10 min supine (spontaneous respiration recorded rate of 0.26–0.27 Hz)	↑HRV	↓HRV	↓HRV
(Buchheit, Chivot, et al., 2010)	14; M; moderately trained runners	9 wk training program with undulating load	Resting waking values measured daily, post-exercise measured every 2 wk	TD	5 min supine on waking, and 3 min standing following a 5 min submaximal exercise test (both spontaneous respiration)		↑HRV (responders); ↓HRV (non-responders)	↑HRV

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**HRV**, heart rate variability; **M** = male; **F** = female; **FD** = frequency domain; **TD** = time domain; **TL** = training-load

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### **2.4 Heart rate variability, positive adaptation and performance**

The following section reviews the HRV response to positive adaptation with endurance training (as measured by improvements in physiological responses, e.g.  $\text{VO}_{2\text{max}}$  or performance). The response of HRV to changes in fitness is also summarised in chapter 5. However, the following provides a more extensive review.

Endurance training elicits marked changes in cardiorespiratory function in both sedentary and active individuals, concomitant to changes in cardiac vagal activity, as evidenced by reduced resting and exercise heart rate (Borresen & Lambert, 2008). Changes in vagal-related HRV are influenced by factors including age (Sandercock, Bromley, & Brodie, 2005), gender (Kiviniemi et al., 2009), baseline physical fitness (Goldsmith, Bigger, Bloomfield, & Steinman, 1997) and training status (Buchheit & Gindre, 2006). It is beyond the scope of this review to detail how each of these factors influence the time course and magnitude of changes to HRV. However, the individualised nature of changes in HRV is fundamental to its utility as a marker of training adaptation.

The changes in HRV in response to endurance training programmes have been extensively studied (Table 3). In sedentary and recreationally-trained individuals, endurance training for 2 (Lee et al., 2003), 6 (Mourot, Bouhaddi, Perrey, Rouillon, et al., 2004; Yamamoto et al., 2001) and 9 (Buchheit, Simpson, et al., 2011) weeks has been shown to induce parallel increases in aerobic fitness and HRV. For example, sedentary men completed 9 weeks of intensive endurance training followed by 4 weeks of overload training and had large and very large increases in maximal aerobic capacity (+20%) and vagal-related HRV (+67%) (Pichot et al., 2002). Gamelin et al. (2007)

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observed moderate improvements in maximal aerobic capacity (+11%), peak treadmill velocity (+12%), and velocity at a blood lactate concentration of

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**Table 3:** Longitudinal studies related to the effects of long-term exercise training on vagal-related HRV and performance/fitness, shown in chronological order by year and alphabetical order within years.

Study	No. of participants; sex; fitness level	Exercise	HRV measurement timing	HRV analysis method	HRV recording method	Main findings
(Hedelin et al., 2001)	8; M, 9; F; elite junior cross-country skiers and canoeists	7 months of training during competitive season	Pre-and post- 7 month training period	FD	5 min supine (spontaneous respiration), 1min supine (0.2 Hz respiration rate), 5 min 70° vertical tilt	↔ HRV following training; higher pre-training HRV related to $\uparrow \dot{V}O_{2\max}$
(Yamamoto et al., 2001)	7; M; healthy students	40 min cycling training @ 80% $\dot{V}O_{2\text{peak}}$ 4 x wk (matched with $\uparrow$ fitness)	Pre-exercise, 10 min, 20 min post-exercise. Baseline, after 4, 7, 28, and 42 days of training	TD, FD	5 min seated (0.25 Hz respiration rate)	$\uparrow$ HRV = $\uparrow \dot{V}O_{2\max}$
(Iellamo et al., 2002)	7; M; elite junior rowers	9 months of progressively $\uparrow$ training load (from detrained to maximal training state)	Baseline, after 3 and 6 months (75% training load), after 9 months (100% training load)	FD	10 min supine (spontaneous respiration)	$\downarrow$ HRV = $\uparrow$ rowing performance
(Pichot et al., 2002)	6; M; sedentary middle aged adults	2 months of intensive training + 1 month of overload	Baseline, 2 $\times$ during wk 3, 5, 7, and 8 (intensive), 1 $\times$ during wk 9 (transition), 2 $\times$ during wk 10–14 (overload), 1 $\times$ during wk 21 (post detraining)	TD, FD	24 h ‘Holter’ recording, 4 h nocturnal period analysed (spontaneous respiration)	$\uparrow$ HRV = $\uparrow \dot{V}O_{2\max}$
(Mourot, Bouhaddi, Perrey, Rouillon, et al., 2004)	8; M; sedentary adults	Control subjects performed 3 $\times$ 45 min sessions per wk for 6 wk according to SWEET protocol	Pre- and post- training intervention for control subjects	TD, NL	10 min supine, standing, steady-state exercise, seated (spontaneous respiration)	$\uparrow$ HRV = $\uparrow \dot{V}O_{2\max}$
(Atlaoui et al., 2007)	9; M, 4; F; elite swimmers	4 wk of overload, 3 wk of taper	Following 27 wk of normal training (pre-overload), following overload, following taper	TD, FD	5 min supine on waking (spontaneous respiration)	↔HRV following training; however $\uparrow$ HRV = $\uparrow$ swimming performance
(Manzi et al., 2009)	8; M; recreational endurance athletes	6 months of individualized training culminating with a marathon race	Pre- training (detrained state), after 8, 16, and 24 wk of training	FD	10 min supine (spontaneous respiration recorded rate of 0.26–0.27 Hz)	$\downarrow$ HRV = $\uparrow$ marathon performance

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(Buchheit, Chivot, et al., 2010)	14; M; moderately trained runners	9 wk training program	Resting waking values measured daily, post-exercise measured every 2 wk	TD	5 min supine on waking, and 3 min standing following a 5 min submaximal exercise test (both spontaneous respiration)	↑HRV = ↑10km running performance and MAS (responders to training).
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**HRV** = heart rate variability; **M** = male; **F** = female; **FD** = frequency domain; **TD** = time domain; **NL** = non-linear; **VO<sub>2max</sub>** = maximal aerobic capacity; **MAS** = maximal aerobic speed; **SWEET** = square-wave endurance exercise test

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4 mmol.L<sup>-1</sup> (+7%) following 12 weeks of endurance training, which was accompanied by an HRV increase of 29% in young men. Eight weeks of detraining was sufficient to reverse training-induced improvements in maximal aerobic capacity (small decreases, -6%) and peak treadmill velocity (-6%), however vagal-related HRV was unaffected (-1%). Buchheit et al. (2011) showed that improvements in maximal aerobic running speed ( $r = 0.52$ , 90% confidence limits  $\pm 0.17$ ) and 10-km run time ( $r = -0.73$  (-0.89; -0.41)) had moderate and large correlations with increases in resting HRV.

While this represents the typical response shown in sedentary and recreationally-trained individuals following a period of endurance training (Buchheit, Chivot, et al., 2010; Lee et al., 2003; Mourot, Bouhaddi, Perrey, Rouillon, et al., 2004; Pichot et al., 2002; Yamamoto et al., 2001), the response in athletes with extensive training histories (e.g., elite athletes) can be markedly different. In these athletes, the HRV response to training is variable, with longitudinal studies showing no change in fitness (i.e., maximal oxygen uptake ( $\dot{V}O_{2max}$ )) despite increases in HRV (Portier et al., 2001) and others showing decreases in HRV, despite increases in fitness (Iellamo et al., 2002). In elite distance runners training for 18 weeks (6 weeks moderately-intensive and 12 weeks intensive) culminating in a half-marathon or marathon competition, there was no change in  $\dot{V}O_{2max}$ , which was accompanied by a 45% increase in HRV (Portier et al., 2001). Elite rowers showed very large decreases in HRV (-19%) compared with pre-training values, despite moderate increases in  $\dot{V}O_{2max}$  (+3.6%) over a 9-month training period (Iellamo et al., 2002). Manzi et al. (2009) found that lower HRV values were strongly related ( $r = 0.81$ ) to marathon finishing time. Furthermore, cross-sectional studies have shown lower HRV to be associated with superior fitness in highly-trained athletes. For example, in 55 young male soccer players, lower HRV was associated with associated with higher  $VO_{2max}$  ( $r = -0.49$ ) and maximum aerobic speed ( $r = -0.39$ )

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(Buchheit, Al Haddad, et al., 2011). Similarly, Bosquet et al. (2007) found that aerobic fitness (measured via  $\text{VO}_{2\text{max}}$ ) was inversely correlated to HRV at rest ( $r = -0.52$ ) in twenty-eight well-trained runners.

In summary, there generally appears to be a bell-shaped relationship between vagally-related HRV and fitness. Whereas HRV tends to increase with gains in fitness in recreationally-trained athletes, HRV is seen to decrease in athletes with extensive training histories. This suggests that reductions in HRV in elites are not always associated with fatigue (Bosquet, Merkari, Arvisais, & Aubert, 2008), but in fact may reflect positive adaptation due to HRV saturation (Iellamo et al., 2002).

### **2.5 Heart rate variability and negative adaptation**

The following section focuses on HRV responses to negative adaption. Again, the changes in HRV in response to maladaptation are briefly summarised in chapter 5, which has subsequently been published in Sports Medicine (Plews, Laursen, Stanley, Kilding, & Buchheit, 2013).

Overtraining is a verb used to describe the process of undergoing intensified training to induce possible overreaching. Overreaching refers to a short-term stress-regeneration imbalance that includes negative outcomes, such as increased fatigue and reductions in performance (Meeusen et al., 2006). While overreaching is typically believed to be an important component of the elite athlete training cycle, prolonged overreaching can push an athlete into a state of non-functional overreaching (NFOR), which is associated with reductions in performance ability that do not resume for several weeks or months (Meeusen et al., 2013). The presumed link between autonomic nervous system dysfunction and overtraining/non-functional overreaching (Lehmann et

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al., 1998) has resulted in extensive research into HRV and maladaptation. A summary of these studies is provided in Table 4.



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**Table 4:** Effects of overtraining on vagal-related HRV, shown in chronological order by year and alphabetical order within years.

Study	No. of participants; sex; fitness level	Exercise	HRV measurement timing	HRV analysis method	HRV recording method	Main findings
(Uusitalo et al., 1998a)	9; F; endurance trained athletes	Individualized training program, progressively increased training load for 6–9 wk + 4–6 wk of recovery training	Pre-, after 4 wk-, after 6–9 wk- of training, after 4–6 wk of recovery training	TD, FD	5 min supine (0.2 Hz respiration rate)	Overtrained athletes trend of ↓ HRV for 4 wk of overload and ↑ HRV following recovery period; non-overtrained athletes ↑ HRV for duration of training period
(A. L. Uusitalo et al., 2000)	9; F; experimental	6-9 week progressive	Pre, after 4 wk and after 9 wks.	TD, FD	25 min supine, 5 min standing	↓ HRV heavy training (supine rest)/over-training ↓ HRV standing ↔ HRV
(Hedelin, Kentta, et al., 2000)	6; M, 3; F; elite canoeists	6 days of overload training (50% ↑ training load)	Pre- and post- the 6 day training camp	FD	Supine and 70° vertical tilt (0.2 Hz respiration rate), duration not reported	
(Hedelin, Wiklund, et al., 2000)	1; junior skier	Monitored during training period	Pre, post and recovery	FD	Supine and head tilt (12 breaths.min <sup>-1</sup> respiration rate)	↑ HRV during OT; ↓ HRV during recovery
(Bosquet et al., 2003)	9; M; well-trained endurance athletes	4 wk overload (volume ↑100%) training + 2 wk recovery	Baseline, after 4 and 6 wk	FD	5 h nocturnal period (spontaneous respiration)	↔ HRV, ↓performance, ↑fatigue, ↓Lct <sub>peak</sub> ,
(Mourot, Bouhaddi, Perrey, Cappellet, et al., 2004)	7; Overtraining syndrome. 8; control; 8; endurance trained	Diagnosed as suffering from overtraining syndrome	Post overtraining syndrome diagnosis	TD, FD	Electrocardiographic 20 min supine, 10 min tilted 60°	↓ HRV when suffering from OT syndrome
(Baumert et al., 2006)	5; M, 5; F; endurance athletes	2 wk overload training	1 wk prior to training, after 1 wk of training and after 4 d of recovery post-training	TD, FD	Supine (no specific details given)	↓ HRV following overload period, ↑ HRV following recovery period
(Hynynen et al., 2006)	6; M, 6; F overtrained; 12 control	Post training period	3-6 wk after overtraining diagnosis	TD, FD	During sleep and 5 min supine rest upon waking	↔ HRV during sleep; overtrained ↓ HRV upon waking
(Hynynen et al., 2008)	6; M, 6; F overtrained; 6; M, 6; F control	Post training period	3-6 wk after overtraining diagnosis	TD, FD	Supine, orthostatic and relaxation	Overtrained ↓ HRV orthostatic, ↓ HRV supine and relaxation
(Plews, Laursen, Kilding, & Buchheit, 2012)	1, F overtrained; 1, M control	77-day period; 23 ± 3 h training per wk	Every-day; values averaged over 1-wk	TD	Morning resting 5 min supine	↓ HRV overtrained
HRV= heart rate variability; F = female; M = male; TD = time domain; FD = frequency domain; OT = overtraining; Lct <sub>peak</sub> = peak blood lactate concentration following an incremental exercise test.						

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To date however, studies that have examined HRV and overtraining have revealed equivocal findings, with increases (Hedelin, Wiklund, et al., 2000), decreases (Hynynen et al., 2006; Uusitalo et al., 2000) and no change (Bosquet et al., 2003; Hedelin, Wiklund, et al., 2000; Uusitalo et al., 1998a) in HRV reported. In a case study of an elite cross-country skier that became overtrained, Hedelin et al. (2000) showed reduced competition performance and lowered profile of mood states, along with substantially increased HRV. Conversely, Uusitalo et al. (1998) showed that overtraining was associated with decreased HRV in moderately-trained female endurance athletes undergoing heavy training over a 6-9 week period. Likewise, Hynynen et al (2006) found slightly diminished HRV upon waking in 12 athletes who were diagnosed as being overtrained. Hedelin et al. (Hedelin, Kentta, et al., 2000) reported unchanged HRV in elite canoeists, despite decreased maximal blood lactate concentration, running time-to-fatigue, maximal and submaximal heart rate, and reduced  $\dot{V}O_{2max}$ . Similarly, Bosquet et al. (2003) reported no change in HRV despite reductions performance and increased fatigue in 9 experienced endurance athletes. However, the inconsistent findings shown between HRV and overtraining to date are likely due to the methodological approaches adopted (e.g. differences in study design, athlete training status), the sensitivity of HRV recordings (Bosquet et al., 2008; Plews et al., 2012), the high intra-individuality in HRV measures (Garet et al., 2004), lack of baseline measure prior to overtraining, and difficulty with discriminating between NFOR and overtraining (Halsen & Jeukendrup, 2004). Furthermore it's unlikely that true overtraining was reached in the studies that purposely induced overtraining (Baumert et al., 2006; Bosquet et al., 2003; Hedelin, Kentta, et al., 2000; Uusitalo et al., 1998a), which also would not reflect real-life training conditions. For example, Bosquet et al. (2003) classified only 6 athletes as overtrained and 3 as being in an overreached state. Only 5 athletes were classified as overtrained by Uusitalo et al. (1998a) due to

significant decreases in maximal aerobic capacity. Hedelin et al. (2000) also conceded that their 6-day training camp only induced a state of overreaching. As well, the recovery of HRV after only 3–4 days of reduced training indicates that athletes in the study by Baumert et al. (2006) were only overreached. Finally, the possibility of two types of overtraining being evident in athletes (parasympathetic and sympathetic; (Kuipers, 1998; Kuipers & Keizer, 1988; Lehmann et al., 1998) may further augment the differences in these equivocal research findings. Therefore the utility of vagal-related HRV as a marker of overtraining remains inconclusive.

### **2.6 Heart rate variability guided training**

Whilst not explored in this thesis, an exciting concept over recent years resides within the promise of HRV as a tool to guide training on a daily basis. This next section will briefly review the studies that have practically applied HRV to guide training. Kiviniemi et al. (2009; 2007) have shown on two occasions how training based on morning resting HRV indices may be more effective than training based upon conventional methods (i.e., pre-planned training program). In these studies, the authors showed greater improvement in  $VO_{2max}$  (Kiviniemi et al., 2009; Kiviniemi et al., 2007) and maximal attainable workload (Kiviniemi et al., 2009)) in groups of trained subjects who performed high-intensity training when morning resting HRV was high, and low-intensity training when these values were low. This was despite the HRV-guided training group performing high-intensity training sessions less frequently than a traditional training group (average 3 vs. 4 high-intensity training sessions per week). Hence, adaptation was improved when lower intensity training was completed when vagal modulation of HR was attenuated. However, such a method of daily exercise prescription based on HRV has yet to be applied in elite athletes during actual training regimes. Due to the complexities between HRV and blood plasma volume expansion

(Buchheit, Laursen, Al Haddad, & Ahmaidi, 2009) and the large variation in HRV recording (Al Haddad et al., 2011), such an approach is unlikely to be practically attainable for elite athletes attempting to maximise training load. Furthermore, there is no clear rationale as to why training with higher HRV is superior.

### **2.7 Heart rate recovery, training adaptation and performance**

While outside the scope of the thesis, another vagally-related heart rate index is the heart rate recovery (HRR), which is the rate at which heart rate declines, usually within minutes after the cessation of physical exercise (Daanen, Lamberts, Kallen, Jin, & Van Meeteren, 2012). As with HRV, HRR has also been used to assess adaptation to endurance training regimes. A faster response of HRR reflects a positive adaptation to endurance training and improved endurance performance capacity (Lamberts, Swart, Capostagno, et al., 2009; Lamberts, Swart, Noakes, & Lambert, 2009; Sugawara, Murakami, Maeda, Kuno, & Matsuda, 2001; Yamamoto et al., 2001). Heart rate recovery has been shown to be related to the progressive withdrawal of sympathetic activity (Perini et al., 1989) and parasympathetic reactivation (Buchheit, Papelier, Laursen, & Ahmaidi, 2007), and the increase in HRR after endurance training is a result of an increase in vagal reactivation (Sugawara et al., 2001). Recently, it has been suggested that HRR might offer an objective tool for predicting the impact of aerobic training on endurance performance (Buchheit, Chivot, et al., 2009; Lamberts, Swart, Capostagno, et al., 2009; Lamberts, Swart, Noakes, et al., 2009). Indeed, improvements in HRR have been shown to have strong correlation with improvements in 40-km time trials ( $r = 0.96$ ;  $P < 0.0001$ ) and peak power output ( $r = 0.73$ ;  $P < 0.0001$ ) after a 4-week training program in well-trained cyclists (Lamberts, Swart, Noakes, et al., 2009). Recently, Buchheit et al. (2010) examined HRR following 5 min of submaximal exercise (60% of MAV (5'5 HRR)) every 2<sup>nd</sup> week during an 8-week endurance

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training program in 14 trained runners. It was shown that HRR had rapid adaptations, and was correlated with improvements in 10-km running performance ( $r = -0.72$ ). As such, Buchheit et al. (2010) suggested that 5'5 HRR performed every second week may be an efficient means of assessing autonomic status. Lamberts et al. (2009a) examined the relationship between continuously increasing and decreasing HRR during a 4-week high-intensity training program in well-trained cyclists. The authors showed greater improvements in the 40-km time trial time in the group that had continuously increasing HRR compared to the group that had continuously decreasing HRR. Accordingly, it was suggested that HRR has the potential to monitor changes in endurance performance and may therefore contribute to a more accurate prescription of training load (Lamberts, Swart, Capostagno, et al., 2009). The authors suggested that the lack of improvement in athletes that displayed continuously decreasing HRR may have been a consequence of an imbalance between training load and recovery, thereby somehow blunting improvements in endurance performance (Lamberts, Swart, Capostagno, et al., 2009). In summary, it has been demonstrated that both HRV (Buchheit, Chivot, et al., 2009; A. J. Hautala et al., 2003; Nummela et al., 2010) and HRR (Buchheit, Chivot, et al., 2009; Lamberts, Swart, Capostagno, et al., 2009) have links with the adaptive responses to endurance training programs. It has also been suggested that HRR may be associated with rapid changes in training load, whereas changes in HRV indices are likely more associated with longer-term adaptation of the autonomic nervous system (Buchheit et al., 2008; Buchheit et al., 2007). Thus, both measures offer potentially useful contributions to assist with the monitoring of training adaptation in athletes.

### 2.8 Conclusion

HRV has been extensively researched as a potential tool to monitor and optimise training and resulting performance. Indeed, research to date has revealed equivocal findings, with both increases (Lee et al., 2003; Mourot, Bouhaddi, Perrey, Rouillon, et

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al., 2004; Yamamoto et al., 2001; Buchheit, Simpson, et al.) and decreases (Iellamo et al., 2002; Manzi et al., 2009) in HRV shown to be associated with increases in fitness/performance. Further equivocal findings reside within the descriptive and experimental studies surrounding maladaptation or overtraining, with no change (Bosquet et al., 2003; Hedelin, Wiklund, et al., 2000; Uusitalo et al., 1998a), decreases (Hynynen et al., 2006; A. L. T. Uusitalo et al., 2000) and increases (Hedelin, Wiklund, et al., 2000) in HRV found to be associated with its occurrence. Whilst high (maximal) training loads are typically associated with decreases in HRV (Iellamo et al., 2002; Manzi et al., 2009; Mourot, Bouhaddi, Perrey, Rouillon, et al., 2004; Pichot et al., 2000), this is not always the case (Le Meur et al., 2013). The discrepancies identified within the literature may be due to the high day-to-day variation in HRV recordings (Al Haddad et al., 2011), inconsistent methodologies (Borresen & Lambert, 2008), HRV saturation (Buchheit et al., 2004) and the complex interactions between changes in HRV and blood plasma volume expansion (Buchheit, Laursen, et al., 2009). Furthermore, many of the methodologies used in these studies are simply not applicable in a real world setting. As such future research is needed to resolve these methodological issues and devise practical methods by which HRV can be used to monitor training in elites.

**CHAPTER THREE:**  
**HEART RATE VARIABILITY IN**  
**ELITE TRIATHLETES: IS**  
**VARIATION IN VARIABILITY**  
**THE KEY TO EFFECTIVE**  
**TRAINING? – A CASE**  
**COMPARISON**

### 3.1 Abstract

**Purpose:** Measures of an athlete's heart rate variability (HRV) have shown potential to be of use in the prescription of training. However, little data exists in elite athletes who are regularly exposed to high training loads. **Methods:** This case study monitored daily HRV in two elite triathletes (one male: 22 yr,  $\dot{V}O_{2\max}$  72.5 ml.kg.min<sup>-1</sup>; one female: 20 yr,  $\dot{V}O_{2\max}$  68.2 ml.kg.min<sup>-1</sup>) training 23 h  $\pm$  2 h per week, over a 77-day period. During this period, one athlete performed poorly in a key triathlon event, was diagnosed as non-functionally over-reached (NFOR) and subsequently reactivated the dormant virus herpes zoster (shingles). **Results:** The 7-day rolling average of the log-transformed square root of the mean sum of the squared differences between R-R intervals (Ln rMSSD), declined towards the day of triathlon event (slope = -0.17 ms/wk;  $r^2$  = -0.88) in the NFOR athlete, remaining stable in the control (slope = 0.01 ms/wk;  $r^2$  = 0.12). Furthermore, in the NFOR athlete, coefficient of variation of HRV (CV of Ln rMSSD 7-day rolling average) revealed large linear reductions towards NFOR (i.e., linear regression of HRV variables vs. day number towards NFOR: -0.65 %/wk and  $r^2$  = -0.48), while these variables remained stable for the control athlete (slope = 0.04 %/wk). **Conclusion:** These data suggest that trends in both absolute HRV values and day-to-day variations may be useful measurements indicative of the progression towards maladaptation or non-functional over-reaching.



### 3.2 Introduction

Training programs of elite athletes typically consist of periods of high training loads with limited periods of rest and recovery (Fiskerstrand & Seiler, 2004; Laursen, 2010; Seiler, 2010). Knowing when to recover and for how long to do so in elite athletes can therefore be difficult. Over-reaching (OR), non-functional over-reaching (NFOR) and over-training (OT) are terms often used to describe a stress-regeneration imbalance, with negative outcomes such as hormonal changes (Meeusen et al., 2004), disturbed sleep, increased levels of fatigue and reductions in performance commonly reported (Meeusen et al., 2006). While short-term OR is typically an important component of the elite training cycle, prolonged OR pushes an athlete into NFOR or OT, which results in performance impairment and possible negative health consequences (Israel, 1976). However, where the point of transition between OR and NFOR/OT has been unsuccessfully sought by researchers and practitioners for decades (Barron, Noakes, Levy, Smith, & Millar, 1985; Morgan, Brown, Raglin, O'Connor, & Ellickson, 1987).

One of the difficulties faced by practitioners attempting to solve this problem is that the symptoms of OR and NFOR are similar, and not necessarily more severe than for OR (Halsen & Jeukendrup, 2004). For example, Rowbottom et al. (1995) showed that, with the exception of glutamine, resting haematological, biochemical and immunological measures in ten over-trained athletes were identical to age-matched controls. As such, detecting early signs of NFOR may be difficult (Halsen & Jeukendrup, 2004). Indeed, the current consensus is that no sound objective tool has been identified which can detect the early signs of NFOR (Halsen & Jeukendrup, 2004).

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Conventionally, an individual's change in resting heart rate has been used by sport practitioners as a practical method to detect early signs of NFOR and central nervous system fatigue (Halson et al, 2002; Lehmann et al, 1992; Dressendorfer et al, 1985). However with advancements in technology, the beat-to-beat variation in resting pulse rates, or heart rate variability (HRV) (TaskForce, 1996), can now be easily assessed and provide an indication of autonomic nervous system (ANS) status (Lehmann et al., 1998), and has become more popular over recent times. It has been proposed that an altered ANS status may co-exist with a number of hormonal changes often observed during OT and NFOR (Kuipers, 1998). As such, for more than a decade, HRV has been suggested to be a practical non-invasive method of assessing cardiac ANS status, and possibly NFOR/OT (Uusitalo et al., 1998b). This has further lead researchers to suggest that HRV may be used to guide the training of elite athletes on a day-to-day basis (Hautala et al., 2009; Kiviniemi et al., 2009; Kiviniemi et al., 2007).

To date, studies that have investigated HRV and OT/NFOR have revealed equivocal findings, with increases (Hedelin, Wiklund, et al., 2000), decreases (Garet et al., 2004; Hynynen et al., 2006, 2008; Uusitalo et al., 2000) and no change (Hedelin, Kentta, et al., 2000; Uusitalo et al., 1998b) in cardiac ANS activity shown. For example, in a case study of one cross country skier that became overtrained, Hedelin et al. (2000) reported reduced competition performance and lowered profile of mood states, along with substantially increased high frequency (HF) indices of HRV, suggestive of increased cardiac ANS activity towards extensive parasympathetic dominance. Similarly, Uusitalo et al. (2000) showed that OT was associated with shifted HRV markers towards reduced parasympathetic activity in nine female endurance athletes undergoing heavy training over a 6-9 week period. Conversely, however, Hedelin et al.

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(2000) reported unchanged HRV in nine canoeists after increasing training load by 50% over a six day training camp, despite observing decreases in maximal blood lactate concentration, running time to fatigue, maximal and submaximal heart rate, as well as  $\dot{V}O_{2\max}$ .

It remains unknown whether or not a relationship exists between indices of HRV and NFOR/OT, and whether or not such a marker could be used to detect the progression to NFOR/OT prior to complete manifestation. While some studies have purposely attempted to induce NFOR/OT (Hedelin, Kentta, et al., 2000; Uusitalo et al., 1998b; Uusitalo et al., 2000), the findings from these studies are limited as it is difficult to differentiate between the three stages (OR, NFOR and OT). Furthermore, studies that have investigated athletes that are already overtrained (Hedelin, Wiklund, et al., 2000; Hynynen et al., 2008) have not been able to provide baseline HRV values prior to its occurrence. This is an important consideration due to the intra-individuality of field-based HRV recordings (Al Haddad et al., 2011). Last, the methods by which HRV have been recorded are inconsistent in the literature, with some studies investigating HRV using the supine to head-tilt method (Hedelin, Kentta, et al., 2000; Hedelin, Wiklund, et al., 2000; Pichot et al., 2000; Uusitalo et al., 2000), supine-to-standing method (Hynynen et al., 2008), during sleep and upon waking (Garet et al., 2004; Hynynen et al., 2006), or post-exercise (Buchheit, Chivot, et al., 2010; Buchheit, Simpson, et al., 2011). More importantly, in most of these studies (Hedelin, Kentta, et al., 2000; Hedelin, Wiklund, et al., 2000; Pichot et al., 2000; Uusitalo et al., 2000; Hynynen et al., 2008) HRV recordings were measured on single isolated days, and not over consecutive days. Day-to-day variability in HRV values is high as they are influenced by environmental factors such as noise, temperature, light (TaskForce, 1996) and exercise

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the day(s) prior to the recordings (Buchheit et al. 2009). Individually and collectively, these factors likely account for the large discrepancy between studies (Hedelin, Kentta, et al., 2000; Hedelin, Wiklund, et al., 2000; Uusitalo et al., 1998b), and, as such, the lack of sensitivity of HRV measures to detect fatigue may at times be due to type 2 errors and the relative ‘noise’ of these recordings (Al Haddad et al., 2011).

We had the opportunity to longitudinally monitor HRV in two elite triathletes over the course of their high volume (~ 25 h/wk) training period in the build-up to key events. One of the athletes became NFOR, as diagnosed by a sports physician, and the other trained effectively, meeting personal best results in all races. In this case comparison, both HRV and the day-to-day variations in HRV of these two athletes are presented. The findings reveal issues with the current way HRV has been presented in the literature to date. We offer a potential new way of using HRV to monitor the stress and recovery balance in elite athletes; a method which could assist coaches and sport scientists to ensure cardiac ANS balance and exercise performance remain positive.

### 3.3 Methods

#### *Subjects*

Two elite triathletes of the national New Zealand triathlon team, one male (age 22 yr; height 181 cm; mass 73.9 kg and  $\dot{V}O_{2\max}$  72.5 ml.kg.min<sup>-1</sup>) training 21 h 30 min  $\pm$  2 h 45 min per week, and one female (age 20 yr; height 164 cm, mass 57.1 kg and  $\dot{V}O_{2\max}$  68.2 ml.kg.min<sup>-1</sup>) training 24 h 38 min  $\pm$  2 h 23 min per week, were being monitored daily for their morning resting HRV. Each triathlete gave signed informed consent to release their data for the purpose of this research, and the study was approved by the Human Research Ethics Committee of AUT University.

## Chapter Three

### *Training and performance assessment*

Training was monitored on a daily basis using a training dairy. Athletes filled out the total duration of training completed in that day in hours (swimming, cycling, running and weight training).

During the daily HRV recordings, both athletes competed in the same “Contact Series Olympic Distance Triathlon” held in Whangamata (New Zealand). The event took place on January 3<sup>rd</sup> 2011 (day forty-eight) and consisted of a 1500-m swim, 40-km bike and 10-km run.

### *Psychometric tests*

Every morning upon waking, each subject gave information regarding sleep quality, muscle soreness and fatigue levels. Sleep quality, muscle soreness, stress and fatigue levels were recorded on a five point Likert scale ranging from 1 (severe) to 5 (not at all) (Hooper, Mackinnon, & Howard, 1999). The present questionnaire was assessed using an in-house designed automated iPhone application (Tri-Ready, SPARC), which sent automatic alerts to coaches when athletes scored  $\leq 2$  on any of the markers.

### *Heart rate variability and resting heart rate analysis*

HRV was measured upon waking via R-R series recorded using the Polar RS800cx heart rate monitor (RS800cx; Polar Electro, Kempele, Finland). Athletes were instructed to leave both heart rate monitor watch and electrode strap by their bedside each evening to ensure minimum disturbances when applying the apparatus. The R-R series data was then analysed with the Protrainer Polar 5 software (version 5.40.171,

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Polar Electro), which has been shown to provide valid and accurate measurements of HRV (Nunan et al., 2009). Occasional ectopic beats were automatically replaced with the interpolated adjacent R-R interval values. The square root of the mean sum of the squared differences between R-R intervals (rMSSD) (TaskForce, 1996) was calculated during the last 5 min of the 6 min supine rest recording. HRV analysis was limited to rMSSD, since it reflects vagal activity (TaskForce, 1996) and has much greater reliability than other spectral indices (Al Haddad et al., 2011), particularly during ‘free-running’ ambulatory conditions (Penttila et al., 2001a). Due to the skewed nature of HRV recordings, rMSSD data were log-transformed (ln) by taking the natural logarithm (Ln). Despite differences in absolute rMSSD values ( $\sigma = 211.2 \pm 29.7$  ms vs.  $\phi = 130.8 \pm 44.6$  ms) there is no evidence that gender would affect the trends in HRV that are of interest for our study. Resting heart rate (RHR) was also calculated from the average R-R interval across the same 5 min time period ( $RHR = 60 / (R-R \text{ interval length} / 1000)$ ).

To examine the relationship between Ln rMSSD and R-R interval length, Ln rMSSD was plotted as a function of the corresponding mean R-R interval value on the same day. The relationship between Ln rMSSD and R-R intervals were identified as “linear”, “low-correlate” or “saturated” in their relationship (Kiviniemi et al., 2004). To observe the time course of the relationship between Ln rMSSD and mean R-R interval length, the ratio of Ln rMSSD to mean R-R interval length was calculated and plotted as a function of time (i.e., day).

Differences and trends in HRV and RHR were also assessed when considering data as follows: Ln rMSSD and RHR on an isolated single day taken on the seventh day of each week ( $\text{Ln rMSSD}_{\text{daily}}$  and  $\text{RHR}_{\text{daily}}$ ), Ln rMSSD and RHR as a 7-day rolling

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average ( $\text{Ln rMSSD}_{\text{rollave}}$  and  $\text{RHR}_{\text{rollave}}$ ; e.g. days 1 to 7, 2 to 8, etc), total average of  $\text{Ln rMSSD}$  over a one-week period (Monday to Sunday) and a 7-day rolling coefficient of variation (CV) of the  $\text{Ln rMSSD}$  and  $\text{RHR}$  7-day rolling average ( $\text{Ln rMSSD}_{\text{CV}}$  and  $\text{RHR}_{\text{CV}}$ ).

#### *Statistical analyses*

Weekly data (e.g. training volume, psychometric indices, and HRV values) are expressed as means and standard deviations (SD). The smallest worthwhile change (SWC) was calculated from the individual coefficient of variation (CV) in  $\text{Ln rMSSD}$  (and  $\text{Ln rMSSD}_{\text{CV}}$ ),  $\text{RHR}$ , training volume and psychometric indices data over the first two weeks of recordings. It has been suggested that a change of more or less than 0.5 of an athletes' CV is worthwhile, and should therefore be deemed of interest (Hopkins, Marshall, Batterham, & Hanin, 2009) .

Weekly differences in training volume and psychometric indices were assessed using an approach based on magnitudes of change (Hopkins et al., 2009). The magnitude of change between weeks was expressed as standardized mean differences (effect size, ES) (Cohen, 1988), using Hopkins' spreadsheet ([www.sportsci.org](http://www.sportsci.org)). The following threshold values for ES statistics were adopted:  $\leq 0.1$  (trivial),  $> 0.2$  (small),  $> 0.6$  (moderate),  $> 1.2$  (large) and  $> 2.0$  (very large) (Hopkins et al., 2009). Quantitative chances of a 'real' change (better/greater, similar, or worse/lower) in psychometric indices from one week to the next were also assessed qualitatively as follows:  $< 1\%$ , almost certainly not; 1-5%, very unlikely;  $> 5\text{-}25\%$ , unlikely;  $> 25\text{-}75\%$ , possible;  $> 75\text{-}95\%$ , likely;  $> 95\text{-}99\%$ , very likely and  $> 99\%$ , almost certain. If the chance of having

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greater or lower psychometric indices were both  $>5\%$ , the week-to-week change was deemed as unclear (Hopkins et al. 2009).

Linear regression was also used to examine the rate of the change in heart rate derived indices (RHR and  $\ln rMSSD_{\text{daily}}$ , RHR and  $\ln rMSSD_{\text{rollave}}$ , RHR and  $\ln rMSSD_{\text{CV}}$ , as well as sleep, stress, fatigue and muscle soreness) as a function of the time (day number) towards NFOR. For consistency between indices and to allow comparison between both control and NFOR athletes, the linear regressions focused on day-1 of the HRV recordings to the day of the triathlon race (day-48). The linear model was used for all variables since it provided the lowest residuals. The  $r^2$  values were converted to  $r$  values in order to use the following adapted criteria to interpret the magnitude of the relationship, where  $>0.1-0.3$  is small,  $>0.3-0.5$  is moderate,  $>0.5-0.7$  is large,  $>0.7-0.9$  is very large and  $>0.9-1.0$  is almost perfect (Hopkins et al., 2009).

### 3.4 Results

#### *Athlete progression and non-functional overreaching*

The average weekly training volume, week-by-week change (ES) can be viewed in Table 5. During the observational period, both athletes completed very high training loads ( $21 \text{ h } 30 \text{ min} \pm 2 \text{ h } 45 \text{ min}$ ; and  $24 \text{ h } 38 \text{ min} \pm 2 \text{ h } 23 \text{ min}$  per week for control and NFOR athletes). Swimming, cycling, running and weight training made up  $27.1 \pm 8.1\%$ ,  $48.2 \pm 5.7\%$ ,  $18.7 \pm 7.0\%$  and  $6.1 \pm 2.9\%$  of total training time (weekly distribution), respectively, for the NFOR athlete. Conversely, swimming, cycling and running made up  $40.4 \pm 7.5\%$ ,  $32.5 \pm 7.5\%$  and  $27.1 \pm 1.3\%$ , respectively, of the total training volume in the control athlete. The control athlete did no weight training as part of his training regime. There was a moderate and large decrease in training volume between weeks 6-7 for both NFOR and control athletes (ES = -0.76; ES = -1.04, respectively) leading into



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the triathlon event. Following this period, the NFOR athlete had large (ES = -1.45) and moderate (ES = -0.65) decreases in her training volume (weeks 7-8 and 8-9). Conversely, the control athlete had a large (ES = 1.35) increase in his training volume between weeks 7-8 after the triathlon race.

**Table 5:** Weekly training volume (hrs) from week 1 until week 10 including effect size (ES) calculations for weekly changes ( $\Delta$ ).

Week Number	NFOR		CON		
	Total weekly training volume (hrs)	Weekly $\Delta$ (ES)	Total weekly training volume (hrs)	Weekly $\Delta$ (ES)	
1	21.5		23.3		
2 (1-2)	19.3	-0.23	22.8	0.07	
3 (2-3)	25.3	0.43	18.6	-0.57	
4 (3-4)	26.0	0.05	19.0	-0.04	
5 (4-5)	27.0	0.08	25.2	0.57	
6 (5-6)	25.8	-0.11	23.2	-0.31	
<b>7 (6-7)</b>	<b>15.6</b>	<b>-0.76<sup>†</sup></b>	<b>11.4</b>	<b>-1.04<sup>†</sup></b>	
8 (7-8)	1.4	-1.45 <sup>††</sup>	24.3	1.35 <sup>††</sup>	
9 (8-9)	0.0	-0.65 <sup>†</sup>	23.3	-0.07	
10 (9-10)	0.0	0.0	21.1	-0.11	

The darker shaded area represents the time of NFOR and the lighter shaded area represents the recovery period for the NFOR athlete. Bold text represents the week of the triathlon race. Calculation of between-week changes in training volume were expressed as effect sizes (ES), and moderate and large week-to-week changes (see “Methods” for the ES thresholds) are identified as follows:

<sup>†</sup>=Moderate change in training volume from previous week.

<sup>††</sup>=Large change in training volume from previous week.

In total, 77 HRV data points were collected from each athlete during the entire observational period. In a post-study interview with the female athlete, it was revealed that she was likely experiencing NFOR by day thirty-seven (week-6), reporting that she found it increasingly difficult to meet her training targets after this day. However the athlete continued to train, and competed in a triathlon on day forty-eight (week-7). In an event she was expected to easily win, she had a slender lead of just 37 s after the cycling section, and was then caught within 5km of the run after slowing considerably. The

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athlete was unable to finish due to perceived fatigue. On day fifty, the athlete was diagnosed by a physician as having the virus herpes zoster, commonly known as shingles. The athlete was then required to take three weeks of enforced rest and recovery and not train. In contrast, the control athlete trained well throughout the same time period, and produced a personal best result gaining a first ever top three placing in this national triathlon event.

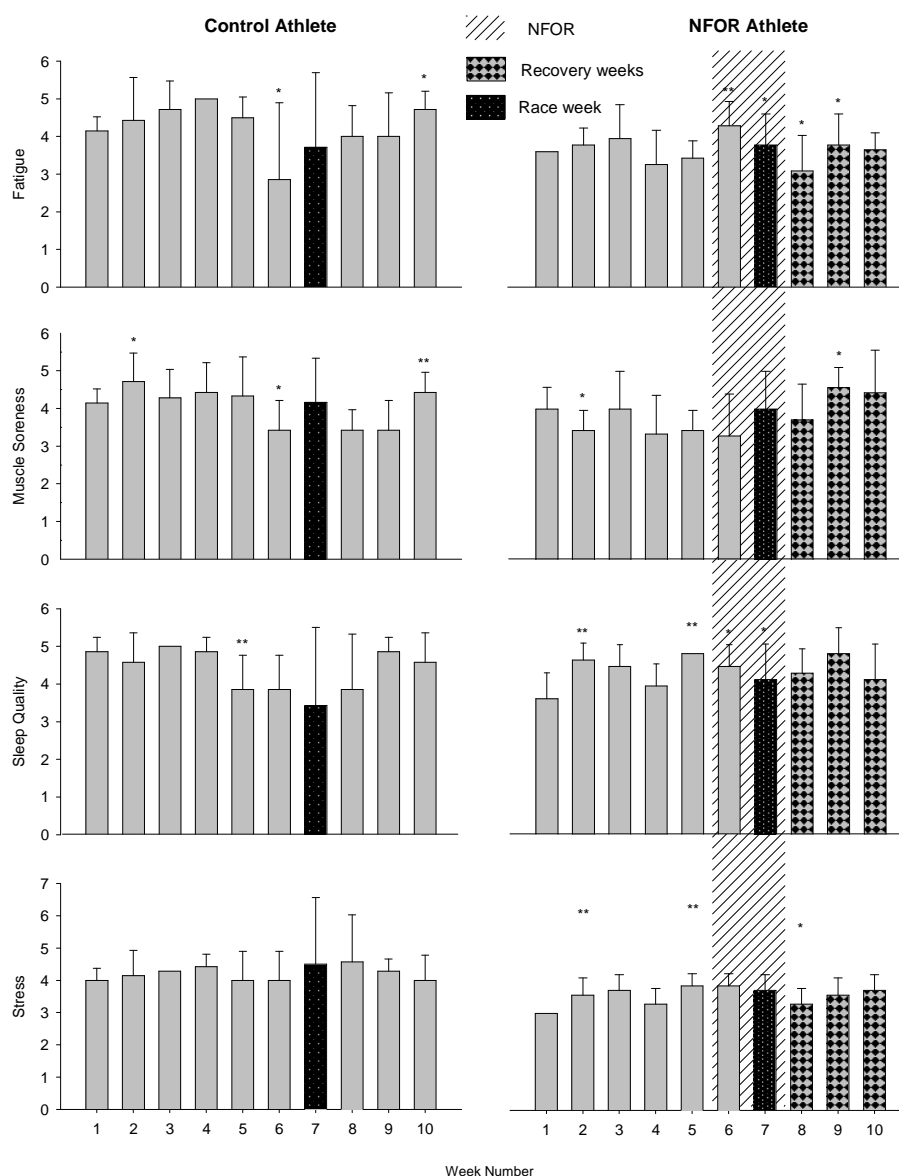
#### *Psychometric tests*

The weekly average of all psychometric indices can be viewed in Figure 3. In the case of the NFOR athlete, indices of stress were very likely better between weeks 1-2 and 4-5 (ES = 1.31, 97/2/1%; ES = 1.14, with chances for better/similar/worse values of 97/2/1%), and likely worse between weeks 7-8 (ES = -0.76, 2/7/92%). Sleep quality was very likely better between weeks 1-2 and 4-5 (ES = 1.53, 99/1/0%; ES = 1.80, 99/0/0%), and likely worse between weeks 5-6, 6-7 and 9-10 (ES= -0.72, 5/10/85%; ES= -0.38, 3/22/75%; and ES= -0.72, 5/10/85%). Muscle soreness was likely worse (ES = -0.89, 5/8/87%) and better (ES= 0.97, 91/6/3%) between weeks 1-2 and 8-9. Fatigue was very likely and likely better between weeks 5-6 and 8-9 (ES=1.34, 96/2/1%; and ES = 0.67, 89/9/2%), and likely worse between weeks 6-7 and 7-8 (ES= -0.60, 5/13/82%; ES = -0.67, 2/9/89%). Although measures changed daily, there was little relationship between sleep quality, muscle soreness and fatigue levels for the entire observational period and towards NFOR for both athletes.

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### *Heart rate derived variables*

The variable derived from the linear regressions between Ln rMSSD and RHR (absolute and CV values) vs. day number towards the triathlon competition on day can be viewed in Table 6.



**Figure 3:** Psychometric indices of stress, sleep quality, muscle soreness and fatigue for non-functionally overreached (NFOR) and control athletes over the 10-week period. Values are weekly means and standard deviations. Scales were represented from 1 (severe) to 5 (not at all) (see “Methods”). Qualitative chances of week-to-week changes being better or worse (see “Methods” for threshold values) are identified as follows: \* : likely better/worse than the previous week; \*\* : very likely better/worse than the previous week.

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*Resting heart rate and Ln rMSSD trends:*  $RHR_{daily}$  and  $RHR_{rollave}$  gradually increased (increasing RHR) to the day of the triathlon event in the NFOR athlete. Conversely,  $RHR_{CV}$  gradually declined. There was a declining trend (decreasing RHR) in all three of these same variables for the control athlete.  $Ln\ rMSSD_{daily}$  and  $Ln\ rMSSD_{rollave}$  for both the control and NFOR athlete can be viewed in Figure 4.  $Ln\ rMSSD_{daily}$  and  $Ln\ rMSSD_{rollave}$  gradually declined towards the day of the triathlon event in the NFOR athlete. However, this trend was absent for both indices in the control athlete towards the day of the competition.

**Table 6:** Linear regression between  $Ln\ rMSSD$  and day number from day 1 of recording toward the point at which both athletes competed in the triathlon event on day 48

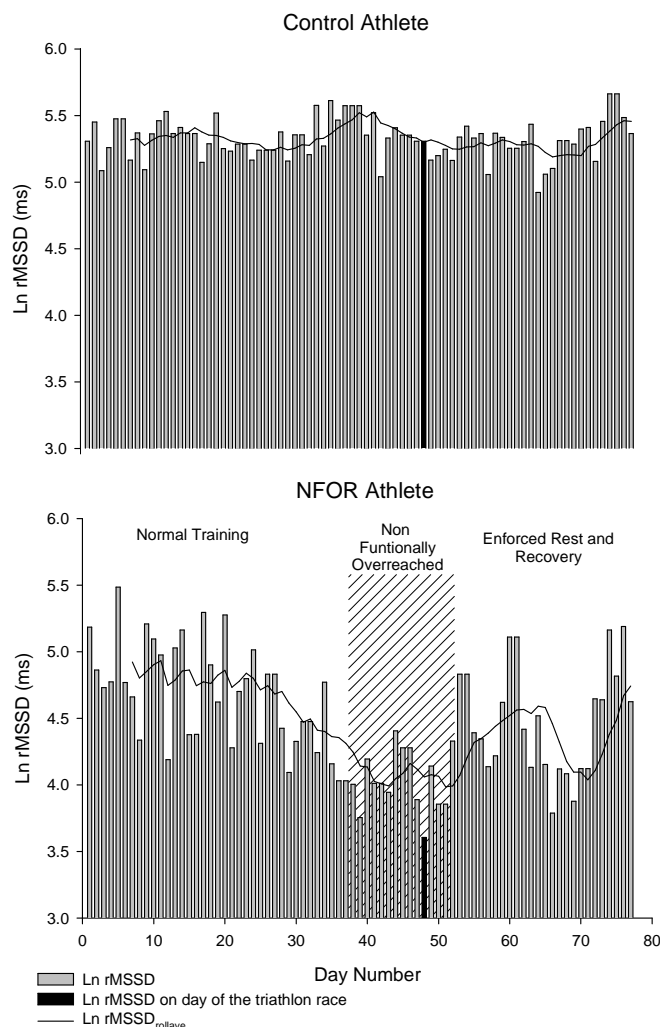
Ln value	rMSSD	NFOR			CON		
		slope (per/week)	$r^2$	Magnitude	slope (per/week)	$r^2$	Magnitude
<b>Ln rMSSD<sub>daily</sub></b>		-0.16 ms	-0.52	Very large	0.01 ms	0.02	Small
<b>Ln rMSSD<sub>rollave</sub></b>		-0.17 ms	-0.88	Almost perfect	0.01 ms	0.12	Moderate
<b>Ln rMSSD<sub>CV</sub></b>		-0.65 %	-0.48	Very large	0.04 %	0.01	Small
<b>RHR<sub>daily</sub></b>		3.12 b.min <sup>-1</sup>	0.50	Very Large	-0.34 b.min <sup>-1</sup>	-	Small
<b>RHR<sub>rollave</sub></b>		2.82 b.min <sup>-1</sup>	0.81	Almost perfect	-0.71 b.min <sup>-1</sup>	-	Very Large
<b>RHR<sub>cv</sub></b>		2.07 %	- 0.67	Very Large	-0.30 %	0.14	Moderate
<b>Stress</b>		0.07	0.16	Moderate	0.02	0.00	Unclear
<b>Sleep</b>		0.09	0.07	Small	-0.21	0.20	Moderate
<b>Muscle</b>						-	
<b>Soreness</b>		-0.05	-0.01	Unclear	-0.06	0.01	Unclear
<b>Fatigue</b>		0.03	0.01	Unclear	0.03	0.01	Unclear

Linear regressions were performed from day 1 to the point of the triathlon event on day 48 to show HR derived indices and psychometric relationships for non-functionally overreached (NFOR) and control (CON) athletes. The logarithm of the square root of the mean of the sum of the squares of differences between successive normal R-R intervals ( $Ln\ rMSSD$ ) and resting heart rate (RHR) for every day ( $Ln\ rMSSD_{daily}$  and  $RHR_{daily}$ ), rolling 7-day average ( $Ln\ rMSSD_{rollave}$  and  $RHR_{rollave}$ ), 7-day rolling standard deviation of the  $Ln\ rMSSD/RHR$  7-day rolling average ( $Ln\ rMSSD_{CV}$  and  $RHR_{SD}$ ) and psychometric indices (stress, sleep, muscle soreness fatigue) are presented.

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*Single day and averaged Ln rMSSD values:* The Ln rMSSD single day HRV measure taken on the seventh day of each week, and weekly Ln rMSSD average can be viewed in Figure 5. Ln rMSSD data (singular and average) fell above and below the SWC in the control and NFOR athlete throughout the observational period (63.6% and 100.0% data outside the SWC for individual values and 36.4% and 54.5% data below the SWC for 7-day averaged values). As such, when Ln rMSSD data was averaged, data points fell outside the SWC less in the control athlete and only around the point of non-functional over-reaching and recovery in the NFOR athlete. During the time of normal training for the NFOR athlete, singular Ln rMSSD values fell outside the SWC 100.0% of the time, compared with 20.0% of the time when averaged over one 7-day period. The weekly average of Ln rMSSD declined by 7.8% and 8.1% from weeks 4-to-5 and 5-to-6. Conversely, the same indices increased by 2.7%, 6.7% and 3.5% from weeks 6-to-7, 7-to-8 and 8-to-9 in the NFOR athlete.

*Day to day Ln rMSSD variability:* Changes in Ln rMSSD<sub>CV</sub> for the NFOR athlete can be viewed in Figure 6. Ln rMSSD<sub>CV</sub> gradually decreased toward the day of the triathlon event to reach a value 21.1% less than the SWC on the day of the event. There was no change in Ln rMSSD<sub>CV</sub> for the control athlete.



**Figure 4:** Daily changes in Ln rMSSD and 7-day rolling average (Ln rMSSDrollave) for non-functionally overreached (NFOR) and control athletes. The rolling average for the control athlete shows little change from day-1 to the day of the triathlon event, whereas the non-functional over-reached athlete (NFOR) shows a gradual decay toward the same time point.

*Relationship between Ln rMSSD and R-R interval length:* The relationship between Ln rMSSD and R-R interval length throughout the recording phase can be viewed in Figure 7 in both athletes. From day-1 until approximately day-28 the NFOR athlete displayed a saturated behaviour, as shown by the spread of the 'x' symbols

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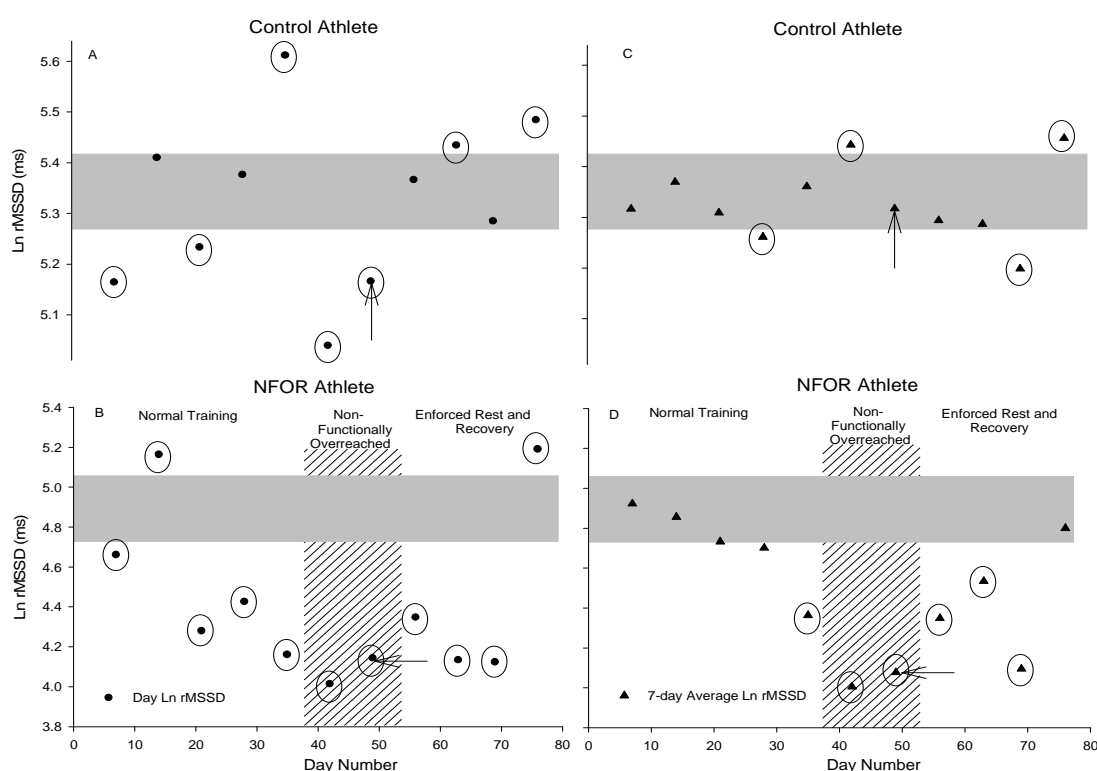
(bottom left) the lower Ln rMSSD: R-R interval length ratio (bottom right) during that period. This athlete displayed however a more linear behaviour by the day of the triathlon event, before showing a low-correlated to saturated behaviour during the enforced rest recovery period. Conversely, the control athlete displayed a low-correlated behaviour over the entire observational period.

### 3.5 Discussion

In the present study, we document the morning resting HRV responses of two elite triathletes during a 77-day competitive period; one who trained effectively and recovered adequately, and the other who completed similar training, performed poorly in a triathlon and subsequently developed signs of NFOR. Because this study was non-invasive and descriptive in nature (i.e., no interference to the athlete's regular training commitments and schedule occurred), NFOR could not be defined with precise reductions in performance as in other studies (Meeusen et al., 2006). Nevertheless, this top athlete was unable to finish a key triathlon event before developing shingles (a reactivation of the dormant virus herpes zoster; chicken pox virus dormant in nerve cells). This virus has been linked to both immunosuppression and chronic fatigue, particularly in young individuals (Goodnick, 1993), and immune function is often impaired in athletes that are overtrained (Mackinnon & Hooper, 1994) and/or OR (Lehmann, Wieland, & Gastmann, 1997; Mackinnon & Hooper, 1996). The virus resulted in the NFOR athlete being unable to train for a period of three weeks, which is obviously an undesirable response resulting in detraining (Mujika & Padilla, 2003) during the peak of the triathlon racing season. Assuming the assessment of non-functional overreaching is correct, the present data highlights at least four important findings. First, HRV appears to be a slightly more sensitive measure of NFOR detection

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than RHR. Second, it is clearly apparent that methodological issues exist when practitioners use isolated HRV values (i.e., isolated days) to detect possible physiological and/or training status changes. Third, a decreasing trend in the 7-day rolling HRV average may be a useful marker to indicate the development of NFOR. And fourth, we have shown for the first time that a decrease in the day-to-day variability HRV values and the transfer from a “saturated” to a “linear” HRV profile may be further indicators of NFOR.



**Figure 5:** Graphs A and B represent Ln rMSSD taken on a single day (every 7th day) for the non-functionally overreached (NFOR) and control athletes, respectively. If Ln rMSSD is assessed from a single day, the interpretation can be misleading. The Ln rMSSD points circled show data that are either higher or lower than the smallest worthwhile change (shaded area, see “Methods”) despite the athlete training normally. Graphs B and C represent Ln rMSSD averaged over a 7-day period for the NFOR and



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control athletes. When viewed this way, such an analysis reduces the risk of making assumptions based on single Ln rMSSD individual values. The day of the triathlon race is indicated by the arrow.

### *Psychometric indices*

In this study, we examined psychometric indices such as sleep quality, muscle soreness, stress and fatigue (Figure 3). There was no clear relationship in any of these indices towards the day of the triathlon event (Table 6). Furthermore, 64.4% of all recorded psychometric data analysed for week-to-week change was deemed unclear. However, markers of sleep quality did decline consistently during the time of NFOR (i.e., likely to be worse than the previous week), which is in agreement with other studies that have correlated reductions in sleep quality with overtraining (Hooper, Mackinnon, Howard, Gordon, & Bachmann, 1995). However, such measures which require “self-assessment” must be viewed with caution. The present questionnaire was assessed using an in-house designed automated iPhone application (Tri-Ready, SPARC), which sent automatic alerts to coaches when athletes scored  $\leq 2$  on any of the markers (Figure 3). In a subsequent interview with the NFOR athlete, she claimed that knowledge of this rule deterred her from entering numbers  $\leq 2$ . Also, changes in psychometric indices are not a prerequisite to diagnose OT or NFOR (Halson & Jeukendrup, 2004). Nevertheless, most of the psychometric indices used here (stress, fatigue and muscle soreness) were unable to identify manifestation of NFOR or poor race performance. Indeed, changes in mood state are more likely part of day-to-day OR in elite athletes, and are therefore likely to be inappropriate for identifying NFOR *per se* (Morgan, Costill, Flynn, Raglin, & O'Connor, 1988; O'Connor, Raglin, & Morgan, 1996). Such subjective measures are dependent on honest answers being given

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(Meeusen et al., 2006). In our case, HRV appeared to offer a more valid indicator of NFOR than psychometric indices.

### *Heart rate variability vs. resting heart rate*

Traditionally, RHR is the more commonly used cardiovascular-related measurement of central nervous system fatigue used by applied sport practitioners to detect NFOR. Indeed, studies have reported either no change (Halsen et al., 2002; Lehmann et al., 1992) or decreases (Dressendorfer, Wade, & Schaff, 1985) in RHR in NFOR subjects. The NFOR athlete in this study displayed a gradually increasing RHR towards the day of the triathlon event (Table. 6), which was consistent with the progressive decrease in cardiac parasympathetic activity (i.e., as inferred from Ln rMSSD). Importantly however, HRV revealed slightly stronger trends towards NFOR than RHR ( $r^2 = -0.88$  vs.  $0.81$ ). Although both measures may be useful; this data suggests that HRV may be a slightly more sensitive tool than RHR when monitoring the potential manifestation of NFOR in elite athletes.

### *Methodological issues related to the use of isolated HRV values*

When we compare the HRV responses in these two elite triathletes, it becomes evident that observing HRV on a single isolated day and attempting to diagnose the athletes' physical status from it, is somewhat of a meaningless exercise. We have shown in both a control athlete training properly and in a NFOR athlete training without adequate recovery, that Ln rMSSD viewed in isolation can fall outside of its SWC (63.6% of total Ln rMSSD points outside the SWC for control athlete and 100.0% for the NFOR athlete during normal training, Figure 5), irrespective of training phase. Regardless of the known problems with OT research (Halsen & Jeukendrup, 2004), the

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present comparison questions the findings from a number of studies that have used HRV assessment on one or two days to identify OT (Garet et al., 2004; Hedelin, Kentta, et al., 2000; Hedelin, Wiklund, et al., 2000; Hynynen et al., 2006, 2008; Pichot et al., 2000; Uusitalo et al., 1998b; Uusitalo et al., 2000) and changes in parasympathetic function due to training (Hedelin et al., 2001; Nummela et al., 2010; Vesterinen et al., 2011); as although chronic changes in HRV may be associated with changes in fitness, this is less likely the case for shorter term day-to-day changes. As such, HRV is influenced by many factors (TaskForce, 1996), but particularly for the endurance athlete, changes in blood plasma volume can have the greatest influence on shorter term day-to-day change (Buchheit, Laursen, et al., 2009). Of course, high-intensity exercise and dehydration are all known to cause marked changes in blood plasma volume (Convertino, 1991). Recently, Buchheit et al. (2011) showed that despite “theoretically” (Achten & Jeukendrup, 2003; Bosquet et al., 2008) unfavourable changes in heart rate-derived indices (over-stimulated sympathetic and depressed parasympathetic activity), highly trained young soccer players had no associated performance decrement. This supports our findings suggesting that heart rate and HRV measures are highly sensitive, and changes based on isolated readings may occur in the absence of fatigue, increased fitness or any “real” change in performance capacity.

#### *The potential use of the decreasing trend in 7-day rolling HRV average to detect NFOR*

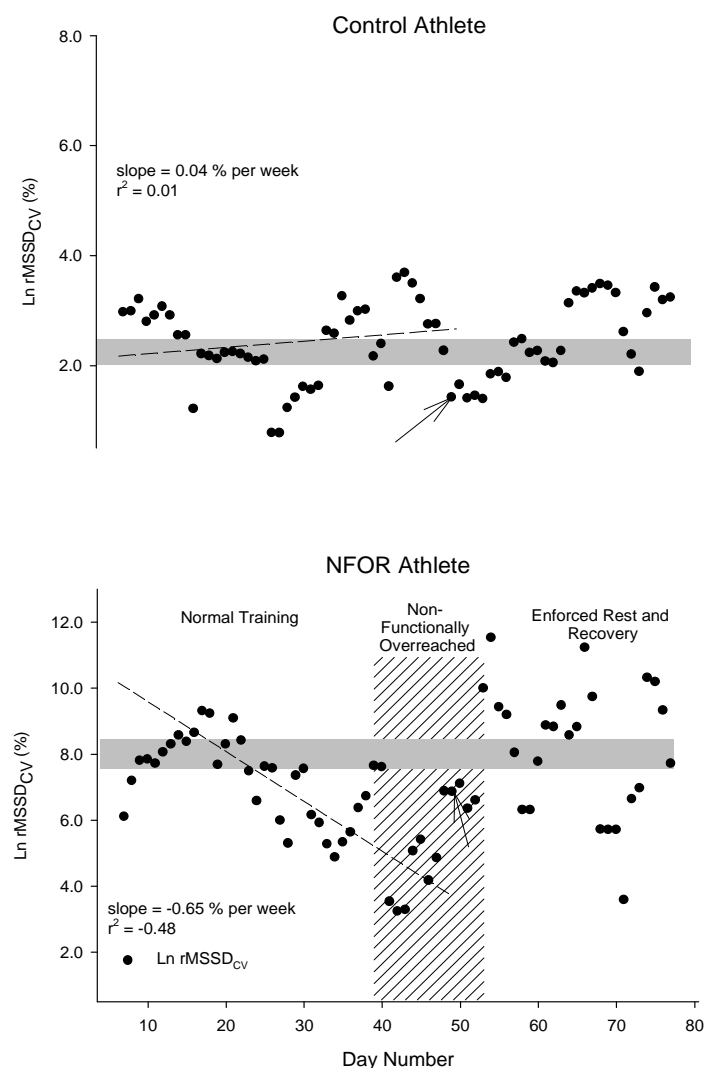
While acknowledging we report daily HRV measures for only two elite athletes, our data clearly suggests that weekly and rolling averages of Ln rMSSD data may be a more meaningful assessment of any consistent change in cardiac ANS balance compared with a single-day value. Indeed, this has been previously demonstrated in 14 moderately trained runners, where changes in weekly-averaged Ln rMSSD had a very

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large correlation with changes in 10km running performance, and likely provides superior methodological validity (Buchheit, Chivot, et al., 2009). Figure 5 (B and D) shows how for the NFOR athlete, when Ln rMSSD values are averaged over a week, Ln rMSSD falls outside of the SWC on fewer occasions (54.5 vs. 100.0%) and more importantly, less (20 vs. 100.0%) during the time of normal training (when training is going well). For the control athlete, points above (18.2% of total points) and below (18.2% of total points) the SWC were still apparent even when averaged over one week. These day-to-day changes are likely a normal part the training cycle associated with acute fatigue and recovery, and not necessary an indication of substantial changes in performance or OT/NFOR (i.e. fitness can improve but these gains can sometimes be hidden by the accumulation of fatigue). As such, perhaps two consecutive weeks of unfavourable HR-derived measurements may be more cause for concern than a single-week value in terms of a diagnostic tool for detecting NFOR (Brink, Visscher, Coutts, & Lemmink, 2010) in elite endurance athletes, whose training regimes are particularly demanding (see weekly training hours; Table 5). In this study, the fact that Ln rMSSD values showed a declining trend (Table 6) toward the point of NFOR, is in agreement with a number of studies that suggest an over-stimulated sympathetic nervous system has the potential to predict fatigue, overload (Achten & Jeukendrup, 2003; Bosquet et al., 2008) and overtraining (Hynynen et al., 2006; Uusitalo et al., 2000). However, our data suggests that an alternate analysis approach using trends (e.g. 7-day rolling and weekly average Ln rMSSD) rather than single isolated HRV assessments may improve the diagnostic utility of HRV indices in athletes.

### *The potential use of day-to-day HRV average to detect NFOR*

An innovative observation within this study was the reduction in day-to-day variability of HRV in the NFOR athlete while she progressed toward the NFOR state (slope = -0.65 %/week, Figure 6), which decreased in conjunction with Ln rMSSD<sub>rollave</sub>. Importantly, there was no change in Ln rMSSD<sub>CV</sub> over the entire observational period for the control athlete (Table 6 and Figure 6). Although both indices may be important, and Ln rMSSD<sub>rollave</sub> pertains to a larger magnitude of the regression in the present study ( $r^2 = 0.48$  vs. 0.88), perhaps Ln rMSSD<sub>CV</sub> may provide a more complete measures for diagnosing NFOR. As it is unknown whether HRV should be expected to increase or decrease in the case of OT, (Garet et al., 2004; Hedelin, Kentta, et al., 2000; Hedelin, Wiklund, et al., 2000; Hynynen et al., 2006, 2008; Pichot et al., 2000; Uusitalo et al., 1998b; Uusitalo et al., 2000), the use of Ln rMSSD<sub>CV</sub> would be applicable irrespective of the trend (towards parasympathetic or sympathetic dominance), and the stage of NFOR or OT (Kuipers, 1998). The reasons behind the low day-to-day variation when HRV values are either very high or low are unknown. However, it has been suggested that when physiological variables are either extremely high or low, they have a progressive tendency towards no response (i.e. non-responsive to further increase or return to initial value) when further stimulated (Wilder, 1958). This “law of initial values” would therefore blunt the ability of the variable to change, and hence the Ln rMSSD<sub>CV</sub>. As such, when athletes undergo periods of high training loads, a desirable response may be high day-to-day variability. With this response, an athlete is likely to be stressing (to training load) and recovering adequately on a day-to-day basis, meaning high day-to-day variation and (likely) positive adaptation. This may be an appropriate monitoring index for sports practitioners looking to ensure positive adaptive trends in their athletes.



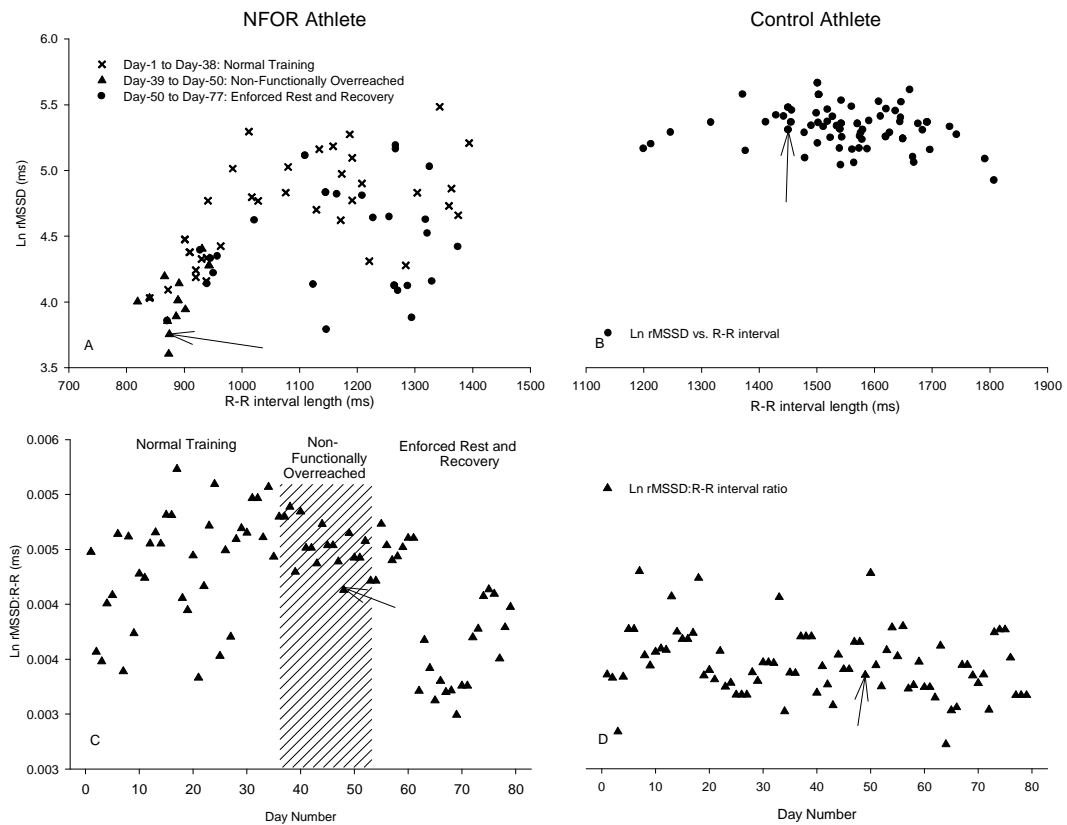
**Figure 6:** Coefficient of variation of the 7-day rolling Ln rMSSD average (Ln rMSSD<sub>CV</sub>) for non-functionally overreached and control athletes. The dashed line represents the linear regression between day number and Ln rMSSD<sub>CV</sub> towards the day of the triathlon race on day 48 (indicated by the arrow). This suggests that a lack of day-to-day change in Ln rMSSD may be an indicator of the early signs of non-functional over-reaching. The smallest worthwhile change (see “Methods”) is indicated by the shaded area.

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### *Ln rMSSD vs. R-R interval length relationship*

It has been previously shown, that at high compared to moderate training loads (>18 h/wk vs. 4-6 h/wk), HRV can decrease despite a decreasing RHR (Buchheit et al., 2004). As such, when vagal activity is already high, there appears to be a limitation in HRV as a marker of vagal modulation. A possible explanation for such saturated profiles at high levels of vagal tone the loss of phasic vagal efferent discharge or saturation of the acetylcholine receptors (Goldberger et al., 1994; Malik et al., 1993). Interestingly, the NFOR athlete displayed a saturated behaviour (Kiviniemi et al., 2004) at the time of normal training (day-1 until day-28) and became “linear” as NFOR manifested (Figure 7, B). Although the control athlete was not saturated, “low-correlated” behaviour between Ln rMSSD and R-R interval length was apparent over the entire observation period. This low correlation between parasympathetic activity and R-R intervals at such low heart rates (average RHR from day-1 until day-48 for control = 39 b.min<sup>-1</sup>) is likely a common phenomenon in elite athletes where low RHR are often observed (Buchheit et al., 2004; Iwasaki, Zhang, Zuckerman, & Levine, 2003b). As such, in some cases with elite athletes who complete high training loads regularly, saturation of HRV may be a normal scenario, and low-correlated or saturated profiles may be markers of a positive response to high training loads. Indeed, in the case of the control athlete, RHR<sub>rollave</sub> was a stronger predictor of positive adaptation than Ln rMSSD<sub>rollave</sub> ( $r^2 = -0.55$  vs. 0.12). This shows how Ln rMSSD was unable to reflect vagal modulation at such high levels. Conversely, RHR gave a better representation of increasing vagal tone, and perhaps fitness, as the triathlon race approached.

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**Figure 7:** Graphs A and B represent the relationship between Ln rMSSD and R-R interval, showing saturated and low-correlated profiles for non-functionally overreached (NFOR) and control athletes', respectively. Graphs C and D show the time scale of change (Ln rMSSD to R-R interval ratio) over the 77-day period. The day of the triathlon race is indicated by the arrows.

At the point of rest and recovery, the R-R interval to Ln rMSSD relationship returned to a saturated profile thereby re-instating vagal tone when fatigue is reduced. The possible mechanisms for such a scenario are unknown, but it does show the positive effect of recovery and how the parasympathetic system rebounded to “pre” NFOR levels within twelve days. Indicating this may be an appropriate amount of time required to recover from NFOR. While this is the first time such an observation has been made in elite athletes with low RHR, and shows the potential for R-R interval and



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Ln rMSSD relationships to be used to identify NFOR, further research with more subjects is required to confirm or refute such observed trends over a competitive season.

### **3.6 Conclusion**

For years, HRV has been studied using single day values to identify the early signs of NFOR and OT. The combined results of these studies have revealed equivocal findings. We demonstrate here, for the first time, that this may be due to the problems associated with making assumptions based on single day HRV values. As such, we propose that further research is needed to assess an optimal method of HRV assessment in well-trained endurance athletes. This method needs to measure both the trend of absolute levels of HRV, the relationship between Ln rMSSD and R-R interval, as well as that of the variation in the day-to-day HRV. Further investigation involving greater subject samples is needed to confirm the observations and presumptions of this case study.

**CHAPTER FOUR:**  
**MONITORING TRAINING**  
**ADAPTATION WITH HEART**  
**RATE MEASURES: A**  
**METHODOLOGICAL**  
**COMPARISON**

### 4.1 Abstract

The aim of this study was to compare two different methodological assessments when analysing the relationship between performance and heart rate derived indices (resting heart rate (RHR) and heart rate variability (HRV)) in order to evaluate positive adaptation to training. The relative change in estimated maximum aerobic speed (MAS) and 10-km running performance was correlated to the relative change in RHR and the natural logarithm of the square root of the mean sum of the squared differences between R-R intervals on an isolated day ( $RHR_{day}$ ;  $\ln rMSSD_{day}$ ) or when averaged over 1 week ( $RHR_{week}$ ;  $\ln rMSSD_{week}$ ) in 10 runners who responded to a 9-week training intervention. Moderate and small correlations existed between changes in MAS and 10-km running performance and  $RHR_{day}$  ( $r = 0.35$ , 90%CL (-0.35; 0.76) and  $r = -0.21$  (-0.68; 0.39)), compared with large and very-large correlations for  $RHR_{week}$  ( $r = -0.62$  (-0.87; -0.11) and  $r = 0.73$  (0.30; 0.91)). While a trivial correlation was observed for MAS vs.  $\ln rMSSD_{day}$  ( $r = -0.06$  (-0.59; 0.51)), a very-large correlation existed with  $\ln rMSSD_{week}$  ( $r = 0.72$  (0.28; 0.91)). Similarly, changes in 10-km running performance revealed a small correlation with  $\ln rMSSD_{day}$  ( $r = -0.17$  (-0.66; 0.42)), versus a very-large correlation for  $\ln rMSSD_{week}$  ( $r = -0.76$  (-0.92; -0.36)). In conclusion, the averaging of RHR and HRV values over a 1-week period appears to be a superior method for monitoring positive adaption to training compared with assessing its value on a single isolated day.

### 4.2 Introduction

Athletes, coaches and sport science practitioners seek useful methods to monitor individual adaptation or maladaptation to training. It has been shown that heart rate (HR) derived indices' such as morning resting heart rate (RHR) and heart rate variability (HRV), decrease and increase, respectively, after endurance training regimes,

and may therefore represent an easy-to-use, non-invasive means by which to assess individual adaptation to endurance training (Achten & Jeukendrup, 2003).

During the monitoring of an elite triathlete that developed signs of maladaptation to training, we recently identified a number of methodological issues associated with determining adaptation using isolated (i.e. single day) RHR and HRV readings (Plews et al., 2012). Factors such as noise, temperature, light (TaskForce, 1996) and prior exercise (Buchheit, Laursen, et al., 2009) can affect HRV on any given day. In the former article, we showed that RHR and HRV data, displayed as rolling or weekly averages, appeared to provide a more consistent representation of the changes in fatigue and maladaptation. As suggested previously, the use of isolated daily values to measure changes in HRV has likely led to the equivocal findings reported throughout the HRV training literature (Plews et al., 2012).

Given the potential of this alternative methodological approach, we re-visited the RHR and HRV data taken from recreationally-trained runners that responded positively to a 9-week training intervention (Buchheit, Chivot, et al., 2010). To assess the best practical methodological approach to analyse positive adaptation to a training intervention, we compared the relationship between isolated vs. weekly-averaged RHR and HRV values and running performance.

### 4.3 Methods

#### *Participants*

Out of the 14 runners described in the original study, (Buchheit, Chivot, et al., 2010) 10 of the positive “responders” (mass  $75.6 \pm 7.4$ ; estimated maximum aerobic speed (MAS)  $17.3 \pm 1.7$  km h<sup>-1</sup>; 10 km  $48:34 \pm 7:45$  min:s) were selected for further assessment to allow fair comparison between two different methodological approaches. Only 10 responders were included for further analysis (compared to  $n = 11$  in the original paper (Buchheit, Chivot, et al., 2010)), due to limited availability of data in the final week of the training intervention for one subject, which would make comparison between single day and weekly-averaged values invalid.

#### *Experimental protocol*

Details of the 9-week training intervention, estimated MAS, 10-km running assessment, as well as RHR/HRV recordings, have been described previously (Buchheit, Chivot, et al., 2010). Only morning RHR and HRV measures were analysed in the present study, as these have superior practicality for regular monitoring. HRV values were restricted to the natural logarithm of the square root of the mean sum of the squared differences between R-R intervals (Ln rMSSD) as Ln rMSSD has much greater reliability than other spectral indices (Al Haddad et al., 2011).

#### *Statistical analysis*

Weekly data are expressed as means  $\pm$  SD. Measures including the coefficient of variation (CV), smallest worthwhile change (SWC; 0.2 multiplied by the between-subject standard deviation) magnitude of correlation ( $r$ ), 90% confidence intervals (90%CL) and Cohen effect sizes (ES) were determined (Buchheit, Chivot, et al., 2010). Data were compared between single day (Tuesday due to consistent data points) and

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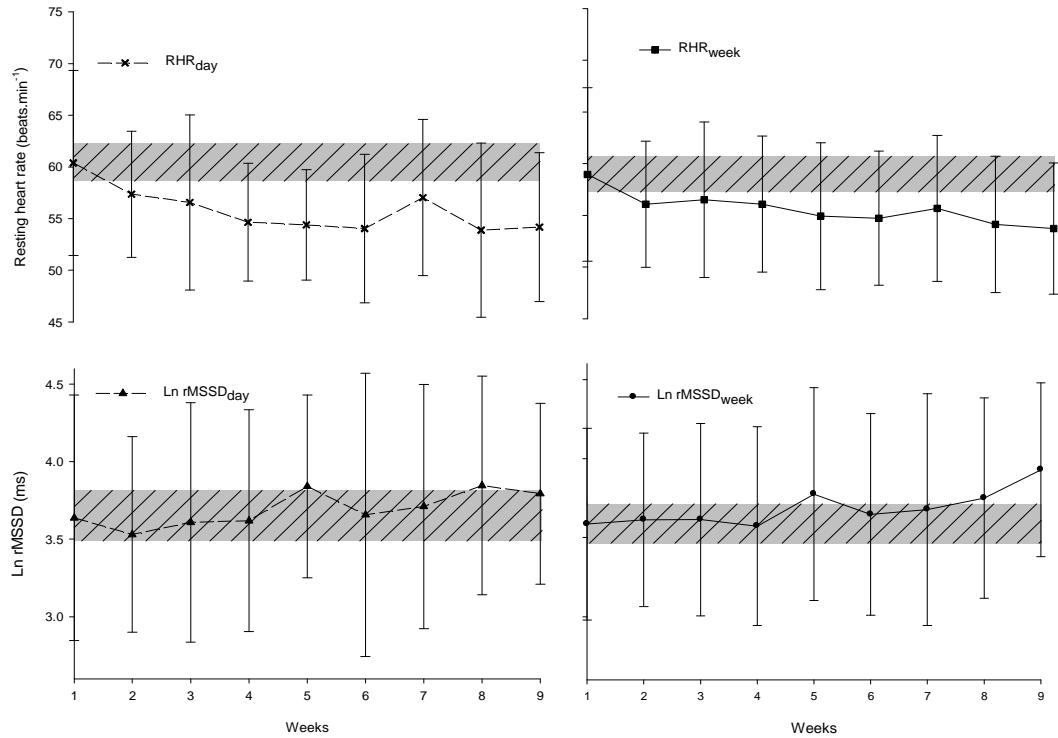
weekly-averaged (Saturday to Friday) RHR ( $RHR_{day}$ ,  $RHR_{week}$ ) and Ln rMSSD ( $Ln\ rMSSD_{day}$ ,  $Ln\ rMSSD_{week}$ ) values. Pearson's product-moment correlation analysis was used to compare the association between the relative (%) change (change from week 1 to week 9) in HR-derived indices (day and week) and performance.

### 4.4 Results

The mean CV for  $RHR_{day}$ ,  $RHR_{week}$ ,  $Ln\ rMSSD_{day}$  and  $Ln\ rMSSD_{week}$  recorded over 9 weeks was 13.0%, 12.2%, 18.7% and 16.5%, respectively.

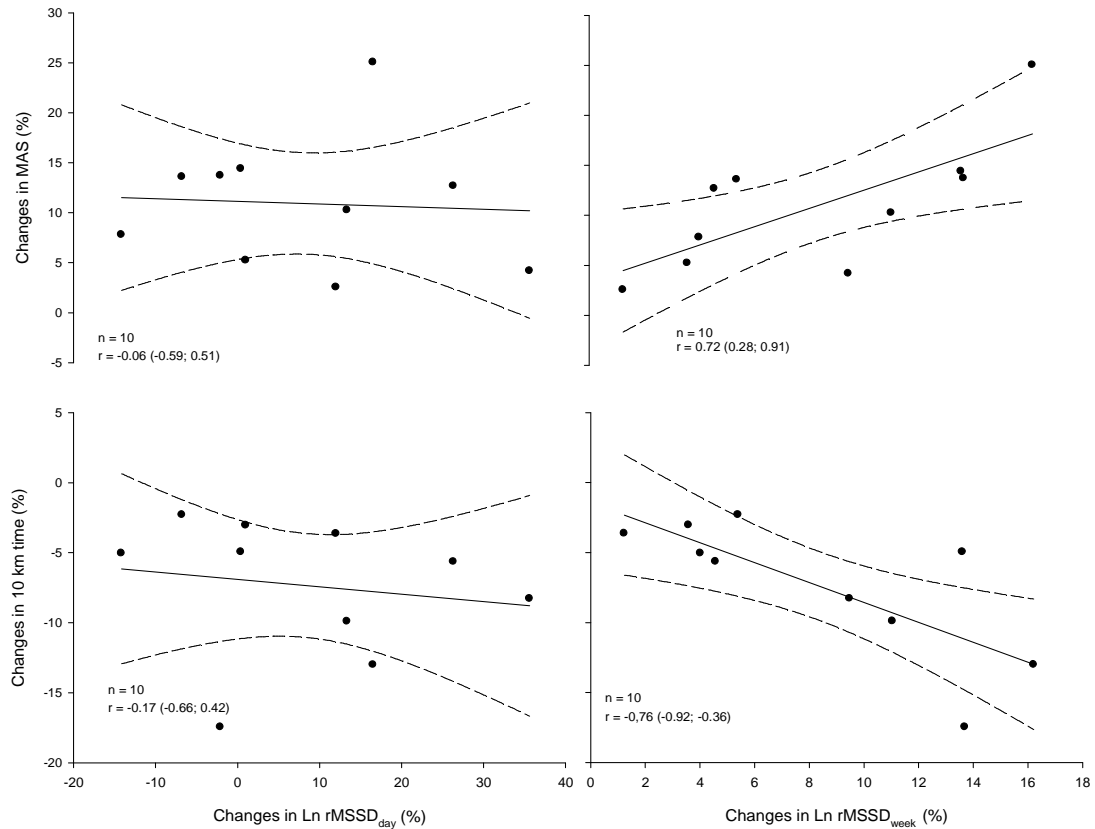
Group weekly differences in both RHR and Ln rMSSD (isolated day and weekly) relative to the SWC are shown in Figure 8. From weeks 1-9,  $RHR_{day}$  changed by  $-10.3 \pm 6.5\%$  ( $ES = -0.66$ ), compared with  $-8.9 \pm 3.2\%$  ( $ES = -0.64$ ) for  $RHR_{week}$ . Conversely,  $Ln\ rMSSD_{day}$  changed by  $4.3 \pm 10.3\%$  ( $ES = 0.20$ ) compared with  $9.6 \pm 4.8\%$  ( $ES = 0.46$ ) for  $Ln\ rMSSD_{week}$ .

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**Figure 8:** Changes in single-day and weekly-average resting heart rate (RHR<sub>week</sub> and RHR<sub>day</sub>) and natural logarithm of the square root of the mean sum of the squared differences between R-R intervals (Ln rMSSD<sub>week</sub> and Ln rMSSD<sub>day</sub>). The shaded areas represent the smallest worthwhile change (i.e. within group SD x 0.2 during the first week; see (Buchheit, Chivot, et al., 2010)). Values are group mean ± SD.

There were moderate and small correlations between % change in both MAS and 10-km running performance and RHR<sub>day</sub> ( $r = 0.35$  90%CL (-0.35; 0.76) and  $r = -0.21$  (-0.68; 0.39)). Conversely, correlations were large and very-large against RHR<sub>week</sub> ( $r = -0.62$  (-0.87; -0.11 and  $r = 0.73$  (0.30; 0.91)). The correlation between relative changes in performance (MAS and 10 km) and Ln rMSSD (isolated day and weekly data) are shown in Figure 9.



**Figure 9:** Relationship between relative changes in maximal aerobic speed (MAS, top) and 10 km running performance (bottom) and relative changes in single-day and weekly-average natural logarithm of the square root of the mean sum of the squared differences between R-R intervals ( $\text{Ln rMSSD}_{\text{day}}$  and  $\text{Ln rMSSD}_{\text{week}}$ ) with 90% confidence intervals.

#### 4.5 Discussion

As shown previously in an elite triathlete displaying signs of maladaptation, (Plews et al., 2012) the use of isolated daily HRV data points may be less accurate than using weekly averages to assess adaptation to training. In the present study, we have demonstrated that large day-to-day variation in RHR and HRV (CV=18.7 and 16.5%) can sometimes lead to a misinterpretation of the ‘true’ change due to training. For example, Figure 8 shows how  $\text{Ln rMSSD}_{\text{day}}$  returns to within the SWC on week 9 when



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athletes are at their fittest; this is not the case when Ln rMSSD values are averaged over a week. Furthermore, large and very-large correlations existed against relative changes in both MAS and 10-km running performance only when RHR and Ln rMSSD values were averaged over 1 week (Ln rMSSD represented in Figure 9). As such, we suggest that practitioners use this new averaging method of RHR and HRV analysis to acquire a more meaningful assessment of positive adaptation or maladaptation (Plews et al., 2012) to training regimes.

Measuring daily RHR is a more common and practical method of assessing cardiorespiratory fitness, and in this instance appears to have provided a closer representation of adaptation than Ln rMSSD when considered on a single day (change from week 1-9:  $ES = -0.66 \text{ RHR}_{\text{day}}$  and  $-0.64 \text{ RHR}_{\text{week}}$  vs.  $ES = 0.20 \text{ Ln rMSSD}_{\text{day}}$  and  $0.46 \text{ Ln rMSSD}_{\text{week}}$ ). This could be due to the lower day-to-day variation in RHR observed in the present ( $CV = 13.0\% \text{ RHR}_{\text{day}}$  and  $12.2\% \text{ RHR}_{\text{week}}$ ,  $CV = 18.7\% \text{ Ln rMSSD}_{\text{day}}$  and  $16.5\% \text{ Ln rMSSD}_{\text{week}}$ ) and other studies (Al Haddad et al., 2011).

### 4.6 Conclusion

Whilst we acknowledge that physiological training adaptations are the consequence of many factors (Rivera-Brown & Frontera, 2012), although longitudinal HRV data is perhaps a slightly more sensitive measure for tracking changes in fatigue and fitness when averaged over a 1-week period (Plews et al., 2012). However, RHR may provide a better indication of training state if only single-day values are available.

**CHAPTER FIVE:**  
**MONITORING TRAINING WITH**  
**HEART RATE VARIABILITY:**  
**HOW MUCH COMPLIANCE IS**  
**NEEDED FOR VALID**  
**ASSESSMENT**

## 5.1 Abstract

**Purpose:** The aim of this study was to establish the minimum number of days that HRV (i.e., the log-transformed square root of the mean sum of the squared differences between R-R intervals, Ln rMSSD) data should be averaged in order to achieve correspondingly equivalent results as data averaged over a 1-week period.

**Methods:** Standardised changes in Ln rMSSD between different phases of training (normal training, functional overreaching (FOR), overall training and taper) and the correlation coefficients of percentage changes in performance vs. changes in Ln rMSSD were compared when averaging Ln rMSSD from 1 to 7 days, randomly selected within the week. **Results:** Standardised Ln rMSSD changes (90% confidence limits, CL) from baseline to overload (FOR) were  $0.20 \pm 0.28$ ;  $0.33 \pm 0.26$ ;  $0.49 \pm 0.33$ ;  $0.48 \pm 0.28$ ;  $0.47 \pm 0.26$ ;  $0.45 \pm 0.26$  and  $0.43 \pm 0.29$  using from 1 to 7 days, respectively. Correlations (90% confidence limits (CL)) over the same time sequence and training phase were:  $-0.02 \pm 0.23$ ;  $-0.07 \pm 0.23$ ;  $-0.17 \pm 0.22$ ;  $-0.25 \pm 0.22$ ;  $-0.26 \pm 0.22$ ;  $-0.28 \pm 0.21$  and  $-0.25 \pm 0.22$  from 1 to 7 days, respectively. There were almost perfect quadratic relationships between standardised changes/r values vs. the number of days Ln rMSSD was averaged ( $r^2 = 0.92$  and  $0.97$ , respectively) in trained triathletes during FOR. This indicates a plateau in the increase in standardised changes/r values magnitude after 3 and 4 respectively days in trained triathletes. **Conclusion:** This suggests that practitioners using HRV to monitor training adaptation should use a minimum of 3 (randomly selected) valid data points per week.

## 5.2 Introduction

The utility of heart rate variability (HRV) assessment to indirectly detect autonomic nervous system (ANS) status in response to training in athletes continues to grow in popularity (Plews, Laursen, Stanley, et al., 2013). Indeed, changes in HRV have been shown to be associated with both positive (Buchheit, Chivot, et al., 2010; Hedelin et al., 2001; Manzi et al., 2009; Pichot et al., 2002) and negative adaptations to training (Hedelin, Kentta, et al., 2000; Hedelin, Wiklund, et al., 2000; Plews et al., 2012; Uusitalo et al., 1998a; Uusitalo et al., 2000). Nevertheless, there remains conjecture within the literature, as both increases (Lee et al., 2003; Buchheit, Simpson, et al., 2011) and decreases (Iellamo et al., 2002; Manzi et al., 2009) in cardiac parasympathetic indexes of HRV have shown associations with increases in fitness or performance. In addition, decreases (Hynynen et al., 2006; Uusitalo et al., 2000), increases (Hedelin, Wiklund, et al., 2000) and no change (Bosquet et al., 2003; Hedelin, Wiklund, et al., 2000; Uusitalo et al., 1998a) in cardiac parasympathetic indices of HRV have been found during periods of maladaptation (e.g. non-functional overreaching or overtraining). The disparate findings within the literature suggest that methods by which HRV can be used to track such changes (i.e. positive or negative adaptation) to endurance training are yet to be established.

We have recently suggested that the equivocal findings within the HRV literature may be due to methodological inaccuracies that occur due to the large day-to-day variation in HRV recordings.(Al Haddad et al., 2011) Indeed, environmental factors influencing measurement noise and training-related acute changes in homeostasis can influence HRV values (TaskForce, 1996). As such, when a single data point is used for analysis, the measurement noise may be exacerbated (Plews et al., 2012; Plews, Laursen, Kilding, & Buchheit, 2013). We have shown that HRV values averaged over 7

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days provides superior methodological validity for assessing both positive adaptation (Plews, Laursen, Kilding, et al., 2013) and maladaptation (Plews et al., 2012) to training. More recently, Le Meur et al. (2013) found that substantial changes in cardiac parasympathetic indexes of HRV were only detected once values were averaged over 7 days in functionally overreached triathletes.

While data averaged over 7 days appears to offer superior methodological assessment of HRV values compared with isolated measures, the minimum number of days needed to reveal correspondingly equivalent results has not yet been established. Such data would be practically useful for practitioners' intent on using HRV to monitor training in athletes, where everyday compliance is sometimes an issue. For example, only 14 out of 40 athletes in the study by Buchheit et al.(2010) managed to collect enough morning resting HRV samples to be deemed acceptable to merit study inclusion. Therefore, we re-visited known data sets that have shown weekly-averaged HRV values as being superior methods of assessment (Buchheit, Chivot, et al., 2010; Le Meur et al., 2013; Plews, Laursen, Kilding, et al., 2013), and randomly selected data points over the week from 1- to 7-days, in order to determine the minimum number of days needed to obtain a valid assessment.

### 5.3 Methods

#### *Previously published work*

Two previously published data sets were used for the present work (Buchheit et al. 2010; Le Meur et al. 2013). In one study, 13 functionally overreached and 7 control triathletes were assessed during a 5-week training intervention.(Le Meur et al., 2013) In the other study, Buchheit et al.(2010) investigated HRV responses in recreational runners during a 9-week training intervention. For further detail on these studies, the reader is referred to these papers. For clarification herein, reference to *trained triathletes*

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refers to the study performed by Le Meur et al., (2013) and reference to *recreational runners* refers to the study published by Buchheit et al. (2010).

### *Participants and training interventions*

The triathletes consisted of 20 participants (age  $32 \pm 8$  years,  $\text{VO}_{2\text{max}}$   $62 \pm 3$   $\text{mLO}_2\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$  and estimated maximum aerobic speed (MAS)  $18.2 \pm 1.1$   $\text{km}\cdot\text{h}^{-1}$ ). All had been competing in triathlon for 2 years, and had an average best Olympic distance triathlon performance time of  $128 \pm 5$  min. Participants were assigned to either an intensified training ( $n = 13$ ) or control group ( $n = 7$ ). The trained triathletes underwent a 5-week training intervention consisting of 1 week of a baseline phase (50% of their normal training load), 3 weeks of overload training (40% increase in training load), followed by a 1-week taper (same as baseline training).

From the 14 recreational runners described in the original study, (Buchheit, Chivot, et al., 2010) 10 “responders” (mass,  $75.6 \pm 7.4$ ; MAS,  $17.3 \pm 1.7$   $\text{km}\cdot\text{h}^{-1}$ ; 10 km time,  $48:34 \pm 7:45$  min:s) were selected to assess positive adaptation to training, as per our previously published work (Plews et al. 2013a). The recreational runners underwent a 9-week structured training programme. This involved 8 weeks of structured continuous and interval-based runs, followed by a 1-week taper, which preceded the 10-km and MAS performance tests.

### *Performance tests*

*Triathletes:* At the end of each training phase (baseline, overload week 1, overload week 2, overload week 3 and taper) triathletes performed a performance test. Total running distance covered during an incremental running test to volitional

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exhaustion (starting at  $11 \text{ km}\cdot\text{h}^{-1}$  for 3 min, and increasing speed by  $1 \text{ km}\cdot\text{h}^{-1}$  every 3 min thereafter) was assessed on a 340-m running track.

*Recreational runners:* A 10-km running time trial was performed twice; once at the beginning (week 1) and once at the end (week 9) of the 9-week training period. This was measured over 3.3-km laps on an outdoor course. Participants were asked to cover the 10-km distance as fast as possible. MAS was estimated via a field test (PROFILD 3-3 test). Subjects ran for 3 min at  $8 \text{ km}\cdot\text{h}^{-1}$  for the first stage, with speed increasing by  $1.5 \text{ km}\cdot\text{h}^{-1}$  every 3 min thereafter; each stage was separated by 3 min of passive recovery.

### *HRV responses to various training phases*

The main variables of interest involved changes in morning resting HRV and relationships with HRV and performance, which were randomly selected and averaged over 1- to 7-days, with specific phases of training (normal training, functional overreaching, overall training and taper). While daily HRV monitoring is generally intended by the majority of athletes, some days of the week are inevitably missed at random. The HRV of 13 trained triathletes who became functionally overreached during a 5-week training intervention were assessed from “baseline” to week 3 (final week) of the “overload” period and from week 3 of “overload” to the taper week. Comparisons were made for HRV measurements taken on an isolated day and subsequently averaged over 2 days, 3 days, 4 days, 5 days, 6 days and 7 days. Days were picked (from 1 to 7) at random using functions in Microsoft excel. As there were no substantial changes in HRV values in the control group, (Le Meur et al., 2013) only the overload group was examined for changes (i.e. standardised changes only) in HRV. However, correlations with performance were assessed in the control training group. For the recreational

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runners, changes in HRV were compared from week 1 to week 9, and the relationships with performance (MAS and 10-km) and HRV were also assessed.

### *HRV recordings*

In alignment with our previous work,(Plews et al., 2012; Plews, Laursen, Kilding, et al., 2013) and because supine morning resting HRV has superior practical application, only supine morning resting HRV was examined in the present study. For this, HRV values were taken from the final 4 min of the 8-min morning sample in the triathletes (using MemoryBelt; Suunto OY®, Vantaa, Finland) and the final 5 min of the 8-min morning measure in the recreational runners (using Polar RS800cx, Polar Electro, Kempe, Finland). In both instances, occasional ectopic beats were automatically replaced with the interpolated adjacent R-R interval values. HRV analysis was limited to the square root of the mean sum of the squared differences between R-R intervals (rMMSD), (TaskForce, 1996) as this reflects vagal activity and has much greater reliability than other spectral indices,(Al Haddad et al., 2011) particularly during ‘free-running’ ambulatory conditions.(Penttila et al., 2001b)

### *Statistics*

Data are presented as means and 90% confidence limits (CL) and intervals (CI). All rMSSD data was log-transformed prior to analysis to reduce bias arising from non-uniformity of error. To assess change in Ln rMSSD from baseline to overload, overload to taper (Le Meur et al., 2013) and week 1 to week 9 (Buchheit, Chivot, et al., 2010), a qualitative approach was used to assess the magnitude of effect, which is more relevant to training prescription and the practical detection of change (Hopkins, 2006). This was performed using a modified statistical spreadsheet (Hopkins, 2006), which calculates the standardised changes or effect sizes (standardised changes, 90% CL (Hopkins,



2006)). Hopkins threshold values for standardised changes (std changes) were  $\leq 0.2$  (trivial),  $>0.2$  (small),  $>0.6$  (moderate),  $>1.2$  (large) and  $>2.0$  (very large). Quantitative chances of either higher or lower Ln rMSSD values after a training period (e.g. more or less than a std change of 0.2) were also evaluated as follows: 25-75% possibly, 75-95% likely, 95-99% very likely,  $>99\%$  almost certain. If the chance of higher or lower differences was  $>5\%$ , then the true difference was assessed as unclear.

Pearson's correlation was used to establish the relationship between changes in performance and Ln rMSSD (during functional overreaching, normal training (training triathletes) and overall training (recreational runners)). As there were a number of performance measures for the triathletes (5 in total), we used within-individual linear modelling. Correlations were made between the delta from the individual mean (% change) for performance and Ln rMSSD (for relationships during functional overreaching and normal training). This was carried out for baseline and the three overload weeks only (excluding the taper), for both the overload and control groups. For positive adaptations in the recreational runners, correlations were made between the percentage change from week 1 to 9 for 10-km and MAS running performance and Ln rMSSD. For consistency, similar correlations techniques were used to assess positive adaption in the triathletes (percentage change from overload to taper for performance and Ln rMSSD). The magnitude of correlation ( $r$  (90% CI)) between Ln rMSSD and performance was assessed with the following thresholds:  $<0.1$ , trivial;  $<0.1-0.3$ , small;  $<0.3-0.5$ , moderate;  $<0.5-0.7$ , large;  $<0.7-0.9$ , very large; and  $<0.9-1.0$ , almost perfect. If 90% CI overlapped small positive and negative values, the magnitude of correlation was deemed 'unclear' (Hopkins et al., 2009).

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To assess the relationship between effect size or correlation and the number of days Ln rMSSD was averaged (1- to 7-days), quadratic relationships ( $r^2$ ) were also assessed. The point of plateau was determined by the day number at which standardised changes, qualitative changes and the magnitude of the correlation ( $r$ ) no longer changed with an increasing number of daily averaged values.

### 5.4 Results

For the trained triathletes, there was no substantial change in performance over the same time period in the control group (std change = 0.10, 90% confidence limits (CL)  $\pm 0.24$  and  $-0.13 \pm 0.24$  for overload and taper period, respectively). Conversely, there was an *almost certain* moderate decrease in running performance (std change =  $-0.62$ , 90% CL  $\pm 0.15$ ) during the overload period, which was subsequently followed by an *almost certain* large increase after the taper (std change =  $1.17 \pm 0.22$ ). Accordingly, for the recreational runners, there was a *very likely* small improvement in 10-km running performance (std change =  $-0.40 \pm 0.17$ ); and a *most likely* large increase in MAS (std change =  $0.75 \pm 0.15$ ).

#### *Overall population averages and coefficient variations*

The average Ln rMSSD values (90% CL) for all triathletes during baseline, overload and taper were  $4.2 \pm 0.2$  ms,  $4.3 \pm 0.2$  ms and  $4.3 \pm 0.2$  ms. The average individual coefficient variation (CV) for all recorded Ln rMSSD data for the triathletes during baseline, overload and taper was as follows: baseline =  $6.7 \pm 2.9\%$ ; overload =  $1.3 \pm 0.4\%$  and taper =  $5.8 \pm 2.0\%$ . The average Ln rMSSD values (90% CL) for all recreational runners for weeks 1 and 9 were  $3.4$  ms  $\pm 0.4$  and  $3.9$  ms  $\pm 0.3$  respectively. The average individual CV for all the recreational runners for the Ln rMSSD values recorded during weeks 1 and 9 were  $10.1 \pm 3.4\%$  and  $7.0 \pm 2.3\%$  respectively.

### *Changes in Ln rMSSD*

The standardized changes for Ln rMSSD values averaged from 1- to 7-days during functional overreaching and tapering in the triathletes and for overall training in the recreational runners are shown in Table 7 and Figure 10. The  $r^2$  values for the quadratic relationships between standardised changes and number of days Ln rMSSD was averaged is also included in Figure 10. Standardised change values plateaued after 3 days during functional overreaching in triathletes (remained *likely* small) and after 5 days in the recreational runners (remained *almost certainly* small).

### *Correlation coefficients*

The correlation coefficients for 1- to 7-day averaged Ln rMSSD values vs. performance for the triathletes during normal training (control group), functional overreaching and taper as well as the recreational runners (overall training) can be seen in Table 8 and Figure 11. There was a large quadratic relationship ( $r^2 = 0.52$ ) between r values and number of days Ln rMSSD was averaged from overload to taper in the trained triathletes. The rest of the quadratic relationships for normal training, functional overreaching and overall training (MAS only) in the recreational runners are presented in Figure 11.

Accordingly, the magnitude of the correlations plateaued after 4 (remained small) and 5 days (remained large) during functional overreaching and tapering in the trained triathletes. In the recreational runners, correlations remained ‘very large’ after 5 days for MAS. Conversely, correlations between 10-km performance and Ln rMSSD did not plateau after 7 days (e.g. 6 day = large, and 7 days = very large).

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Number of daily recordings used for analysis	Triathletes (Functional Overreaching)			Triathletes (Tapering)			Recreational Runners (Overall Training)		
	Std change in Ln rMSSD ( $\pm 90\%$ CL)	% Chances (+/trivial/-)	Qualitative	Std change in Ln rMSSD	% Chances (+/trivial/-)	Qualitative	Std change in Ln rMSSD ( $90\%$ CL)	% Chances (+/trivial/-)	Qualitative
1	0.20 $\pm$ 0.28	49/50/1	Unclear	-0.15 $\pm$ 0.34	5/50/39	Unclear	0.24 $\pm$ 0.31	60/28/1	Unclear
2	0.33 $\pm$ 0.26	80/20/0	Likely small	-0.01 $\pm$ 0.20	5/90/6	Unclear	0.35 $\pm$ 0.28	82/17/0	Likely small
3	0.49 $\pm$ 0.33	93/7/0	Likely small	-0.12 $\pm$ 0.22	1/72/26	Unclear	0.63 $\pm$ 0.34	98/2/0	Very likely large
4	0.48 $\pm$ 0.28	95/5/0	Likely small	-0.11 $\pm$ 0.19	1/79/20	Unclear	0.39 $\pm$ 0.25	90/10/0	Likely small
5	0.47 $\pm$ 0.26	96/4/0	Very likely small	-0.08 $\pm$ 0.17	1/87/12	Unclear	0.46 $\pm$ 0.14	100/0/0	Most likely small
6	0.45 $\pm$ 0.26	93/7/0	Likely small	-0.08 $\pm$ 0.17	1/80/13	Unclear	0.47 $\pm$ 0.11	100/0/0	Most likely small
7	0.43 $\pm$ 0.29	91/9/0	Likely small	-0.09 $\pm$ 0.19	1/89/15	Unclear	0.46 $\pm$ 0.13	100/0/0	Most likely small

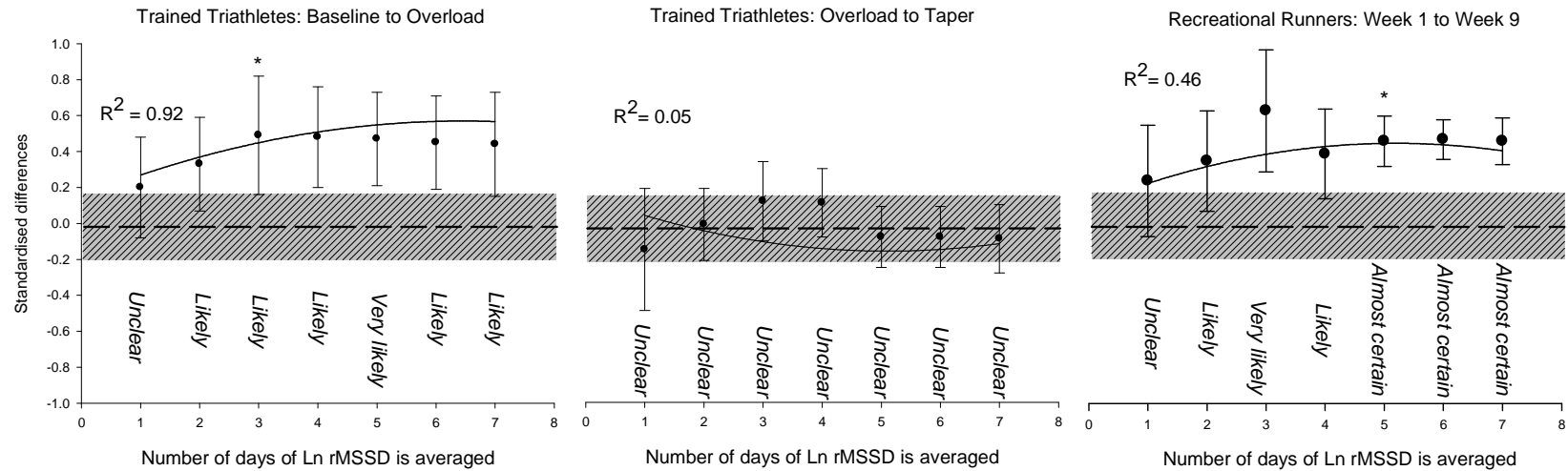
**Table 7:** Standardised changes (Std change) and  $\pm 90\%$  confidence limits (CL) with qualitative chances of change for Ln rMSSD values averaged over 1 to 7 days. Standardised changes are compared from baseline to overload week 3 (functional overreaching) and overload week 3 to the week of the taper (tapering) in the triathletes. Accordingly, standardised changes were compared from week 1 to week 9 in the recreations runners (positive adaptation to overall training) with the same averaged Ln rMSSD values.

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Ln rMSSD Averaged Value	Trained triathletes (Normal Training)			Trained triathletes (Functional Overreaching)			Trained triathletes (Taper)			Recreational Runners (Overall Training – M AS)		
	Slope ( per % change; ± 90% CL)	r (± 90% CL)	Magnitud e	Slope ( per % change; ± 90% CL)	r (± 90% CL)	Magnitud e	Slope ( per % change; ± 90% CL)	r (± 90% CL)	Magnitude	Slope ( per % change; ± 90% CI)	r (± 90% CL)	Magnitude
1 day	-0.04 ±0.15	0.02 ±0.28	Unclear	-0.01 ±0.14	-0.02 ±0.23	Unclear	0.40 ±0.57	0.42 ±0.41	Moderat e	0.26 ±0.43	0.39 ±0.49	Unclear
2 days	-0.03 ±0.19	0.01 ±0.28	Unclear	-0.04 ±0.13	-0.07 ±0.23	Unclear	0.90 ±0.92	0.57 ±0.35	Large	0.39 ±0.42	0.53 ±0.43	Large
3 days	-0.03 ±0.23	0.02 ±0.28	Unclear	-0.12 ±0.16	-0.17 ±0.22	Unclear	0.54 ±0.65	0.38 ±0.42	Moderat e	0.36 ±0.23	0.73 ±0.31	Very Large
4 days	-0.01 ±0.28	0.01 ±0.28	Unclear	-0.25 ±0.12	-0.25 ±0.22	Small	0.78 ±0.77	0.46 ±0.40	Moderat e	0.35 ±0.38	0.64 ±0.37	Large
5 days	-0.02 ±0.34	0.02 ±0.28	Unclear	-0.28 ±0.06	-0.26 ±0.22	Small	0.96 ±0.79	0.52 ±0.37	Large	0.29 ±0.33	0.51 ±0.44	Large
6 days	0.00 ±0.34	0.01 ±0.28	Unclear	-0.33 ±0.07	-0.28 ±0.21	Small	0.96 ±0.82	0.51 ±0.39	Large	1.21 ±0.75	0.76 ±0.28	Very Large
7 days	0.00 ±0.40	0.02 ±0.28	Unclear	-0.28 ±0.06	-0.25 ±0.22	Small	1.10 ±0.70	0.65 ±0.31	Large	1.02 ±0.65	0.72 ±0.32	Very Large

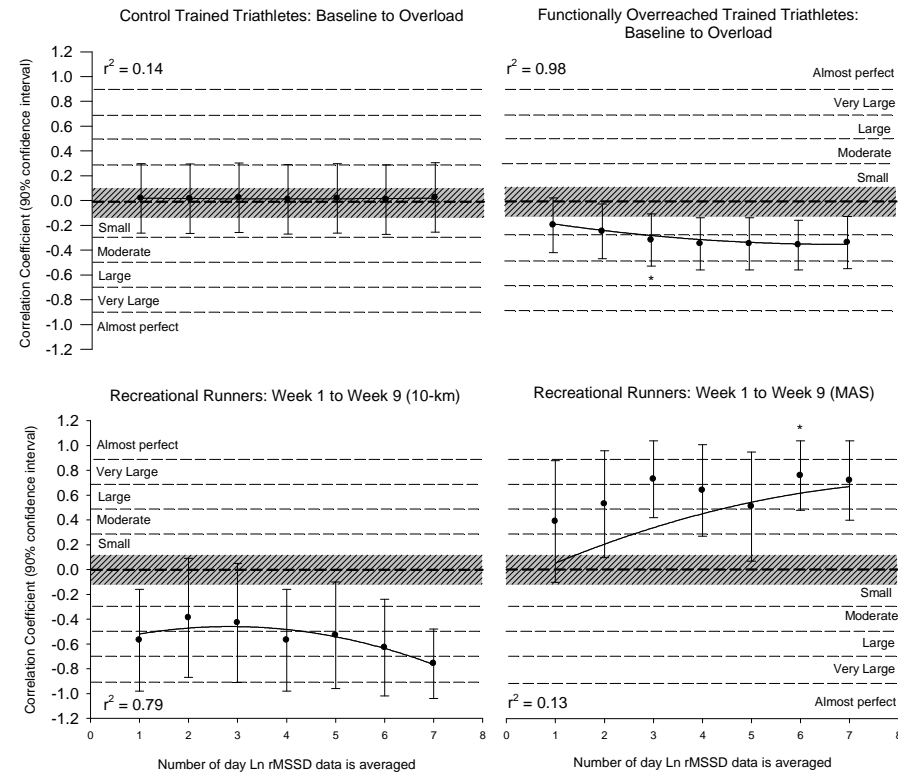
**Table 8:** Slope (per % change in Ln rMSSD; ±90% confidence limits), correlation coefficient (±90% confidence limits) and magnitude of correlation subsequent for performance (total distance completed) vs. Ln rMSSD for values averaged over 1- to 7-days in both triathletes (normal training (control group), functional overreaching and taper) and recreational runners (overall training; to maximum aerobic speed (MAS)). Correlations were made as delta from the individual mean (% change) for the triathletes and as percentage change from week 1 to 9 for the recreational runners.

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**Figure 10:** Standardised changes in Ln rMSSD after the same training period for Ln rMSSD values averaged over 1 to 7 days. Error bars indicate 90% confidence intervals. When 90% confidence intervals overlapped the zero line (black dashed line), changes were deemed ‘unclear’. The  $r^2$  value for the quadratic relationship is included in the top left hand corner of each graph. The shaded area represents a trivial effect size (see “Methods”). Quantitative chances of change are quantified qualitatively at the bottom of each chart (see “Methods”). \* indicates the point at which standardised changes values stabilize (i.e. the standardised changes and qualitative chances no longer alter with the increasing number of days Ln rMSSD is averaged).

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**Figure 11:** Correlation coefficients with 90% confidence intervals for the % change performance and % change Ln rMSSD values averaged over 1- to 7-days for triathletes during normal training (control) and functional overreaching and recreational runners (10km running performance and maximum aerobic speed). The grey shaded area represents a trivial correlation. When 90% confidence interval overlapped the zero line (black dashed line), correlations are deemed unclear. The  $r^2$  value for the quadratic relationship is included in the bottom left-hand corner of each chart. \* indicates the point at which correlation coefficient values stabilize (i.e. the magnitude of the correlation no longer changes with the increasing number of days Ln rMSSD is averaged).

### 5.5 Discussion

To assess substantial changes in training status, we have previously shown that HRV values averaged over a 1-week period provide superior methodological validity than an isolated HRV measurement taken on a single day (Plews et al., 2012; Plews, Laursen, Kilding, et al., 2013). The primary aim of this study was to establish the minimum number of days HRV must be averaged (1 to 7 days) in order to elicit similar responses to that of data averaged over a week. This is an important practical consideration for sport practitioners and researchers intent on using HRV to monitor training, and who find difficulty obtaining athlete compliance every day of their assessment period. The main finding of this study was that, in trained athletes, practitioners must have a minimum of 3 HRV recordings per week in order to get a reasonable assessment of training status. In lesser trained individuals (e.g. recreational), the minimum number of days needed is likely to be greater (~ 5 days) due to the observed greater day-to-day variations in HRV (e.g. CV (90% CL) for recreational runners =  $10.1 \pm 3.4\%$  for week 1; CV for triathletes =  $6.8 \pm 2.9\%$  for baseline).

Although not within the main aims of this study, we must first acknowledge the power of HRV as tool to monitor training (Buchheit, Chivot, et al., 2010; Pichot et al., 2000; Plews, Laursen, Stanley, et al., 2013; Portier et al., 2001). Indeed, we observed *likely to almost certain* small, standardised changes in Ln rMSSD with different phases of training. There were also some small, moderate and large correlations between Ln rMSSD and performance. Furthermore, when athletes did not perform a training load sufficient enough to elicit adaptation and changes in performance, there was no substantial change in HRV, irrespective of the number of days HRV was averaged (Table 8).



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When we examined the standardised changes for HRV values averaged from 1 to 7 days, we found that the increased HRV associated with functional overreaching in the trained triathletes was consistent after 3 days of averaging (e.g. after 3 days, the increase in Ln rMSSD remained *likely* small with functional overreaching). Although chances were also *very likely* small after 2 days, percentage chances increased by 13% from 2 days to 3 days of averaging, remaining stable thereafter (e.g. average percentage chances of small standardised changes from 3-7 days =  $93.6\% \pm 2.0$ ; Table 7). Positive adaptation during the 9-week training intervention in the recreational runners was identified after 5 days of averaging (i.e., increases remained *most likely* small after 5 days of averaging; Figure 10).

For the correlation data, stabilization in the  $r$  values occurred after 4 days in the functionally-overreached triathletes (the magnitude of the correlation remained small), but not until 6 days (i.e. correlations became, *very large* (clear) for 6 and 7 days) in the recreational runners for MAS. However, the magnitudes of the correlation for 10 km run performance were not stabilized until after 7 days. These data suggest that recreational athletes would need to record more daily HRV values within a week than trained athletes, and that the minimum of days required might also depend on the performance tests considered. The lower Ln rMSSD day-to-day variations in the triathletes may be due to higher Ln rMSSD values due to their extensive training backgrounds (e.g. recreational runners Ln rMSSD at week 1 =  $3.4 \pm 0.4$  ms; trained triathletes at baseline =  $4.2 \pm 0.2$  ms). As such, the day-to day variations are less likely to be caused by ‘the law of initial values’ (Plews et al., 2012), particularly during high training loads (e.g. the triathletes CV is at its lowest during overload). Accordingly, the number of days HRV must be averaged to reduce measurement noise will be more for

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athletes with greater day-to-day variations, since the noise is reduced by a factor of  $1/\sqrt{n}$  measures (Hopkins, Schabort, & Hawley, 2001). Furthermore, as there were 5 repeated measures with the trained triathletes, compared with just one in the recreational runners, the 90% confidence limits will be smaller and the magnitude of the correlations become clearer at lower  $r$  values. Also, the modality and subsequent noise of the performance test must be reflected when considering the number of daily HRV recording required; for example in this study, the magnitude of the correlation between Ln rMSSD and MAS stabilized more readily than 10-km time in the recreational runners. However, running time trial tests have shown similar CV values to incremental MAS tests previously (Hopkins et al., 2001). Another possible explanation could be related to the greater individual responses for 10-km (between-athlete SD in the change expressed as a CV = 99%) than MAS (70%) in this study (Buchheit, Chivot, et al., 2010).

The correlation data for the trained triathletes from overload to taper periods should be viewed with caution. While there appears to be a positive correlation between performance and HRV changes in the triathletes from overload through to taper, correlation does not imply causation. Closer examination of the data reveals 8 triathletes with reductions in HRV values compared to overload, and 5 athletes with increased HRV values compared to overload (despite all improving performance). This reinforces the complexities and individualisation of HRV responses to training (Plews, Laursen, Stanley, et al., 2013), and how effect sizes are a more valid statistical assessment of change (i.e. although there was a *large* correlation, the effect size was *trivial*) (Buchheit & Rabbani, 2013).

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We also found a stronger association between the standardised changes or  $r$  values and the number of days Ln rMSSD is averaged in trained triathletes than recreational runners. This is supported by the much higher quadratic relationships in the triathletes ( $r^2 = 0.92$  (std changes) and  $0.98$  ( $r$  value functional overreaching)) compared with the recreational runners ( $r^2 = 0.46$  (std changes);  $0.13$  ( $r$  value MAS)) and  $0.79$  ( $r$  value 10 km). As such, the ability to assess a plateau in the standardised changes and correlations with the quadratic relationships is less clear in the recreational runners. Because of the greater day-to-day HRV variations in the recreational athletes, sampling variations within a reduced number of days might have a greater effect on the magnitude of the standardized changes and correlations. Since we randomly selected the days within the week to be included in the analysis, final result would be dependent on which day out of the 7 days are selected, and re-drawing the data again would likely lead to slightly different results. This is evidenced by the unexpected large and very large standardised changes/ $r$  values after just 3 days of averaging (e.g. since these values decreased at 4 days and then increased thereafter; Table 7 and 8). Finally, it must also be acknowledged that it is difficult to draw conclusive outcomes around changes in HRV through different phases of training when using different populations (trained and recreationally trained). Indeed, elites and athletes with extensive training histories are unlikely to demonstrate increasing performance with concomitant increases in HRV values (Iellamo et al., 2002; Manzi et al., 2009; Plews, Laursen, Stanley, et al., 2013), as demonstrated in these recreational runners.

### 5.6 Conclusion

Whilst the number of daily recorded HRV values is likely a balance between the minimum number required and what is practically applicable; the more consistent the daily recordings, the more confident practitioners can be in evaluating HRV changes (e.g. 90% confidence limits continued to decline until around 5 days of Ln rMSSD value averaging (Table 7)). Finally, HRV measure over a 1-week period (or that micro-cycle) will allow both effective and practically applicable evaluations of training responses, provided that the minimum number of required daily recording (3 in trained athletes) is recorded. We have previously demonstrated HRV values averaged over 1 week provide a superior representation of training-induced changes (Plews et al., 2012; Plews, Laursen, Kilding, et al., 2013) than HRV values taken on a single day. In the current study, we have shown that HRV values averaged at random over a minimum number of 3 days will allow for an equivalent representation of training adaptation than values averaged for up to 7 days in trained triathletes. Conversely, recreationally athletes will need a slightly greater number of days averaging (~5 days) due to their greater day-to-day variations in Ln rMSSD values.

**CHAPTER SIX:**  
**TRAINING ADAPTATION AND**  
**HEART RATE VARIABILITY IN**  
**ELITE ENDURANCE ATHLETES:**  
**OPENING THE DOOR TO**  
**EFFECTIVE MONITORING**

### 6.1 Abstract

The measurement of heart rate variability (HRV) is often considered a convenient non-invasive assessment tool for monitoring individual adaptation to training. Decreases and increases in vagal-derived indices of HRV have been suggested to indicate negative and positive adaptations, respectively, to endurance training regimes. However, much of the research in this area has involved recreational and well-trained athletes, with the small number of studies conducted in elite athletes revealing equivocal outcomes. For example, in elite athletes, studies have revealed both increases and decreases in HRV to be associated with negative adaptation. Additionally signs of positive adaptation, such as increases in cardiorespiratory fitness, have been observed with atypical concomitant decreases in HRV. As such, practical ways by which HRV can be used to monitor training status in elites are yet to be established. This article addresses the current literature that has assessed changes in HRV in response to training loads and the likely positive and negative adaptations shown. We reveal limitations with respect to how the measurement of HRV has been interpreted to assess positive and negative adaptation to endurance training regimes and subsequent physical performance. We offer solutions to some of the methodological issues associated with using HRV as a day-to-day monitoring tool. These include the use of appropriate averaging techniques, and the use of specific HRV indices to overcome the issue of HRV saturation in elite athletes (i.e. reductions in HRV despite decreases in resting heart rate). Finally, we provide examples in Olympic and World Champion athletes showing how these indices can be practically applied to assess training status and readiness to perform in the period leading up to a pinnacle event. The paper reveals how longitudinal HRV monitoring in elites is required to understand their unique individual

HRV fingerprint. For the first time, we demonstrate how increases and decreases in HRV relate to changes in fitness and freshness, respectively, in elite athletes.

### 6.2 Introduction

One of the more promising methods to monitor individual adaptation to training involves the regular monitoring of cardiac autonomic nervous system (ANS) status, through the measurement of resting or post-exercise heart rate variability (HRV) (Buchheit et al., 2007; Buchheit, Simpson, et al., 2011; Hautala et al., 2009). Indeed, non-functional over-reaching (NFOR) and/or negative adaptation to training is thought to be generally associated with reductions in vagal-related indices of HRV (Bosquet et al., 2008; Hynynen et al., 2006, 2008), whereas increases in fitness (Lee et al., 2003; Mourot, Bouhaddi, Tordi, et al., 2004; Vesterinen et al., 2011) and exercise performance (Atlaoui et al., 2007; Buchheit, Simpson, et al., 2011; Garet et al., 2004) are thought to be more associated with increases in vagal-related indices of HRV. While findings from studies involving recreational and well-trained athletes suggest that HRV may be a valuable tool for assessing individual adaptation to endurance training, data obtained from elites an athlete with longer training history remains equivocal (Buchheit, Al Haddad, et al., 2011; Iellamo et al., 2002; Manzi et al., 2009; Pagani & Lucini, 2009; Portier et al., 2001).

The purpose of this article is to present a brief summary of the studies where HRV has been investigated in response to adaptation and changes in training load. In doing so, we highlight the methodological issues inherent in its use and interpretation to date. We advance our current opinion of how HRV should best be monitored and assessed with examples from elite endurance athletes. All references to HRV throughout this manuscript refer to vagal-related indices of HRV (TaskForce, 1996). All HRV data

presented herein were recorded upon waking and measured as the last 5 min of the 6 min supine rest period (for more details on the methodology, including calculation of the “smallest worthwhile change”, please refer to Plews et al., (2012).

### **6.3 HRV in response to different training loads**

The influence of intensified and reduced training loads on HRV has been thoroughly studied. In moderately-trained subjects, moderate training loads increase aerobic fitness, as well as HRV (Buchheit, Chivot, et al., 2010; Iwasaki et al., 2003a; Manzi et al., 2009; Pichot et al., 2002). However, when training loads approach higher levels (100% of an individual's maximal training load), HRV indices are reduced, (Iwasaki et al., 2003a; Manzi et al., 2009; Pichot et al., 2000) and are thought to rebound after periods of reduced training (e.g. taper). (Garet et al., 2004; Pichot et al., 2002; Pichot et al., 2000) For example, after 3 weeks of overload training in swimmers and distance runners, HRV was reduced by 22% (Garet et al., 2004) and 38% (Pichot et al., 2000), respectively. Following 2 weeks of reduced training (69% reduction in training load compared with overload), HRV rebounded and increased by 7% in swimmers,(Garet et al., 2004) and after 1 week (40% reduction in training load compared with overload) increased by 38% in distance runners (Pichot et al., 2000). Like in moderately trained athletes, elites and athletes with extensive training histories also show increase and decreases in HRV after moderate and high training loads, respectively (Iellamo et al., 2002; Iwasaki et al., 2003a; Manzi et al., 2009)). Conversely, however, HRV can remain depressed in the lead up competition (e.g. tapering), despite achievement of an optimal performance (Iellamo et al., 2002; Manzi et al., 2009). In the case of these athletes, the reduction in HRV prior to competition possibly reflects the HRV response to consecutive days of high intensity training (with a reduction in training volume in the case of a taper) (Kaikkonen et al., 2008; Seiler et al., 2007) and/or HRV saturation at



low HR levels (Buchheit et al., 2004) (see section 4.1 and 4.2, respectively). With just one study examining the HRV responses of elites leading into competition (Iellamo et al., 2002), the optimal HRV response to training overload and pre-competition tapers (in elites) is yet to be fully understood.

### **6.3.1 HRV and positive adaptation to training**

The changes in HRV in response to endurance training have been extensively studied. In sedentary and recreationally-trained individuals, endurance training for 2 (Lee et al., 2003), 6 (Mourot, Bouhaddi, Perrey, Rouillon, et al., 2004; Yamamoto et al., 2001), and 9 (Buchheit, et al. 2010) weeks has been shown to induce parallel increases in aerobic fitness and HRV. For example, Buchheit et al. (2010) showed that improvements in maximal aerobic running speed and 10 km run time had moderate ( $r = 0.52$  (confidence intervals (CI) 0.08; 0.79) and large ( $r = -0.73$  (CI -0.89; -0.41)) correlations with increases in resting HRV, respectively. While this is the typical response shown in sedentary and recreationally-trained individuals following a period of endurance training (Buchheit, Chivot, et al., 2010; Lee et al., 2003; Mourot, Bouhaddi, Perrey, Rouillon, et al., 2004; Pichot et al., 2002; Yamamoto et al., 2001), the response in athletes with extensive training histories (e.g., elite athletes) can be markedly different. In these athletes, the HRV response to training is variable, with longitudinal studies showing no change in fitness (i.e., maximal oxygen uptake ( $\dot{V}O_{2\max}$ )) despite an increase in HRV (Portier et al., 2001), and other studies showing decreases in HRV despite increases in fitness (Iellamo et al., 2002). As such, there is generally a bell-shaped relationship between vagally-related HRV and fitness.

### **6.3.2 HRV and negative adaptation to training**

Overtraining is a verb used to describe the process of undergoing intensified training to induce possible overreaching. Overreaching refers to a short-term stress-regeneration imbalance that includes negative outcomes such as increased fatigue and reductions in performance (Meeusen et al., 2006). While overreaching is typically believed to be an important component of the elite athlete training cycle, prolonged overreaching can push an athlete into a state of non-functional overreaching (NFOR), which is associated with reductions in performance ability that do not resume for several weeks or months (Meeusen et al., 2013). To date however, studies that have examined changes in HRV with NFOR/OT have revealed equivocal findings, with increases (Hedelin, Wiklund, et al., 2000), decreases (Hynynen et al., 2006; Uusitalo et al., 2000) and no changes (Bosquet et al., 2003; Hedelin, Wiklund, et al., 2000; Uusitalo et al., 1998a) in HRV reported. In a case study of an elite cross-country skier that became OT, Hedelin et al. (2000) showed reduced competition performance and lowered profile of mood states, along with substantially increased HRV. Conversely, Uusitalo et al. (2000) showed that OT was associated with decreased HRV in endurance athletes undergoing heavy training over a 6-9 week period. Hedelin et al. (2000) also reported unchanged HRV in elite canoeists, despite decreased run time to fatigue and reduced  $\dot{V}O_{2max}$ . However, the inconsistent findings shown between HRV and OT/NFOR to date are likely due to the methodological approach adopted (see section 3) and difficulty with discriminating between the different stages of the OT process (e.g. overtraining, overreaching, NFOR and OT syndrome) (Meeusen et al., 2013; Halson & Jeukendrup, 2004). This is particularly evident in studies that have purposely induced overtraining, (Baumert et al., 2006; Bosquet et al., 2003; Hedelin, Kentta, et al., 2000; Uusitalo et al., 1998a) which

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unlikely reflects real-life training conditions (Halson & Jeukendrup, 2004; Meeusen et al., 2013). Finally, the possibility that two types of OT may occur in athletes (parasympathetic vs. sympathetic; (Kuipers, 1998; Kuipers & Keizer, 1988)) may further contribute to the equivocal research findings shown.

### **6.3.3 Literature summary**

It has been suggested that increases and decreases in HRV are associated with positive (Atlaoui et al., 2007; Buchheit, Simpson, et al., 2011; Garet et al., 2004; Lee et al., 2003; Mourot, Bouhaddi, Tordi, et al., 2004; Vesterinen et al., 2011) and negative (Hynynen et al., 2006, 2008; Uusitalo et al., 2000) adaptation to endurance training regimes, respectively. However, the bell-shaped relationship typically apparent between both HRV and training load (Iellamo et al., 2002; Iwasaki et al., 2003a; Manzi et al., 2009; Pichot et al., 2002), and HRV and fitness (Bosquet et al., 2007; Buchheit, Al Haddad, et al., 2011; Iellamo et al., 2002), in elites and athletes with extensive training histories, makes it difficult to practically use HRV to maximise training in these populations.

### **6.4 Methodological consideration with the assessment of HRV**

Indices of HRV display a naturally high day-to-day variation (Al Haddad et al., 2011). We have recently suggested that both environmental factors influencing measurement ‘noise’ and acute changes in homeostasis may contribute to discrepancies in interpretation when a single data point is used for analysis. When HRV is used to assess changes in both negative (Plews et al., 2012) and positive adaptation (Plews, Laursen, Kilding, et al., 2013), both weekly (Plews et al., 2012; Plews, Laursen, Kilding, et al., 2013) and 7-day rolling (Plews et al., 2012) averages have been shown to provide better methodological validity compared with values taken on a single day. For example, when HRV data points were averaged over 1 week, a meaningful

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representation of training status was apparent in a NFOR elite athlete (e.g. worthwhile reductions in weekly-averaged HRV were observed only during the period of NFOR) (Plews et al., 2012). Comparatively, when single day values were used for analysis, the HRV data were misleading (i.e., worthwhile reductions in HRV indicative of NFOR occurred when the athlete was training and performing effectively). Conversely, when percentage changes in 10-km running performance were correlated with percentage changes in HRV, very-large relationships were observed when HRV values were averaged over one week ( $r = -0.76$  (CI -0.92; -0.36)) but not when using single day values ( $r = -0.17$  (CI -0.66; 0.42)) (Plews, Laursen, Kilding, et al., 2013). This suggests that averaged morning resting HRV data provide a more consistent representation of actual changes in an athlete's autonomic balance with training compared to a single isolated value. Most recently, morning resting HRV was shown to deviate little, irrespective of the prior day/s training, when positive adaptations to training occur in well-trained individuals (Stanley, Peake, & Buchheit, 2012).

Another methodological issue apparent within the literature is the variety of HRV indices that have been used to assess autonomic balance (TaskForce, 1996). It has been shown that time domain indices of HRV have a lower typical error of measurement (when expressed as a coefficient variation (CV)) than other spectral indices of HRV (e.g. the natural logarithm of the square root of the mean sum of the squared differences between R-R intervals (Ln rMSSD), CV = 12.3%; normalised high frequency power (HFnu), CV = 52.0%) (Al Haddad et al., 2011). We suggest that practitioners and researchers using HRV measurements to evaluate training adaptations choose just one vagally-derived HRV variable for assessment. We prefer Ln rMSSD, as it is the most practically applicable HRV index for a number of reasons. First, Ln rMSSD is not significantly influenced by breathing frequency, unlike other spectral indices of HRV,

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and is therefore more suited to ambulatory measures (Penttila et al., 2001b). Second, Ln rMSSD can capture levels of parasympathetic activity over a short time frame, which is more convenient for athletes that have limited time to acquire a reading (Hamilton, McKechnie, & Macfarlane, 2004). Last, Ln rMSSD values can be easily calculated in MS Excel using R-R intervals (Aubert et al., 2003). In our opinion therefore, the equivocal findings apparent throughout the HRV literature are likely due to the large day-to-day variation in HRV and the variety of HRV indices used for analysis that are more prone to errors.

### **6.5 The relevance of HRV in elite athletes**

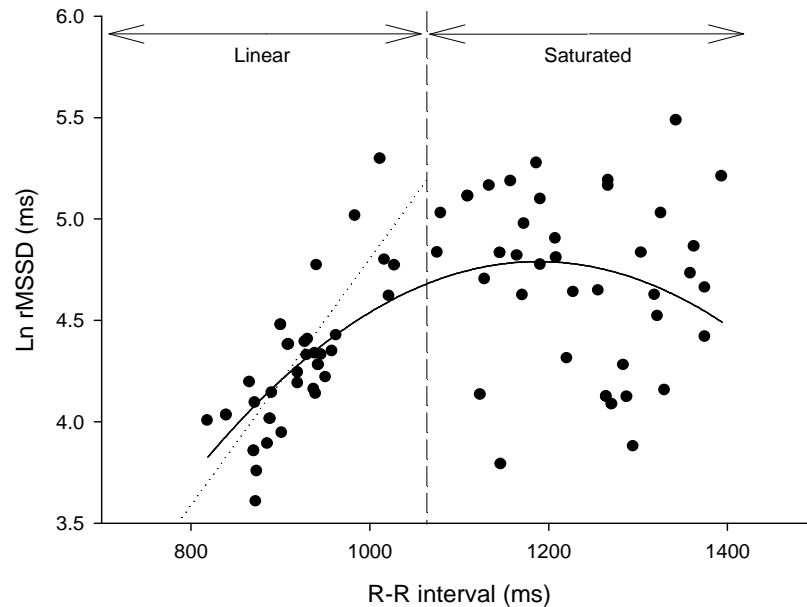
Training programs of elite athletes typically consist of periods of high training loads with limited periods of rest and recovery (Fiskerstrand & Seiler, 2004; Laursen, 2010). Such athletes are always pushing the boundary between functional and NFOR in an attempt to gain the greatest possible fitness level. Despite this, published data in elite athletes is rare, with most HRV research to date involving recreational/well-trained subjects (Buchheit, Chivot, et al., 2010; Lee et al., 2003; Manzi et al., 2009; Mourot, Bouhaddi, Perrey, Rouillon, et al., 2004; Pichot et al., 2002; Yamamoto et al., 2001). Due to genetics and training history (Tucker & Collins, 2012), elite athletes may respond differently to training stresses and subsequent recovery (Barnett, 2006). In the following sections, we describe some of the different HRV profiles of elite athletes we have observed, and how these fluctuations may be reflective of training adaptation and the ability to perform at peak levels.

### 6.5.1 HRV profiles in elite athletes

A common misconception made by sports practitioners using HRV to assess ANS status is that there is a direct linear relationship between vagal-related indices of HRV and the parasympathetic influence on heart rate (HR). In reality, however, the relationship is quadratic (Goldberger et al., 1994; Goldberger et al., 2001) (see example in Figure 12). This means that at both low (high HR) and high (low HR) levels of vagal tone, vagal-related HRV indices are reduced. For instance, while well-trained athletes generally present both a low resting HR and increased HRV indices, a reduced HRV has also been observed in many athletes with a low resting HR (Kiviniemi et al., 2004). This reduction of HRV at low HR is related to the fact that vagal-related HRV indexes more reflect the magnitude of modulation in parasympathetic outflow as opposed to an overall parasympathetic tone *per se* (Hedman, Hartikainen, Tahvanainen, & Hakumaki, 1995). The underlying mechanism is likely the saturation of acetylcholine receptors at the myocyte level: a heightened vagal tone may give rise to sustained parasympathetic control of the sinus node, which may eliminate respiratory heart modulation and reduce HRV (Malik et al., 1993). This is an important consideration for practitioners using HRV to assess training status in elites, who typically have a low resting HR, undergo high training loads and are therefore prone to saturation (Buchheit et al., 2004; Sacknoff, Gleim, Stachenfeld, Glace, & Coplan, 1992). For example, during different phases/loads of training, reductions in HRV can occur, “theoretically” indicating ANS stress/NFOR (Borresen & Lambert, 2008; Bosquet et al., 2008). However, this trend should only be interpreted in light of the respective changes in resting HR, to assess whether this decrease can be the result of the saturation phenomenon or not. This can be achieved by using the Ln rMSSD to R-R interval ratio (Plews et al., 2012), which

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simultaneously considers changes in both vagal tone (R-R interval) and vagal modulation (HRV) (Buchheit et al., 2007).



**Figure 12:** Example of the relationship between the R-R interval and the natural logarithm of the square root of the mean sum of the squared differences between R-R intervals (Ln rMSSD) in a subject with increasing bradycardia. Here, a saturation of heart rate variability is seen with long R-R intervals. Note how at shorter R-R intervals there is a linear relationship between Ln rMSSD (dotted line), which becomes disassociated as the duration of the R-R interval increases, indicating heart rate variability saturation.

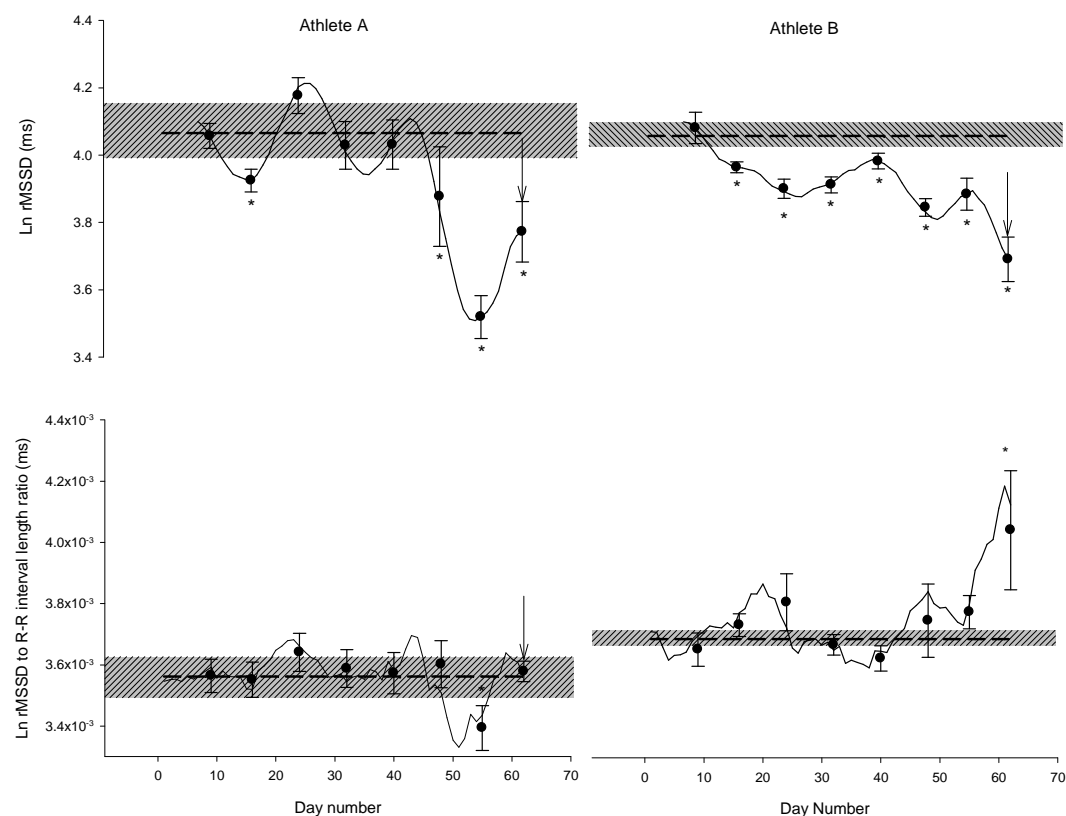
Figure 13 shows two athletes competing at the same international rowing world cup event (Lucerne FISA World Cup 2012), both with suppressed HRV values before the race. However in the case of Athlete A who performed optimally (2<sup>nd</sup> place in his event, 0.12% behind the winner), the reduction in Ln rMSSD (falling below the smallest worthwhile change (SWC); see (Hopkins et al., 2009; Plews et al., 2012)) in the lead up to the race was a result of HRV saturation (as demonstrated by the Ln rMSSD to R-R

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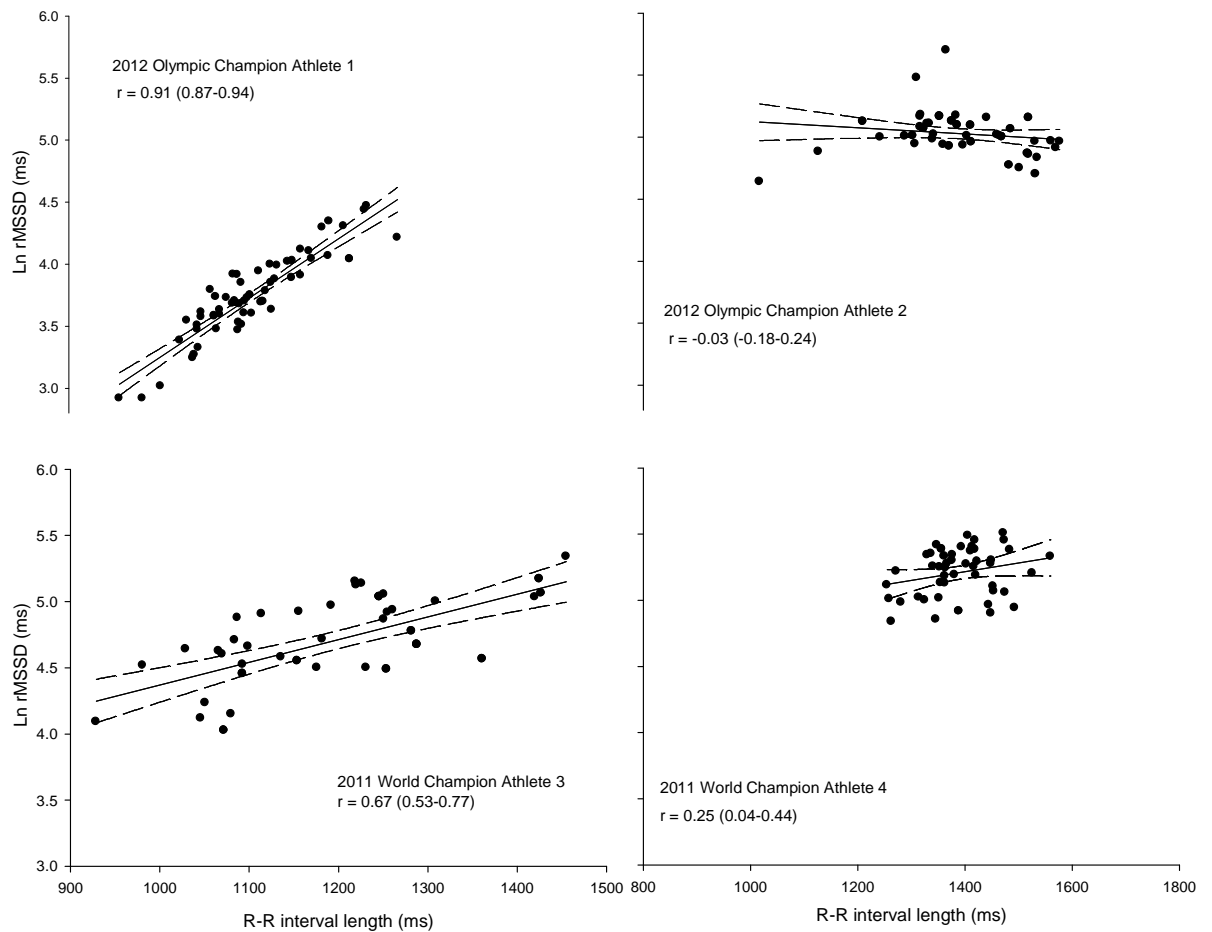
interval ratio falling below the SWC) and unlikely fatigue (Borresen & Lambert, 2008), However, Athlete B (performing poorly; 5th place in her event; 1.92% behind the leader despite being a 2011 world championship medallist) incurred reductions in Ln rMSSD and increases in the Ln rMSSD to R-R interval ratio, suggesting both a loss in vagal tone and modulation. This was likely due to poor adaptation to her training load (NFOR) and sympathetic over-activity.

Recently, we have also shown changes in the relationship between Ln rMSSD and the R-R interval during effective training and NFOR in an elite female triathlete (Plews et al., 2012). In this instance, the athlete was saturated when training effectively and became linear as NFOR manifested. In our opinion however, it is unlikely that either occurrence predicts NFOR; instead each individual has their own unique cardiac autonomic status and HRV relationship (Goldberger et al., 2001), which is likely related to situational and genetic factors. Figure 14 reveals the unique morning resting Ln rMSSD to R-R interval ratio profile of 4 Olympic and World champions in the 62-day lead up to winning their 2011/2012 event. All athletes show distinctly different profiles, irrespective of the fact that all athletes executed gold medal winning performances.





**Figure 13:** Changes in the natural logarithm of the square root of the mean sum of the squared differences between R-R intervals (Ln rMSSD) and the Ln rMSSD to R-R interval ratio with 90% confidence limits (CL) for Athlete A (performing well) and Athlete B (performing poorly; see text) over a 62-day period in the build-up to a key rowing world cup event. Black circular symbols indicate the weekly average values for both Ln rMSSD and Ln rMSSD to R-R interval ratio respectively; while the black line represents the 7-day rolling average. The arrows indicate the day of the final (medal) race. The grey shaded area indicates the individual smallest worthwhile change in both values (see methods in reference (Plews et al., 2012)); the black dashed line represents the zero line of the SWC to indicate clear/unclear changes when of the 90% CL overlaps (Hopkins et al., 2009). \* indicates a 'clear' change in both weekly averaged values, \*\* indicate an 'unclear' change in both weekly averaged values that are above the SWC in the weeks prior to the race.



**Figure 14:** Correlation and 90% confidence limits between the natural logarithm of the square root of the mean sum of the squared differences between R-R intervals (Ln rMSSD) and R-R interval length in two 2012 Olympic Champion rowers and two 2011 World Champion rowers taken every morning upon waking in their 62-day build-up to each pinnacle event. Correlation coefficients were almost perfect ( $r = 0.91$  (confidence interval (CI) 0.87; 0.94)) and trivial ( $r = -0.03$  (CI -0.18; 0.24)) for Olympic Champion rowers 1 and 2 respectively. Comparatively these values were large ( $r = 0.67$  (CI 0.53; 0.77)) and small ( $r = 0.25$  (CI 0.04; 0.44)) for World Champion rowers 3 and 4.

In summary, reductions in HRV have been associated with fatigue and/or NFOR in recreationally-trained and well-trained subjects (Borresen & Lambert, 2008; Bosquet et al., 2008; Hynynen et al., 2008; Uusitalo et al., 2000). However, conclusions from past literature reporting isolated HRV values should be viewed with caution (Plews et al., 2012; Plews, Laursen, Kilding, et al., 2013). We suggest the use of both the Ln rMSSD (weekly average) and Ln rMSSD to R-R interval ratio to correctly interpret fatigue, or a 'readiness to perform' in elite athletes (e.g. worthwhile reductions in Ln rMSSD with concomitant increases in the Ln rMSSD: R-R interval ratio are more indicative of fatigue, with decreases in both indicating readiness to perform; i.e., Figure 13). Furthermore, the optimal relationship between HRV and R-R interval for training and performance alone is likely to be individual (Figure 14; i.e. correlated, non-correlated or saturated (Kiviniemi et al., 2004)). This implies that longitudinal monitoring and an understanding of a particular athlete's response to training and competition (i.e., recognizing each athlete's optimal Ln rMSSD to R-R interval fingerprint) is needed before this relationship can be useful enough to assist with training prescription.

### **6.5.2 Changes in HRV and performance in elites**

As mentioned previously, studies have shown that during intense training periods, vagal indices of HRV decrease acutely, and rebound beyond their pre-training level during subsequent recovery or lighter training periods (Atlaoui et al., 2007; Garet et al., 2004; Hautala et al., 2001; Iellamo et al., 2002; Pichot et al., 2000). The rebound of HRV has been shown to be associated with improved performance in recreationally-trained and well-trained athletes (Atlaoui et al., 2007; Garet et al., 2004). However as mentioned in "HRV is response to training load", the bell-shaped relationship between fitness and HRV sometimes apparent in both elites and athletes with extensive training

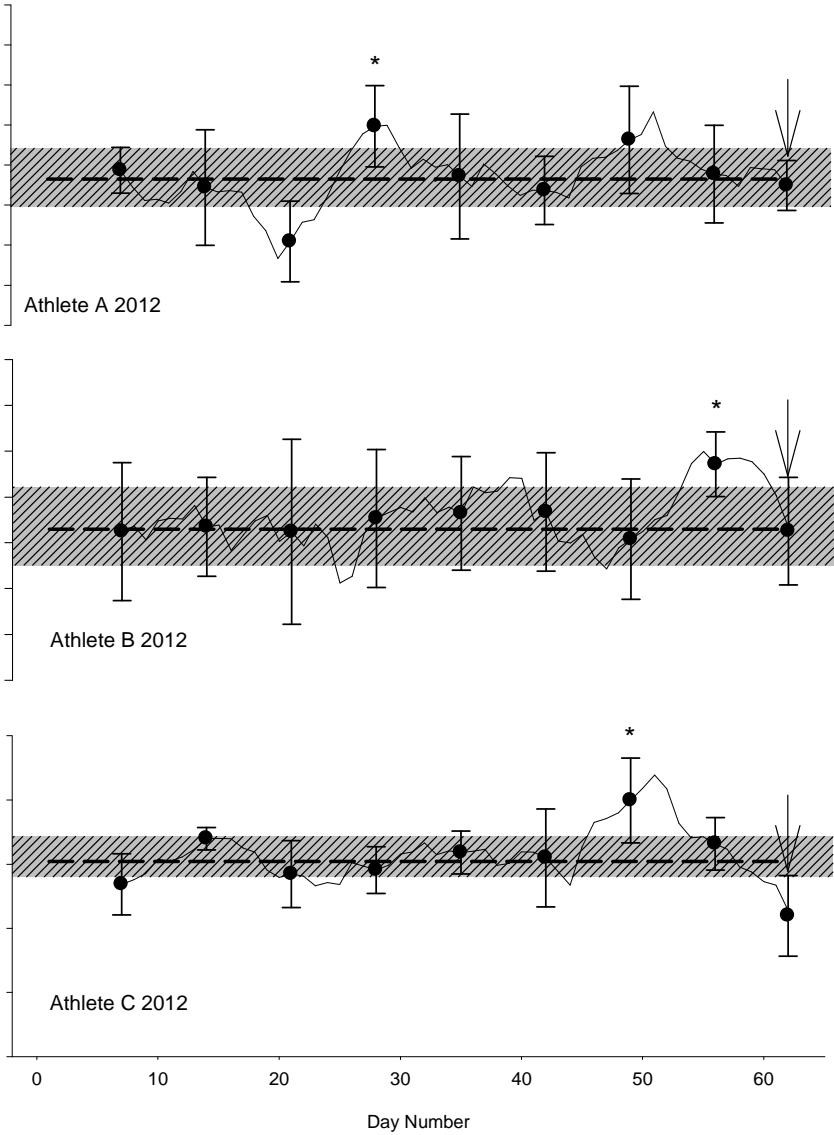
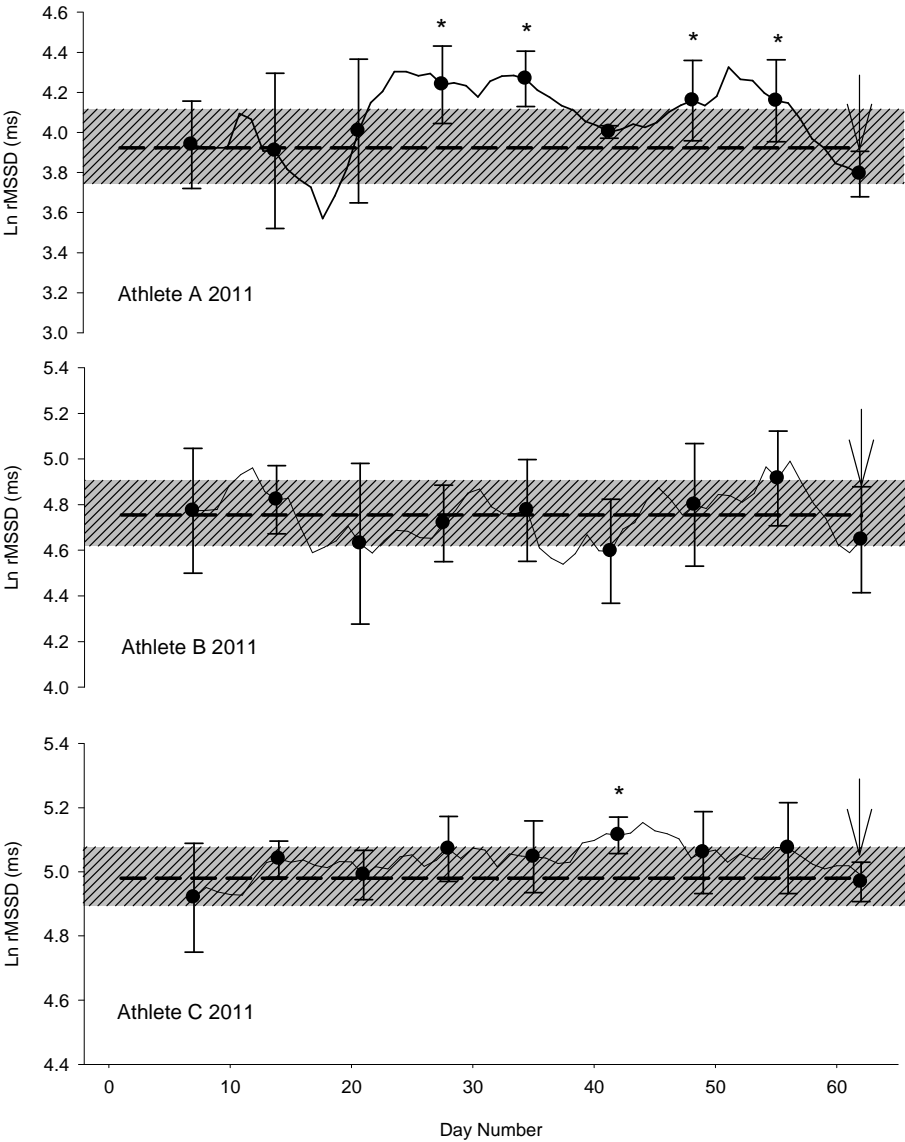
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histories means that making this assessment is more challenging. Figure 15 shows Ln rMSSD values in three elite rowers during their preparation for the 2011 World Rowing Championships and 2012 London Olympic Games. Each athlete won their event. Over this 62-day period, training was at a high intensity (DJ Plews, personal observations) with training volumes reaching  $17 \text{ hr } 21 \text{ min} \pm 3 \text{ hr } 51 \text{ min/wk}$  (2011) and  $16 \text{ hr } 44 \text{ min} \pm 5 \text{ hr } 05 \text{ min/wk}$  (2012). In these athletes, HRV generally increased in the week(s) before each event (going above the SWC; 4 out of 6 points being “clear” 1-3 weeks prior to the event), before the values decreased to slightly lower levels (generally within the SWC) before the race.

As such, it appears that for elite athletes, increases in HRV in the weeks before their event, during their highest training loads (Figure 15), are likely associated with a positive performance outcome. This may indicate an athlete is ‘coping’ with the applied training load and is making positive adaptations. Conversely, Iellamo et al. (2006) reported small, non-significant decreases in HRV profiles in Olympic rowers during strenuous training, which is likely due to the very long history of intensive training and small (undetectable) changes in fitness. It is possible however, that the use of “individual” SWCs may permit a better representation of meaningful changes in HRV in elites for the purpose of monitoring and assessing adaptation.

The fact that HRV values declined as competition approached is in agreement with other studies, in that lower levels of HRV prior to competition tend to be associated with superior performances (Iellamo et al., 2002; Manzi et al., 2009). As such, it is clear that in these athletes, that are the best in the world at their event, a high HRV does not necessarily imply superior fitness (Atlaoui et al., 2007; Garet et al., 2004; A. Hautala et al., 2001; Pichot et al., 2000) and/or performance (Atlaoui et al., 2007; Garet et al.,

2004). The reason why HRV reduces to lower values prior to competition, from both a physiological and performance perspective, is unknown. However, as mentioned, a lower HRV does not necessarily imply fatigue (i.e., saturation), and is therefore unlikely to 'rebound' in elites when training load is reduced and freshness increased. Furthermore, tapers leading into competition typically consist of reductions in training volume with the maintenance of intensity (Mujika et al., 2000). The reduction in training volume might elicit lowered blood plasma volume, and in turn, HRV (Buchheit, Laursen, et al., 2009; Convertino, 1991). However, the maintenance of high intensity exercise during the taper should, in theory, attenuate HRV reductions (Kaikkonen et al., 2007; Kaikkonen et al., 2008; Seiler et al., 2007). Another possible explanation for the reduced HRV observed around the time of competition in elites may be due to pre-competition stress. However, changes in parasympathetic activity have not been shown to be associated with pre-competition anxiety (Iellamo et al., 2003), and the HRV values reported here (Figure 15) have all been averaged over 7-day periods to reduce noise. From a performance perspective, the higher background of parasympathetic activity that is associated with intensified training loads (Buchheit et al., 2004) may compromise cardio-acceleration during exercise, thereby limiting oxygen delivery and performance (Parouty et al., 2010).



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**Figure 15:** The morning resting weekly averaged values of the natural logarithm of the square root of the mean sum of the squared differences between R-R intervals (Ln rMSSD) with 90% confidence limits (CL) over a 62-day period leading up to the 2011 World Rowing Championships and 2012 London Olympic Games in three elite rowers. All athletes won their events and the performance was perceived to be optimal. The black circles indicate the weekly averaged Ln rMSSD value, while the black line represents the 7-day rolling average. The arrows indicate the day of the final (medal) race. The grey shaded area indicates the individual smallest worthwhile change (SWC) in Ln rMSSD values (see methods in reference (Plews et al., 2012)). The black dashed line represents the zero line of the SWC to indicate clear/unclear changes when of the 90% CL overlaps (Hopkins et al., 2009). \* indicates a 'clear' change in weekly averaged Ln rMSSD values, \*\* indicate an 'unclear' change in the weekly averaged Ln rMSSD values that are above the SWC in the week/weeks prior to the medal race.

Additionally, increases in sympathetic activity have been linked to improvement in peripheral adaptations such as faster time to peak torque.(Hedelin et al., 2001) Therefore, it is reasonable to assume that the reduced background of parasympathetic activity/increases sympathetic activity (Iellamo et al., 2002; Manzi et al., 2009) that occurs during the taper may reflect increased ‘freshness’ (Bannister, 1991), and readiness to perform. However, more research is needed to establish why HRV changes in this manner during the lead-up to competition in elites, and what magnitude of change may predict ‘detrimental’ or ‘optimal’ performance.

### 6.6 Conclusion

The measurements of vagal-related indices of HRV remain promising tools for the monitoring of training status in endurance sports. However, it is clear that HRV responses are individual and dependent on fitness level and training history. As such, and although the data presented in this paper focused on elite athletes, the HRV response in any athlete with a long history of training will likely be similar to that reported here (moderately-trained or elite). Accordingly, it is important to be aware of the different responses of these variables and the athlete being monitored. In this current opinion, we suggest that longitudinal monitoring is required to understand each athlete’s optimal HRV to R-R interval fingerprint (i.e., Figure 14). The possible indices of HRV that are practically useful for monitoring training status in elite athletes include weekly and 7-day rolling averaged Ln rMSSD, and the Ln rMSSD to R-R interval ratio, using the individual SWC to represent a meaningful change in fatigue and/or fitness (Plews et al., 2012). Further, we encourage practitioners to use just one HRV index for analysis; research suggests Ln rMSSD provides the most reliable and practically applicable measure for day-to-day monitoring. In the case of elite athletes, increasing HRV values (as competition approaches) may be a sign of positive adaptation and/or coping with



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training load, while reductions in HRV in the week/days before pinnacle events may represent increasing freshness and readiness to perform. Further research is needed to confirm this initial finding and gain a clearer understanding of how changes in HRV relate to training intensity distribution (Seiler et al., 2007), as well as to describe further the HRV trends for elite athletes leading into major competition.

**CHAPTER SEVEN:**

**HEART RATE VARIABILITY AND  
TRAINING INTENSITY  
DISTRIBUTION IN ELITE  
ROWERS**

## 7.1 Abstract

**Purpose:** Elite endurance athletes may train in a ‘polarised’ fashion, such that their training intensity distribution preserves autonomic balance. However, field data supporting this is limited. **Methods:** We examined the relationship between heart rate variability and training intensity distribution in 9 elite rowers during the 26-week build-up to the 2012 Olympic Games (2 won gold and 2 won bronze medals). Weekly-averaged log-transformed square root of the mean sum of the squared differences between R-R intervals ( $\ln rMSSD$ ) were examined, with respect to changes in total training time (TTT) and training time below the first lactate threshold ( $<LT_1$ ), above the second lactate threshold ( $LT_2$ ), and between  $LT_1$  and  $LT_2$  ( $LT_1-LT_2$ ). **Results:** After substantial increases in training time in a particular training zone/load, standardized changes in  $\ln rMSSD$  were +0.13 (unclear) for TTT, +0.20 (51% chance increase) for time  $<LT_1$ , -0.02 (trivial) for time  $LT_1-LT_2$ , and -0.20 (53% chance decrease) for time  $>LT_2$ . Correlations ( $\pm 90\%$  confidence limits) for  $\ln rMSSD$  were small vs. TTT ( $r = 0.37 \pm 0.8$ ), moderate vs. time  $<LT_1$  ( $r = 0.43 \pm 0.10$ ), unclear vs.  $LT_1-LT_2$  ( $r = 0.01 \pm 0.17$ ) and small vs.  $>LT_2$  ( $r = -0.22 \pm 0.5$ ). **Conclusion:** These data provide supportive rationale for the polarised model of training, showing that training phases with increased time spent at high-intensity suppress parasympathetic activity, whilst low-intensity training preserves and increases it. As such, periodised low-intensity training may be beneficial for optimal training programming.

## 7.2 Introduction

Successful training programs for elite endurance athletes typically involve relatively long periods of high training loads where the stress/re-generation balance is challenged (Seiler & Kjerland, 2006). The ability to distribute this training intensity optimally may be important to maximise adaptation without developing non-functional

overreaching (Fiskerstrand & Seiler, 2004). Two best-practice endurance training theories are thought to exist (K. S. Seiler & Kjerland, 2006). These include the *threshold* model of training, where athletes tend to train predominantly between the first and second lactate thresholds (Londeree, 1997), and the *polarised* model of training, with athletes performing 75-80% of their training below the first lactate threshold, and the balance above the second lactate threshold (Steinacker et al., 2000). Although the threshold model of training may induce significant physiological improvements in recreational and untrained athletes (Gaskill et al., 2001), the polarised model of training appears to be a more effective strategy used amongst elites and well-trained endurance athletes (K. S. Seiler & Kjerland, 2006),

The autonomic nervous system is an important regulator of homeostasis during periods of high training loads (Plews et al., 2012; K. S. Seiler & Kjerland, 2006). The cardiac autonomic nervous system, as measured via heart rate variability (HRV), can show large reductions during such training, indicative of non-functional overreaching (NFOR) and/or overtraining (Plews et al., 2012). As such, the ability to preserve autonomic balance in elite athletes via the maximisation of the training intensity distribution may be important. It has been suggested that exercise intensity is the key determinant of acute cardiac parasympathetic suppression, and may therefore be an important variable to consider when looking to optimise the day-to-day training distribution (Seiler et al., 2007; Stanley et al., 2013). For example, Seiler et al.(2007) suggested that the first ventilatory threshold (VT<sub>1</sub>) may demarcate the 'binary' threshold for ANS recovery, by allowing elite athletes to preserve autonomic balance by performing large training volumes at exercise intensities below VT<sub>1</sub> (in line with the polarised model of training). This preservation of the ANS is thought to subsequently

allow for “maximal sympathetic mobilization” during high-intensity efforts (i.e. above  $VT_2$ ), yet still permit achievement of high training volumes.(Seiler, 2010)

While a laboratory-based study by Seiler et al. (2007) using the cardiac ANS response to acute exercise in elite athletes provides supportive rationale for the *polarised* model of training, data from elite athletes working through multiple training cycles towards key events is lacking. Training at varying exercise intensities in isolation may elicit different effects on ANS balance (Seiler et al., 2007; Stanley et al., 2013), but in reality, this seldom happens in actual training. In the daily training life of the elite, athletes will often perform both low (below  $VT_1$ ) and high intensity (above  $VT_2$ ) training sessions in the same day, or even in the same training session. Thus, examining Seiler’s hypothesis using data from an elite cohort of athletes working towards a pinnacle event is needed to examine this theory. We therefore longitudinally monitored the morning resting HRV and training distribution in nine elite rowers during their build-up to the 2012 London Olympic Games. In doing so, our aim was to examine the interaction between HRV and training intensity/load.

### 7.3 Methods

#### *Subjects*

Nine elite heavyweight rowers (4 female: mass  $74.1 \pm 1.6$  kg, height  $178.8 \pm 5.9$  cm; 5 male: mass  $95.0 \pm 5.9$  kg, height  $191 \pm 6.0$  cm) were monitored in the  $30 \pm 2$  week build-up to the 2012 London Olympic Games. The publication of this data was also approved by the Human Research Ethics Committee of AUT University.

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### *Intensity zone determination*

At the start of the monitoring period, all athletes underwent a progressive exercise test to assess first (LT<sub>1</sub>) and second (LT<sub>2</sub>) lactate thresholds. The incremental exercise test was performed according to the Australian rowing physiological assessment guidelines (Hahn, Bourdon, & Tanner, 2000), of which all athletes were well accustomed to. Here, the starting power output and step increments were chosen based on each athlete's personal best time recorded over 2000m performed on the Concept II ergometer. The drag factor on the rowing ergometer was adjusted to match the gender of the rower (heavyweight female = 110 units, heavyweight male = 130 units), as per Rowing New Zealand standards. Subjects performed 7 x 4-min incremental steps, with the last step being an all-out effort. Rowers were asked to maintain their target power output during each step of the test. All stages were followed by 1 min of passive rest during which an earlobe capillary blood sample was collected to determine blood lactate concentration (Lactate Pro, Arkray, Japan). Both LT<sub>1</sub> and LT<sub>2</sub> were determined via the modified D-max method (Newell et al., 2007), as calculated via a software package (ADAPT V3.8; Australian Institute of Sport).

### *Training data analysis*

All training sessions (rowing and cross-training) were recorded via a heart rate monitor (Polar RS800cx, Polar Electro, Kempele, Finland) and downloaded via the relevant software (Polar ProTrainer 5 – version 5.40.172). Training intensity was based on heart rates (HR) at LT<sub>1</sub> and LT<sub>2</sub> determined during the progressive exercise test. Every week, the total average time recorded in each zone was calculated using the function available on the Polar ProTrainer software, which determined the actual time in each zone (hours: min: sec). Training variables were broken down into the total training time (TTT), time spent at a HR below LT<sub>1</sub> (<LT<sub>1</sub>HR), time spent in the HR zone

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between  $LT_1$  and  $LT_2$  ( $LT_1-LT_2HR$ ) and time spent at a HR above  $LT_2$  ( $>LT_2HR$ ). If more than one entire training day was not recorded in a week, that week was removed from the overall analysis. Training time spent  $<100 \text{ b.min}^{-1}$  was also excluded from analysis.

Training load was calculated via the HR training stress score method using Trainingpeaks.com.(www.trainingpeak.com) This is a HR-derived TRIMP measure (Bannister, 1991) which has recently been used to quantify endurance training load (Wallace, Slattery, & Coutts, 2014).

### *Heart rate variability recordings*

Heart rate variability was measured upon waking via R-R series recorded using the same Polar RS800cx HR monitor. Athletes were instructed to leave both HR monitor watch and electrode strap by their bedside each evening to ensure minimum disturbances when applying the apparatus. The R-R series data was then analysed using the ProTrainer Polar 5 software (version 5.40.171, Polar Electro) Occasional artefact-noise was automatically replaced with the interpolated adjacent R-R interval values using the software (filter power = moderate; minimum protection zone = 6). The square root of the mean sum of the squared differences between R-R intervals (rMSSD)(TaskForce, 1996) was calculated during the last 5 min of 6 min supine rest recording. HRV analysis was limited to rMSSD since it reflects vagal activity (TaskForce, 1996) and has much greater reliability than other spectral indices (Al Haddad et al., 2011), particularly during ‘free-running’ ambulatory conditions (Penttila et al., 2001b). The rMSSD values were averaged over a 1-week period, as this better represents changes in the autonomic nervous system with training (Plews et al., 2012; Plews, Laursen, Kilding, et al., 2013). If athletes missed more than three days of HRV recording in a week, the data point was removed from the overall analysis (Plews,

Laursen, Le Meur, et al., 2013). The total amount of weeks used for analysis of each rower was subsequently matched in order for each athlete to have the same number of weeks used in the data analysis.

### *Statistics*

Data are presented as means, standard deviations (SD) and 90% confidence limits (CL). To take into account individual differences, all data (HRV and training) were log-transformed. To assess when substantially higher amounts of time per week were spent at a particular training intensity (or load), each training variable for each individual athlete was ranked from highest to lowest. The Ln rMSSD values for the highest four data points of each individual's 26-week period (for each training variable) were averaged and compared with the average of the individual's remaining Ln rMSSD values. If the subsequent data point (data point 5) was similar to the fourth (within 1%), this was also included. This procedure was repeated for each training variable. Four to 5 data points was chosen as it represented the top echelon of all 26 data points (13-16%). Furthermore, this resulted in all standardised differences ('normal' to 'high') for each training variable to be similarly classified as large (Hopkins et al., 2009). These data were then further analysed using a modified statistical spreadsheet (Hopkins, 2006), which calculates the standardised differences/changes or effect sizes (ES, 90% CI). Threshold values for ES statistics were  $\leq 0.2$  (trivial),  $>0.2$  (small),  $>0.6$  (moderate),  $>1.2$  (large),  $>2.0$  (very large). The smallest worthwhile change (SWC) was also calculated using the between-athlete standard deviation of only the averaged "normal" training variable (e.g., 0.2 multiplied by the between-athlete SD of the average Ln rMSSD values during "normal" training only, carried out individually for total time,  $<LT_1$ ,  $LT_1-LT_2$ ,  $>LT_2$  and training load). This was calculated in order to assess substantial changes in Ln rMSSD after considerably larger amounts of time within



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particular training variables (Hopkins et al., 2009), Quantitative chances of either higher or lower Ln rMSSD values after a greater amount of time at a particular training intensity/load were also evaluated qualitatively as follows: 25-75% possibly, 75-95% likely, 95-99% very likely, >99% almost certain. If the chance of higher or lower differences was >5%, then the true difference was assessed as unclear.

Pearson's correlation was used to establish the relationships between HRV and training variables (i.e., time below LT<sub>1</sub>, etc.) taken as a percentage difference from the average of the variable measured over the entire training period. The *r* values were calculated individually, and then the average of all subjects was taken.<sup>14</sup> We chose this method as each subject trained slightly differently (total hours in zone etc.), and had different inherent rMSSD values. To evaluate three-factor predictor models (linear model), partial correlation coefficients were also used to assess the relationship between the training variables (time spent below LT<sub>1</sub>HR, time spent between LT<sub>1</sub>-LT<sub>2</sub>HR, and time spent above LT<sub>2</sub>HR only) and HRV, while controlling for the effect of other training variables (i.e. the unique relationship of the assessed variable). Subsequently, a three-factor multiple-regression model of each training variable was computed to determine the predictors of all three variables on HRV. The magnitude of correlation (*r* (90% CI)) between HRV and the training variable was assessed with the following thresholds: <0.1, trivial; <0.1-0.3, small; <0.3-0.5, moderate; <0.5-0.7, large; <0.7-0.9, very large; and <0.9-1.0, almost perfect (Hopkins et al., 2009). If 90% CI overlapped small positive and negative values, the magnitude of correlation was deemed 'unclear'. (Hopkins et al., 2009) Multiple and partial regression analysis was carried out using SPSS 19 (SPSS Inc, Chicago, USA).

## 7.4 Results

Each athlete had 26 weeks of training and HRV data that could be used in the overall analysis; this allowed for an equal number of data points across all nine subjects. The average weekly training hours across all nine rowers during this period was 17 h 46 min  $\pm$  4 h 23 min. Of this time,  $77.3 \pm 12.7\%$  was spent  $<LT_1HR$ ,  $16.9 \pm 11.2\%$  between  $LT_1$  and  $LT_2$  and  $5.8 \pm 4.4\%$   $>LT_2HR$ . The average weekly training load was  $905.3 \pm 258.3$  arbitrary units (AU). All performances at the Olympic Games were perceived as optimal by athletes and support team, with two athletes winning gold, and two athletes winning bronze medals. The remaining two and three athletes placed in the 'A' (4<sup>th</sup> and 6<sup>th</sup>) and 'B' finals (8th).

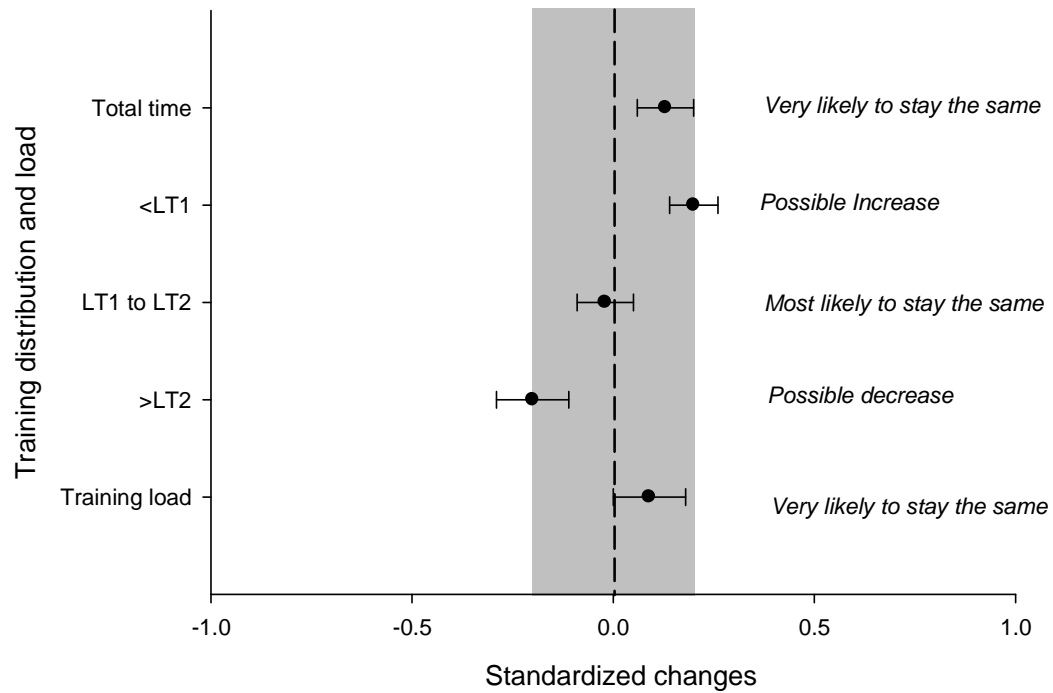
Mean values of 'normal' vs. 'high' amounts of time spent in a particular training zone/load are presented in Table 9. These changes in Ln rMSSD after substantially greater time at a particular training zone/load are expressed as standardised differences (effect sizes (ES), Figure 16). Large increases in time spent at a low intensity ( $<LT_1HR$ ) and high intensity ( $>LT_2HR$ ) resulted in *possible* small increases (ES  $\pm 90\%$  CL:  $0.20 \pm 0.07$ ; 51/49/0) and decreases ( $-0.20 \pm 0.06$ ; 0/47/53) in Ln rMSSD. Conversely, large increases in total training time and training load resulted in *very likely* trivial changes in Ln rMSSD ( $0.13 \pm 0.07$ ; 5/95/0 for total training time;  $0.09 \pm 0.09$ ; 3/97/0 for training load). Last, large increases in time spent between  $LT_1$ - $LT_2HR$  resulted in *most likely* trivial changes in Ln rMSSD ( $-0.02 \pm 0.06$ ; 0/100/0).

Table 9: Mean values of ‘normal’ vs. ‘high’ amounts of time spent in a particular training zone/load

Training variable	Normal training duration/load (hh:mins $\pm$ 90% CI)	High training duration/load (hh:mins $\pm$ 90% CI)	Standardised difference ‘normal’ vs. ‘high’ ( $\pm$ 90% CI); rating
Total training time	16:00 (14:43; 17:18)	19:32 (18:17; 20:48)	1.60 (0.63; 2.58 ); large
<LT1	11:32 (10:15; 12:49)	15:57 (14:31; 17:23)	1.70 (1.16; 2.24); large
LT1-LT2	2:07 (1:36; 2:38)	3:53 (3:08; 4:38)	1.51 (1.32; 1.71); large
>LT2	0:43 (0:24; 1:01)	1:19 (0:56; 1:42)	1.21 (0.87; 1.49); large
Training load (AU)	764.1 (667; 861.1)	1047 (923.9; 1169)	1.35 (0.86; 1.85); large

Values are means with 90% confidence intervals (CI). Training zones/variables are total training time, time below first lactate threshold (<LT<sub>1</sub>) heart rate, time training between LT<sub>1</sub> and LT<sub>2</sub> heart rates (LT<sub>1</sub>-LT<sub>2</sub>), time training above the second lactate threshold (>LT<sub>2</sub>) heart rate, and training load.

The data for zero-order and partial correlations are presented Figure 17. Additionally, the individually correlation data for the training variables is also presented in Table 10. For zero-order correlations, there were small positive and negative relationships between changes in total training time ( $r \pm 90\% \text{ CL} = 0.37 \pm 0.09$ ) training load ( $r = 0.23 \pm 0.16$ ), time spent >LT<sub>2</sub>HR ( $r = 0.22 \pm 0.05$ ) and Ln rMSSD. Alternatively, there was a moderate relationship between changes in time spent at a low training intensity (<LT<sub>1</sub>HR) and Ln rMSSD ( $r = 0.43 \pm 0.09$ ). The relationship between changes in time spent between LT<sub>1</sub>-LT<sub>2</sub>HR and Ln rMSSD was unclear ( $r = 0.01 \pm 0.17$ ). These relationships were similar for both zero-order and partial correlations.



**Figure 16:** The mean standardised change in Ln rMSSD after substantially greater periods of time in particular training zones or with training load. Error bars indicate uncertainty in the true mean changes with 90% confidence intervals; if error bars overlap the opposing positive or negative trivial thresholds, changes are deemed unclear. Values to the right indicate an increase, whereas values to the left indicate a decrease in Ln rMSSD. The shaded area represents a trivial effect size (see “Methods”). Training variables are total training time, time spent training below the first lactate threshold ( $LT_1$ ) heart rate, time spent training above the second lactate threshold ( $LT_2$ ) heart rate, time spent training between the  $LT_1$  and  $LT_2$  heart rates ( $LT_1$  to  $LT_2$ ) and training load. Quantitative chances of change are quantified qualitatively in the text to the right (see “Methods”).

## 7.5 Discussion

The present study is the first to show the interaction between cardiac autonomic balance and the distribution of training intensity in elite athletes. The present study is particularly unique due to the fact that the data are ecologically valid (not lab-based), and obtained in very high calibre athletes who subsequently won Olympic medals. Although there was no controlled performance trials used in the study, the ideal performance outcomes achieved at the Olympic Games suggest that training preparation was optimal. While effect sizes and correlations were all only small-to-moderate (“possible” increase/decrease), there was a “clear” association between increases in Ln rMSSD and time spent  $<LT_1HR$ , and conversely, a “clear” association between lower Ln rMSSD values and greater amounts of time spent  $>LT_2HR$ . Indeed, small clear increases and decreases in Ln rMSSD were only seen after substantially greater amounts of training time  $<LT_1HR$  (Figure 16). Whilst acknowledging that the qualitative inferences for increases and decreases were only ‘possible’ (51/49/0 for  $<LT_1HR$  and 0/47/53 for  $>LT_2HR$ ), all nine athletes showed positive relationships between the change in HRV and time spent  $<LT_1HR$ , and negative relationships with HRV and time spent  $>LT_2HR$  (Table 10). Last, as both zero-order and partial regressions were similar (Figure 17), these relationships appear to occur independently of time spent in other training zones (e.g. the effect of time spent  $>LT_2HR$  on HRV was independent from time spent  $<LT_1HR$ ).

Individual and overall relationship between heart rate variability (Ln rMSSD) and each training variable												
Variable		Athlete 1	Athlete 2	Athlete 3	Athlete 4	Athlete 5	Athlete 6	Athlete 7	Athlete 8	Athlete 9	Average r	Rating
Total Time	<i>r</i>	0.55	0.34	0.40	0.22	0.18	0.21	0.36	0.52	0.50	0.37 (0.28; 0.45)	Small
Time below LT1	<i>r</i>	0.54	0.36	0.31	0.40	0.23	0.34	0.43	0.62	0.66	0.43 (0.32; 0.52)	Moderate
	<i>Partial r</i>	0.48	0.30	0.29	0.55	0.08	0.38	0.47	0.56	0.66	0.42 (0.31; 0.53)	Moderate
Time between LT1 and LT2	<i>r</i>	0.13	0.23	-0.14	0.13	0.25	-0.32	0.00	0.26	-0.50	0.01 (-0.16; 0.17)	unclear
	<i>Partial r</i>	0.07	0.18	-0.16	0.43	0.15	-0.28	0.09	0.11	-0.34	0.03 (-0.12; 0.18)	unclear
Time above LT2	<i>r</i>	-0.16	-0.21	-0.19	-0.18	-0.14	-0.35	-0.14	-0.36	-0.24	-0.22 (-0.27; -0.17)	Small
	<i>Partial r</i>	-0.12	-0.17	-0.16	-0.47	-0.10	-0.31	-0.26	-0.15	-0.16	-0.19 (-0.28; -0.09)	Small
Training load	<i>r</i>	0.47	0.58	0.48	-0.22	0.21	0.04	0.17	0.35	0.12	0.24 (0.09; 0.40)	Small

**Table 10:** Zero-order and partial-order correlations for each training variable with 90% confidence limits. Training time below the first lactate threshold (LT<sub>1</sub>) heart rate, training time above the second lactate threshold (LT<sub>2</sub>) heart rate, and training time between LT<sub>1</sub> and LT<sub>2</sub> heart rate (LT<sub>1</sub> to LT<sub>2</sub>) were included in the multiple regression model (forced entry model) for its associated correlation coefficient (*r* (90% confidence limits)). The overall *r* for the multiple regression was *r* = 0.51(Large), 0.43; 0.60)).

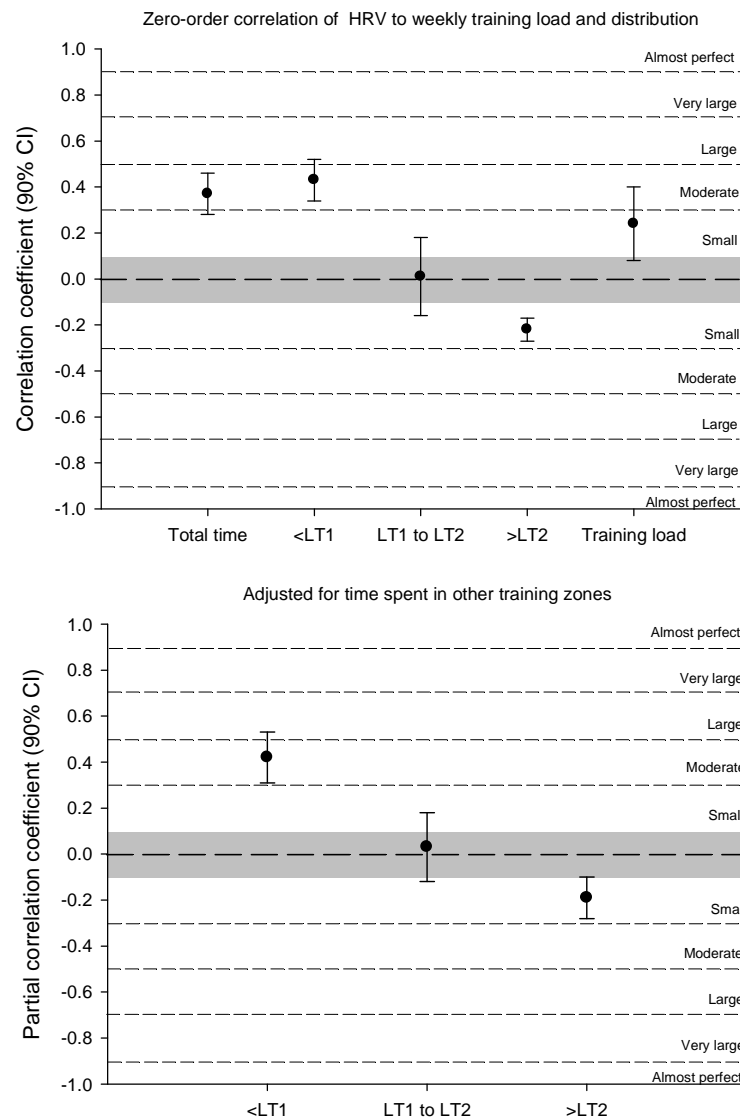
It is important to note that training intensity was governed by rowing stroke rate prescriptions and not HR during this 26-week observation period. As such none of these athletes were given instruction by their coach to train in a particular HR training zone, and only naturally appeared to fall into the polarised model of training (77.4% <LT<sub>1</sub>HR/ 12.7% LT<sub>1</sub>-LT<sub>2</sub>HR/ 5.8% >LT<sub>2</sub>HR). This is in line with other studies (Fiskerstrand & Seiler, 2004; K. S. Seiler & Kjerland, 2006; S. Seiler) showing elite athletes successfully training in accordance with the polarised model of training, rather than the threshold model, as described previously (Steinacker et al., 2000). In the present study, training time spent between LT<sub>1</sub>-LT<sub>2</sub>HR is slightly higher than previously reported in rowers (e.g. 75-80% <LT<sub>1</sub>HR, 10-20% >LT<sub>2</sub>HR) (Steinacker et al., 2000). However, it is well known that the HR lag apparent with the commencement of intense (or interval) exercise (Cerretelli & Di Prampero, 1971) likely overestimates the metabolic work performed between LT<sub>1</sub>-LT<sub>2</sub>HR, and underestimates the amount of time spent above >LT<sub>2</sub>HR. It is therefore possible that the training distribution with respect to external power production was more polarized than that observed with these HR measures.

### *Time spent below LT<sub>1</sub>HR*

The relationships shown between the higher Ln rMSSD values and the greater amounts of training time spent <LT<sub>1</sub>HR (Figure 16 and Figure 17) is not surprising. Indeed, low-intensity exercise appears to do little to affect reductions in cardiac parasympathetic activity, irrespective of the training duration. (Stanley et al., 2013) Furthermore, given that HRV recordings in this study occurred every morning (rather than immediately post-exercise), Ln rMSSD was likely to have rebounded above pre-existing levels before the subsequent HRV recording took place (Stanley et al., 2013). This is particularly the case in Olympic athletes, who are more resilient to exercise stress and have faster cardiac autonomic recovery (Hautala et al., 2001). Whilst acknowledging that these relationships are only small, the physiological processes that

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are responsible for the possible increase ('rebound') in vagal-related HRV measures after low-intensity exercise are likely two-fold.



**Figure 17:** Mean correlation coefficient (upper panel) and partial correlation (lower panel) with 90% confidence intervals for the percentage change from the mean for Ln rMSSD and training time with zones/load values. The shaded grey area represents trivial correlations. Training variables are total training time, time spent training below the first lactate threshold (LT<sub>1</sub>) heart rate, time spent training above the second lactate threshold (LT<sub>2</sub>) heart rate, time spent training between the LT<sub>1</sub> and LT<sub>2</sub> heart rates (LT<sub>1</sub> to LT<sub>2</sub>) and training load. Partial correlations are adjusted for time spent in the respective two training zones.



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First, such elite athletes likely have less blood metabolite accumulation at the same relative exercise intensity and hence less parasympathetic suppression post-exercise (Buchheit, Duche, Laursen, & Ratel, 2010). Second, the known increase in blood plasma volume (i.e. hypervolemia) following endurance exercise (Pugh, 1969) likely elicits an increase in parasympathetic activity via baroreflex stimulation (Buchheit, Laursen, et al., 2009). As such, the resultant combination of dehydration-induced plasma volume super-compensation through extensive training duration, and lack of metabolite accumulation, means the rebound in cardiac parasympathetic activity is likely to be maximised.

### *Time spent between LT<sub>1</sub>-LT<sub>2</sub>HR*

An increase in the time spent training between LT<sub>1</sub>-LT<sub>2</sub>HR appeared to have no effect on Ln rMSSD (Figure 16 and 17). This opposes what has previously been reported by Seiler et al., (2007) who marked LT<sub>1</sub> as the binary threshold for cardiac autonomic disturbances. However, as mentioned previously, the obvious difference is in the timing of the HRV recording (immediately post exercise vs. morning resting), and the uniqueness of these individuals (Hautala et al., 2001). We chose morning resting HRV recordings due to the practicality of the measurement, particularly during the long timeframe (26 weeks) that these data were collected for. Furthermore, the relative intensity of this training band may limit increases in parasympathetic activity due to reduced exercise-induced hypervolemia in comparison with longer time <LT<sub>1</sub>HR; which is likely due to a combination of metabolite accumulation and the shorter duration this training intensity is typically carried out for.

The possible reductions in Ln rMSSD shown after periods including a greater amount of high-intensity exercise was also not surprising (Stanley et al., 2013), and somewhat supports the consensus that training intensity is the primary determinant of post-exercise cardiac parasympathetic suppression. Although both standardized difference and the magnitude of correlations were small (Figure 16 and Table 10), the reductions in Ln rMSSD were still clear (53% chance of a decrease in Ln rMSSD; “possible decrease”) after large increases in training time  $>LT_2HR$  (Figure 16). Interestingly, the two athletes who had the smallest correlations between Ln rMSSD and time  $>LT_2HR$  were the two gold medallist rowers (Table 10, Athlete 5 and 7). These smaller magnitudes of correlation were likely due to these athletes having the quickest cardiac autonomic recovery and therefore the least HRV suppression the following morning when the HRV recordings took place. This perhaps reflects their superior fitness and training status (Hautala et al., 2001). As aforementioned, the autonomic nervous system regulates a number of physiological processes that are responsible for returning the body back to homeostasis after exercise (Stanley et al., 2013). Given that many of these physiological mechanisms are more prominently elevated (or suppressed) with higher exercise intensity (e.g. increased blood acidosis (Buchheit, Duche, et al., 2010), blood lactate (Buchheit, Al Haddad, et al., 2011), plasma epinephrine (Perini et al., 1989), body temperature (Nybo, 2008)) the time taken to regulate these variables back to homeostatic levels is accordingly longer. This is particularly important given the strong association between elevations in such physiological processes and parasympathetic suppression (Buchheit, Al Haddad, et al., 2011). Last, it should be noted that although high-intensity training can also have marked effects on hypervolemia (and in turn cardiac parasympathetic activity) (Buchheit, Laursen, et al.,

2009); large and chronic doses of high-intensity exercise likely nullifies any associated increase in HRV via hypervolemia due to metabolites accumulation.

### *Total training time and training load*

Compared with training time spent  $<LT_1HR$  alone, changes in total training time and the average training load had smaller positive correlations with HRV (Figure 17). Given that total training time and training load have aspects of both training volume and intensity, it makes sense that these relationships are slightly smaller. However, when we considered the changes in Ln rMSSD after substantially greater amounts of total training time or training load in the present study, there were unsubstantial, changes in Ln rMSSD, respectively (Figure 16). Other studies have shown quadratic relationships between training load and vagal-related indexes of HRV (Manzi et al., 2009). However, the correlations in this study were based on percentage changes rather than HRV vs. training load directly which possibly explains the contrasting observation (Manzi et al., 2009). As such, similar to time spent between  $LT_1HR$  and  $LT_2HR$ , the interactions between training volume/intensity and the positive adaptive responses these athletes had, likely nullified any potential increases or decreases in HRV.

### **Practical Application**

The findings of this study have a number of implications for sports practitioners and coaches looking to both maximise training load by distributing training intensity, and for using HRV as a tool to monitor training adaptation. Accordingly, the data has provided further supportive rationale as to why the polarised model of training is likely to be a more successful training strategy than the threshold model (Neal et al., 2013). Seiler et al. (2006) suggested that the preservation of autonomic balance through low-intensity training allows for “maximal sympathetic mobilization” to be retained and

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utilized during high-intensity training. Potentially, a higher resting cardiac parasympathetic activity may reflect increased freshness (at least perceived), and in turn, the ability to perform interval training workouts at a higher exercise intensity later in the day/week. This might be achieved through greater sympathetic mobilization (as signified by high HR; Stanley et al., 2012), potentially eliciting superior training adaptation (e.g. through greater motor recruitment, mitochondrial biogenesis of fast twitch fibres, etc., Seiler et al., 2006) and parasympathetic suppression during the sessions. As the magnitude of cardiac parasympathetic rebound is likely dependent on its suppression during the prior exercise session (and the extent of the suppression is dependent on the intensity; Stanley et al., 2013), it makes sense for such a distribution of training intensity model to be successful, particularly as the rebound in cardiac parasympathetic activity also reflects blood plasma volume expansion (Buchheit, Laursen, et al., 2009). Accordingly, low-intensity training might not only assist in the acute regulation/recovery of ANS, but also allow athletes to perform large volumes of training (77% of total volume) with possibly positive ANS status (Seiler, 2010).

## Conclusion

The present study provides evidence that cardiac autonomic balance in Olympic rowers is possibly influenced by low- ( $<LT_1HR$ ; increases in  $\ln rMSSD$ ) and high- ( $>LT_2HR$ ; decreases in  $\ln rMSSD$ ) intensity training, with total training time, training load and training time spent between  $LT_1$ - $LT_2HR$  having either small or unclear effects on this index, respectively. The increase in cardiac autonomic balance after low-intensity training has a number of potential benefits that may work in synergy with high-intensity training to potentially facilitate superior training adaptations (e.g. the maintenance of ANS balance during periods of high-intensity training). Accordingly, this paper potentially lends further support to the notion that increases in vagal-related

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HRV during peak volume-based training loads may be a sign of positive adaption to training; reductions as a result of the taper should be expected and are potentially a sign of readiness to perform (Plews, Laursen, Stanley, et al., 2013). Finally, when monitoring HRV to assess training adaptation, it is necessary to consider the athlete's individual training phase.

## **CHAPTER EIGHT: OVERALL DISCUSSION and CONCLUSION**

### 8.1 Summary of findings

Elite endurance athletes are always pushing the boundaries between functional and non-functional overreaching in an attempt to gain the greatest possible fitness level (Seiler & Kjerland, 2006). Insight into whether an athlete is adapting well to a training load or approaching non-functional overreaching is, to date however, mostly lacking. The ability therefore to increase or reduce an athlete's training in accordance with a quantitative assessment of an athlete's individual homeostatic status would be highly advantageous and allow more athletes to perform to their potential. The regular indirect measurement of cardiac autonomic nervous system status using morning resting heart rate variability (HRV) represents such a tool, that practitioner, coach and athlete might use to maximise training. Unfortunately, a good understanding in this area and the establishment of 'best practice' methods is currently lacking. The aim of this thesis therefore was to explore HRV further in elite athletes, and develop methods by which morning resting HRV measures could be used to monitor training adaptation.

To date, using HRV to monitor training adaptation is not considered to be entirely straightforward, as equivocal findings have been shown with respect to how HRV responds with positive (Lee et al., 2003; Mourot, Bouhaddi, Perrey, Rouillon, et al., 2004; Yamamoto et al., 2001; Buchheit, Simpson, et al. 2010; Iellamo et al., 2002; Manzi et al., 2009) and negative (e.g. non-functional overreaching and overtraining) (Hynynen et al., 2006; Uusitalo et al., 2000; Hedelin, Wiklund, et al., 2000; Bosquet et al., 2003; Hedelin, Wiklund, et al., 2000; Uusitalo et al., 1998a) adaptation to training. The disparate findings within the literature may have arisen for a number of reasons. First, the large day-to-day variation in HRV recording affects its validity for tracking changes due to training (Al Haddad et al., 2011). Second, the quadratic relationship between HRV and the R-R ratio (Goldberger et al., 1994; Goldberger et al., 2001)

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complicates these relationships (i.e. between positive and negative adaptation (Buchheit & Gindre, 2006)). Third, there is a complex interaction between training load/intensity and HRV responses, and these interactions must be considered within the model (Plews, Laursen, Stanley, et al., 2013). Last but not least, there is a substantial lack of HRV studies carried out in elite athletes (Plews, Laursen, Stanley, et al., 2013), and specifically with reference to data obtained in a practical setting with realistically applicable methodologies.

In light of the limitations identified in the literature, my PhD thesis set out to answer the following questions, using the studies outlined in the previous chapters of the thesis. These were as follows:

**Chapter 3:** What are the HRV responses of elite athletes during maladaptation and how can these changes effectively be tracked?

**Chapter 4:** If we know how to effectively track HRV responses during maladaptation, are these same methodological approaches appropriate to track positive adaptation to training?

**Chapter 5:** Knowing that weekly-averaged HRV values represent a superior procedure for tracking changes with training (chapters 3 and 4), what is the optimal averaging methodology needed to effectively evaluate and assess HRV changes with training?

**Chapter 6:** If we know how changes in HRV can be more effectively tracked, what are some of the typical HRV responses of ‘real’ elite athletes building up to pinnacle events with data collected in a practical setting? Also, given the intricacies of the ANS and HRV, what else must be considered?



**Chapter 7:** Given the complexities of the interactions between HRV and training load/intensity, what are some of the expected changes that occur in HRV within training cycles of elite athletes? How can this be used to monitor training effectively?

The following section discusses the findings from these studies, and how they interrelate.

In the first study (chapter 3), the regular morning resting HRV responses of two elite triathletes over a 77-day period were tracked. One of these athletes became non-functionally overreached (NFOR) and raced poorly, while the other appeared to cope with their training load and raced optimally in the same triathlon event. It was shown that both weekly-averaged values of Ln rMSSD and the individual smallest worthwhile change (SWC; 0.5 of the coefficient variation for the first two weeks of effective training) were successful methods for effectively tracking individual changes in HRV, for both the NFOR and control athlete. Conversely, when isolated day Ln rMSSD values were used for analysis, the assessment of NFOR was often inaccurate. For example, Ln rMSSD viewed in isolation fell outside of its SWC irrespective of training phase (63.6% of total Ln rMSSD points outside the SWC for control athlete and 100.0% for the NFOR athlete during normal training (Figure 5; (Plews et al., 2012)). The reductions in Ln rMSSD shown during overreaching are not surprising, and are in line with “sympathetic” type overtraining (Lehmann et al., 1998; Hynynen et al. 2006; Uusitalo et al. 2000). The most important outcome of this study however was that these changes could be effectively monitored using an averaging methodological approach. It was proposed that a total of two consecutive weeks of 7-day averaged Ln rMSSD values below the SWC would be sufficient to warrant alterations in a training plan (Brink et al. 2010b; Plews et al. 2012).

Another novel finding that arose from this study was that reductions in the day-to-day variation of Ln rMSSD values was also evident for the NFOR athlete ( $r^2 = 0.88$ ; slope = -0.65% per week). More recently, Schmitt et al (2013) challenged this theory, reporting the exact converse of these findings in 57 elite skiers. In the study by Schmitt et al (2013), they used increases in the variation of HRV values to imply fatigue. However, HRV measures were recorded randomly throughout a training cycle using a variety of HRV measures (e.g. time domain and frequency domain), making it difficult to understand the practical relevance of their methodologies. Furthermore, with fatigue measures (done so via questionnaire), it would be difficult to differentiate between functional and non-functional overreaching (Halsen & Jeukendrup, 2004). Nevertheless, evidence supporting the findings shown in chapter 3 of reductions in the day-to-day variation of HRV as indicative of NFOR is yet to be demonstrated elsewhere, and requires further study.

Last from this study, I showed that HRV may be a slightly more sensitive measure compared with resting heart rate (RHR) for monitoring the potential manifestation of NFOR ( $r^2 = -0.88$  vs.  $0.81$  respectively). However, both HRV and RHR should be taken into account due to HRV saturation, with RHR providing potentially a stronger predictor of positive adaptation in the control athlete ( $R^2 = -0.55$  for RHR and  $0.12$  for HRV).

Given that chapter 3 revealed weekly averaged values to be superior at tracking maladaptation compared with isolated values in the second study (chapter 4), this study aimed to assess whether a similar methodological approach was applicable to track positive adaptation to a training stimulus. Here it was shown that weekly-averaged

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vagally-derived indices of HRV were more effective at detecting positive adaptation than single-day values in 10 recreationally trained runners. For example, trivial correlations were observed for maximum aerobic speed vs. Ln rMSSD taken on a single day (Ln rMSSD<sub>day</sub>) ( $r = -0.06$  (-0.59; 0.51)) while a very-large correlation was shown vs. Ln rMSSD averaged over 1 week (Ln rMSSD<sub>week</sub>;  $r = 0.72$  (0.28; 0.91)). Similarly, changes in 10-km running performance revealed a small correlation with Ln rMSSD<sub>day</sub> ( $r = -0.17$  (-0.66; 0.42)) and large correlations for Ln rMSSD<sub>week</sub> ( $r = -0.76$  (-0.92; -0.36)). Furthermore, Ln rMSSD values only went above the SWC in the last week (week 9, when athletes were at their fittest) when values were averaged, which is a more realistic representation of their training status. Last, although HRV values averaged over 1 week were more sensitive at detecting positive adaptation than RHR (correlations with performance were  $R = 0.72$  (0.28; 0.91) for Ln rMSSD and  $R = -0.62$  (-0.87; 0.11) for RHR), practically speaking, in isolation (i.e. values taken from just one day), RHR values were superior (change from week 1-9:  $ES = -0.66$  RHR<sub>day</sub> and  $-0.64$  RHR<sub>week</sub> vs.  $ES = 0.20$  Ln rMSSD<sub>day</sub> and  $0.46$  Ln rMSSD<sub>week</sub>) at evaluating positive adaptation. This could be due to the lower day-to-day variation in RHR that was observed ( $CV = 13.0\%$  RHR<sub>day</sub> and  $12.2\%$  RHR<sub>week</sub>,  $CV = 18.7\%$  Ln rMSSD<sub>day</sub> and  $16.5\%$  Ln rMSSD<sub>week</sub>). As such, if every day HRV recordings are not practically achievable, RHR values should be used.

The third study (chapter 5) explored further methodological approaches to detect meaningful change in HRV data sets. Given that 7-day averaged values were superior at detecting HRV changes due to training (chapters 3 and 4), this study set out to determine the minimum number of days per week that HRV measurements were needed to reveal correspondingly equivalent results. Such data would be practically useful for both researchers and practitioner's intent on using HRV for research and/or monitoring

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in athletes, particularly when athlete compliance may be an issue. The main findings of this study were that HRV must be averaged for a minimum of 3 days in order to elicit comparable results to data averaged over a 1-week period. For example, standardised Ln rMSSD changes and correlations with Ln rMSSD and performance from baseline to overload in trained triathletes (functional overreaching) plateaued after 3 and 4 days respectively (e.g. standardised changes (90% CL):  $0.20 \pm 0.28$ ;  $0.33 \pm 0.26$ ;  $0.49 \pm 0.33$ ;  $0.48 \pm 0.28$ ;  $0.47 \pm 0.26$ ;  $0.45 \pm 0.26$  and  $0.43 \pm 0.29$  using from 1 to 7 days, respectively. Correlations (90% CL) over the same time sequence and training phase were:  $-0.02 \pm 0.23$ ;  $-0.07 \pm 0.23$ ;  $-0.17 \pm 0.22$ ;  $-0.25 \pm 0.22$ ;  $-0.26 \pm 0.22$ ;  $-0.28 \pm 0.21$  and  $-0.25 \pm 0.22$  from 1 to 7 days, respectively. This was supported by the standardised difference data, plateauing after four day of averaging, and reinforced by the apparent very large and almost perfect correlations ( $R^2 = 0.92$  and  $0.97$  respectively). Furthermore, it was also shown that the athlete population (e.g. recreationally trained and well-trained) also needs to be considered when deciding on the number of daily HRV recordings required for averaging. Recreationally trained athletes likely need to average HRV values over more days (~5), due to the higher day-to-day variations in HRV values shown for recreationally trained athletes (e.g. CV for recreational runners =  $10.1 \pm 3.4\%$  for week 1; CV for triathletes =  $6.8 \pm 2.9\%$ , for baseline). Practically speaking, the number of actually recorded HRV values will likely fall between the minimum number of days required and what is practically applicable. The more consistent the daily recordings, the more confident practitioners can be in evaluating changes in HRV (e.g. 90% confidence limits continued to decline until around 5 days of Ln rMSSD value averaging). HRV measured over a 1-week period (or that micro-cycle) will allow both effective and practically applicable evaluations of training responses. This is of course provided the minimum number of required daily recording (3 in trained athletes) is obtained.

Having assessed the HRV trends of many elite athletes over a number of competitive seasons and learning from my developed HRV assessment methods in studies 1, 2 and 3 I realised that limitations existed within its interpretation. This is of particular consideration when the HRV trends of real athletes building up to actual major event competition are the primary focus. The current opinion piece (Plews, Laursen, Stanley, et al., 2013) (chapter 6) explored the HRV responses of Olympic athletes building up to pinnacle events, and from this data, a number of unique observations are discussed. First, when we take into account changes in vagally-derived indices of HRV, practitioners must also consider changes in RHR (R-R interval) due to HRV saturation. Indeed, due to the quadratic relationship evident between Ln rMSSD and the R-R interval (in the case of HRV saturation) (Goldberger et al., 1994; Goldberger et al., 2001; Malik et al., 1993), HRV in elite athletes can decline (below the SWC; suggestive of reductions in vagal-modulation and potentially fatigue) but performance might still be maintained (as vagal tone is still high). Therefore, I suggest the use of the Ln rMSSD to R-R interval ratio to run alongside changes in Ln rMSSD to mitigate this problem. For example, when Ln rMSSD declines at the same time as the ratio, there is no need for alarm (although vagal modulation has been diminished, vagal tone is still high). However, declines in HRV alongside concomitant increases in the Ln rMSSD to R-R interval ratio likely implies fatigue, suggestive of a loss in vagal-tone and modulation (and thereby sympathetic over-activity). Using data from elite athletes in international events, this application is demonstrated (Figure 13).

Second, I showed how HRV profiles (e.g. Ln rMSSD to R-R) are individual (Figure 14) and unlikely to be predictive of performance or training optimization. Although, in my first study, the NFOR triathlete demonstrated altering from a saturated

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to a linear profile (Kiviniemi et al., 2004) as NFOR ensued (Plews et al., 2012). The Ln rMSSD to R-R interval in elite athletes winning Olympics and World Championship rowing events, all showed different profiles (linear, saturated and non-linear (Kiviniemi et al., 2004)). Therefore, profiles should be individually tracked and profiled (e.g. linear, saturated and non-linear profiles) so that an athlete's HRV fingerprint is known.

Last, the HRV response of Olympic and World Champion rowers in the 62-day build-up to their events was displayed. Importantly, during overload, HRV values increased (going above the SWC) before decreasing (to back within the SWC) as the event approached (during the taper). As such, it appears that for elite athletes, increases in HRV in the weeks before their event, during their highest training loads (Figure 15), are likely associated with a positive performance outcome. This may indicate an athlete is 'coping' with the applied training load and making positive adaptations. This finding was in agreement with other studies in elites, and athletes with extensive training histories (Iellamo et al., 2002).

In the final study (chapter 7), changes in HRV in response to training load and intensity were measured longitudinally in elite rowers. This is an important consideration as, practically speaking, knowing the HRV response to differences in load and intensity could assist training prescription to maximise adaptation or mitigate maladaptation. Furthermore, from studies 1 and 3, methodologies were adjusted so that changes in HRV might be more effectively tracked by the use of 7-day averaged values. In study 4, I gained a greater understanding of the typical desired responses of vagally-derived indices of HRV with training. Prior to this study, changes in HRV in response to training load and intensity in elite athletes were not well understood. The key findings from this study were that high-intensity training (greater than first lactate

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threshold) suppress HRV (ES = -0.20; 53% chance of lower values) whereas low-intensity training increases it (ES = 0.20; 51% chance of higher values). Furthermore, although effect sizes were trivial, moderate and small positive correlations between total training time ( $r = 0.37$  (0.28; 0.45)) and training load (0.24 (0.09; 0.40)) were observed. Findings from this study provide support for the polarised model of training (Laursen, 2010; S. Seiler, 2010), whereby athletes perform 75-80% of their training below the first lactate threshold, and approximately 10-20% of their training above the second lactate threshold (Steinacker et al., 2000; Steinacker, Lormes, Lehmann, & Altenburg, 1998). As such, large proportions of low-intensity training allow for the preservation of autonomic balance, thereby subsequently allowing high-intensity training to be performed optimally (Fiskerstrand & Seiler, 2004; S. Seiler et al., 2007). Practically speaking, during periods of overload (provided the training distribution is optimal), increases in HRV suggest that the athlete is 'coping' with the training load. Accordingly, when training loads are reduced (e.g. during a taper), reductions in HRV should be expected. Last, taking into account the slopes of the correlations, during exercise programming, ~5% increases in high-intensity training should be accompanied by ~6% increases in low-intensity training, so that autonomic balance is preserved.

### **8.2 Limitations of the research**

Consideration of the data in this thesis requires attention to the following limitations:

- (i) Although throughout the thesis the autonomic nervous system and cardiac parasympathetic activity are discussed, it was not assessed directly through nerve activity, but inferred indirectly via HRV. However, the measures needed to be practically applicable to obtain such data in elite athletes training 20-30 hours per week.

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- (ii) It is unethical to purposely induce overtraining or non-functional overreaching in elite athletes training for competitions. As such, with just one athlete suffering NFOR, it was impossible to draw definitive conclusions surrounding changes in HRV and its time course with likely maladaptation. Indeed, fatigue/NFOR through excessive training volume (glycogen depletion) and repeated sprint activities (neuromuscular fatigue) may not be accounted for by HRV. Furthermore, changes in autonomic function in response to high-volume vs. high-intensity are presumably different (Lehmann et al., 1998), with parasympathetic type (inappropriate high-volume training) and sympathetic type (inappropriate high-intensity training) overtraining being reported (Lehmann et al., 1998).
- (iii) Although this thesis is aimed at elite athletes, in study 2 I used data from recreational athletes to investigate the use of weekly-averaged values to evaluate positive adaptation. However, this study was purely methodological to support the findings from study 1. Furthermore, I developed this theory further in study 3 when combining the data with well-trained athletes.
- (iv) This thesis could be criticised for a lack of controlled, laboratory-based experimental studies (e.g. most are observational). However, the studies within the thesis needed to be practically applicable to coaches and sports scientist working in the field with elite athletes. As such, data from elite athletes building up to pinnacle events at the peak of their athletic career were used as my performance data. In such unique athletes, it is unrealistic to manipulate their training in an experimental fashion and disturb daily training regimes. Indeed, it would be expected that such athletes winning Olympic and World championship events do so with optimal build-ups to maximise performance.



### **8.3 Practical application and significance**

The findings of this thesis have a great deal of practical application for those wishing to use HRV to monitor training adaptation. Based on the findings of this thesis, the following section offers a step-by-step guide for using HRV as a tool to monitor training adaptation to endurance training regimes in well-trained and elite athletes, which was the overarching aim of this industry-supported PhD.

#### *Heart rate variability recordings*

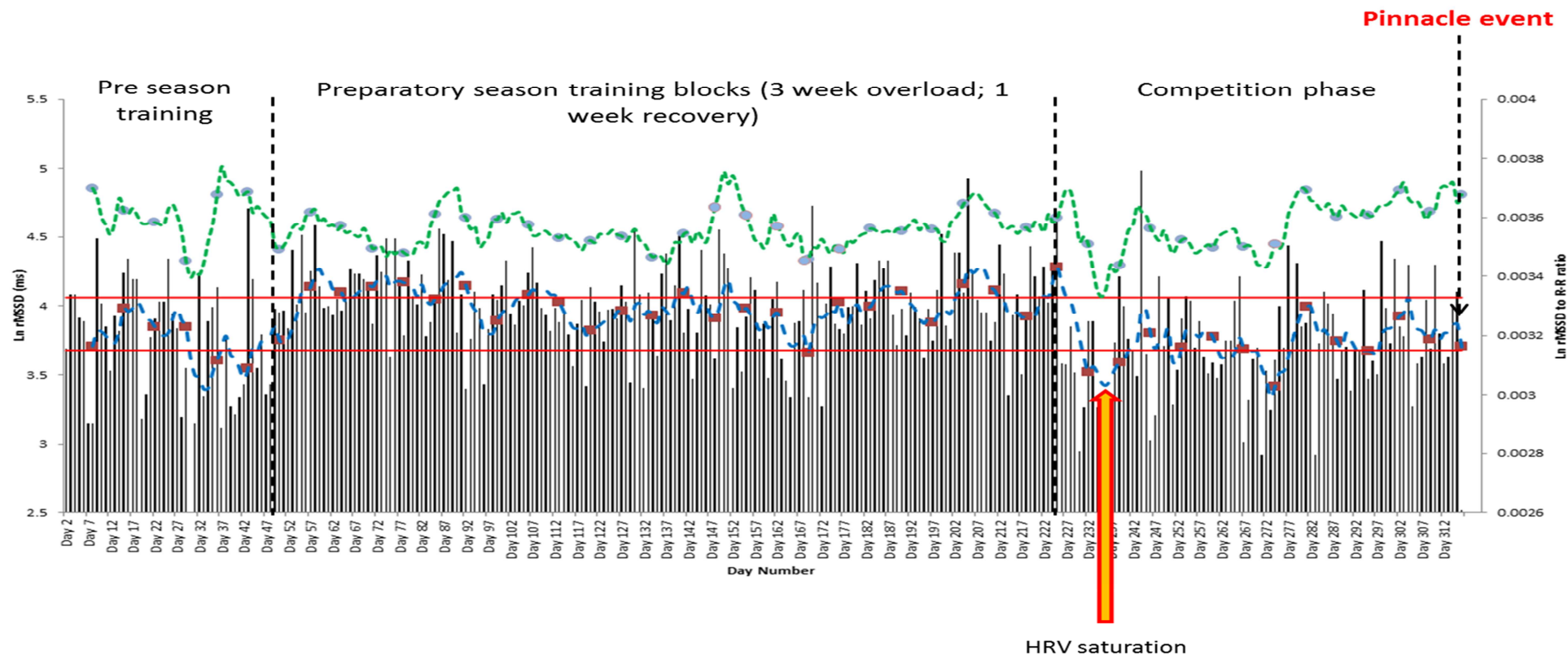
Throughout this thesis, I used the last 5 min of the 6 min daily morning resting HRV measurements to monitor training using Polar RS800cx watches. This is very easy to administer, and as such obtains good athlete compliance. For simplicity, I also restricted the HRV index to Ln rMSSD of the time domain analysis. As mentioned in chapter 6 (Plews, Laursen, Stanley, et al., 2013), the Ln rMSSD was preferred, as it is the most practically applicable HRV index for a number of reasons. First, it has the lowest coefficient variation of all HRV indices (Al Haddad et al., 2011). Second, Ln rMSSD is not significantly influenced by breathing frequency, unlike other spectral indices of HRV, and is therefore more suited to ambulatory measures (Penttila et al., 2001b). Third, Ln rMSSD can capture levels of parasympathetic activity over a short time frame, which is more convenient for athletes that have limited time to acquire a reading (Hamilton et al., 2004). Last, Ln rMSSD values can be easily calculated in MS Excel using R-R intervals (Aubert et al., 2003).

#### *Detecting meaningful change*

A large focus of this thesis was to uncover methods by which meaningful changes in HRV could be detected. In the studies that examined individual changes (e.g. studies 1 and 6), I based the smallest worthwhile change (SWC) on 0.5 of the

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coefficient variation at baseline (Hopkins et al., 2009). It is recommended that in order to establish the SWC, practitioners use two weeks of baseline measures, as this has been effective in these studies (Plews et al., 2012; Plews, Laursen, Stanley, et al., 2013). This should be recorded during a “normal” training load period, when the athlete is progressing as expected (e.g. performance during training is normal). HRV should then be monitored using a 7-day rolling average (or micro-cycle) with HRV values averaged at the end of the 7-day week or micro-cycle. As established, a minimum of 3 HRV recordings measured per week are required in trained athletes. As also mentioned, two consecutively averaged values below the SWC, alongside a continually reducing rolling average is likely a potential early warning sign of maladaptation, and the training program should be altered accordingly (during high training loads). Figure 18 provides a real example of how such a monitoring system can be practically applied in an athlete over various training cycles. The aforementioned should also be tracked alongside the averaged Ln rMSSD to R-R interval ratio (rolling and micro-cycle averaged), in order to track HRV saturation should it ensue. For clarification, a decrease in averaged Ln rMSSD values alongside decreases in the Ln rMSSD to R-R interval ratio (averaged) infers HRV saturation and training modifications are not likely required. Conversely, decreases in Ln rMSSD alongside increases in the Ln rMSSD to R-R interval ratio is likely to be a sign of maladaptation/fatigue and/or sympathetic over-activity and changes to the training program (rest or reduction in intensity) are recommended.



**Figure 18:** Example of HRV values in an elite rower through different phases of training in the 322 day build-up to winning a pinnacle event (2012 Olympic Games). The solid black bars represent the daily Ln rMSSD values. The solid horizontal red lines represent the individual smallest worthwhile change. The red squares are Ln rMSSD values averaged over 1 micro-cycle (in this case 7 days), and the blue dashed line represents the 4-day rolling average. The blue dots and green dashed line represents these same values respectively for the Ln rMSSD to R-R intervals. The yellow arrow indicates points of HRV saturation, demonstrating the importance of the concomitant use of these two indices (Ln rMSSD and Ln rMSSD to R-R interval ratio). Data were recorded using Polar RS800cx, and exported to excel. The above should be tracked alongside the Ln rMSSD to R-R interval ratio, using the same methodological approach.

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### *Taking into account the training phase*

As discovered in study 5, interpreting changes in HRV without taking into account the current training phase is somewhat meaningless. As such, during increases in training load (specifically when low-intensity training is increased) increases in HRV should be expected. Conversely, when high-intensity training is increased, reductions in HRV should be expected. This is particularly the case if low-intensity training is reduced concomitantly (e.g. during pre-race taper). Figure 19 presents a flow chart to provide guidelines for practitioners that wish to use HRV to monitor training adaptation in endurance athletes.

### *Other considerations*

Although this thesis focuses on the use of HRV alone to monitor the adaptations to endurance training, we must acknowledge the complexities of human physiology and training adaptation (Rivera-Brown & Frontera, 2012). Indeed, to monitor effectively, a more holistic approach is needed, whereby, HRV is monitored alongside other psychological (Pierce, 2002; Rushall, 1990; Watson et al., 1988) and performance metrics. This multifaceted approach allows programming decisions to be made around positive or negative adaptation to training.

## **8.4 Future research**

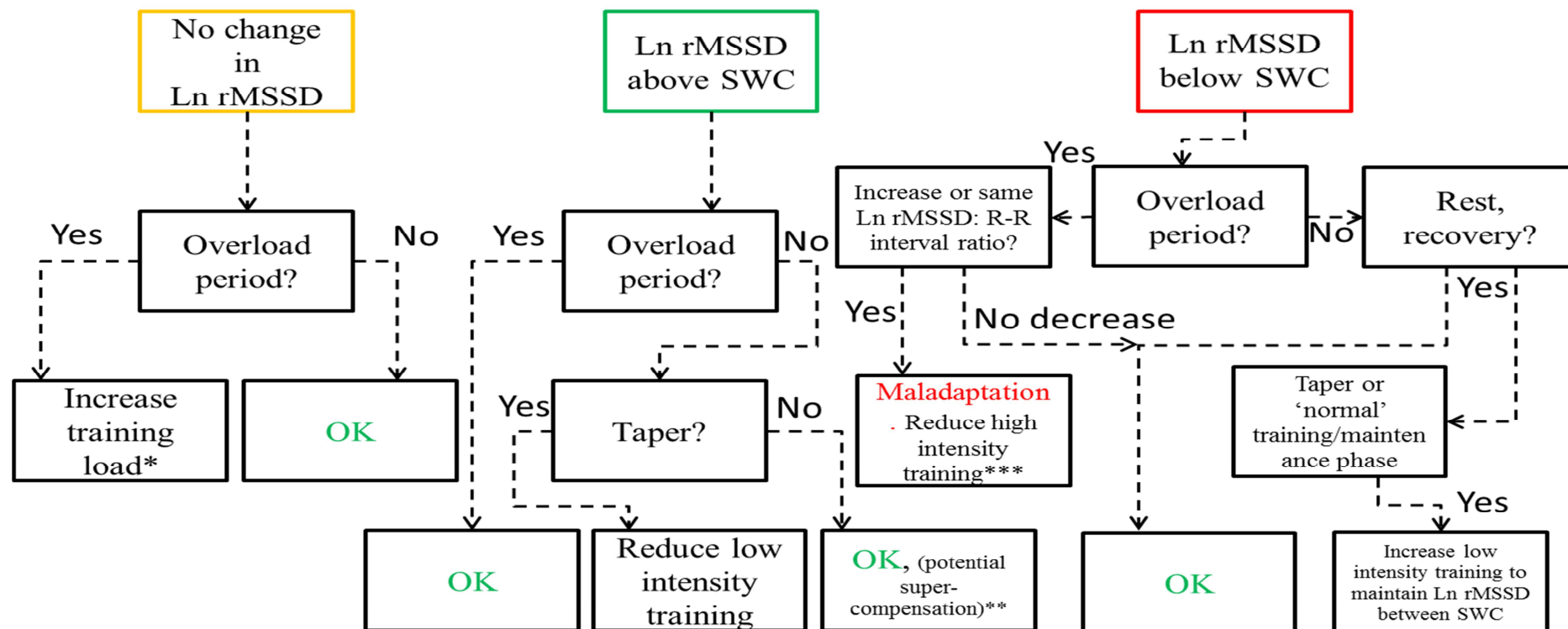
This thesis focused on the HRV response in athletes training for endurance sports (rowing and triathlon). However, little is known about the HRV responses in athletes training for strength and power sports (e.g. athletic throwing events, sprinting), where the adaptations made are known to be more peripheral than central. It is assumed that the HRV changes in accordance with positive and negative adaptation for such sports are different. Second, the sports focused on this thesis, although endurance-based,

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still had a high-intensity element. Likewise, little is known surrounding the HRV responses to ultra-endurance training for sports such as Ironman triathlon or ultra-distance running. Last, we know little around the time course of the changes in HRV values with subsequent maladaptation (e.g. exactly how many weeks an athlete can have HRV values below the SWC and not be considered to be NFOR). The answers to these questions will arise from properly planned longitudinal studies using HRV monitoring alongside repeated fitness testing.

### **8.5 Concluding remarks**

The aim of this Doctoral Thesis was to establish practical methods to assess training adaptation/responses using morning resting HRV measures. It is hoped that the research presented herein has made a significant contribution to the area of HRV and its application in athletes. These methods can be used by coaches and practitioners to monitor adaptation effectively throughout a training programme, so as to help maximise the performance outcome.



**Figure 19:** Flow chart to guide and monitor training adaptations based on morning resting HRV values (Ln rMSSD). \* The training load is unlikely high enough for that individual to make sufficient metabolic adaptations. Training load can be increased via volume or intensity. Low-intensity training is more effective at increasing Ln rMSSD. However using a polarised training distribution is recommended\*\*. Increases in Ln rMSSD during recovery phases of training are more expected in moderately trained athletes. \*\*\* Reductions in Ln rMSSD during overload periods are most likely due to inappropriate high-intensity training. However, overall load can also be reduced to mitigate Ln rMSSD reductions.

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## **CHAPTER TEN: APPENDIX**

# Participant Information Sheet



13<sup>th</sup> January 2011

## **Project Title**

Vagal-related indices as a tool to guide training in world class athletes

## **An invitation**

Hi, my name is Daniel Plews and I am a PhD student at AUT and Performance Physiologist for High Performance Sport New Zealand. Along with Adjunct. Prof Paul Laursen, Assoc. Prof. Andrew Kilding and Dr Martin Buchheit, I am inviting you to help with a project that examines how heart rate variability (the variation in the gap between heart beats) can be used to monitor the adaptation to training in elite rowers.

You are aware of heart rate variability as you have been collecting such data for some time. However, it is entirely your choice as to whether you would like to have your data further analysed and used as part of my PhD thesis. Furthermore you can withdraw your data from this research at any time should you wish to do so.

## **Purpose of this research?**

Training programs of elite athletes typically consist of periods of high training loads with limited periods of rest and recovery. Knowing when to recover and for how long in the elite athlete can therefore be difficult to determine. The right balance between training stimulus and rest and recovery is critical to ensure optimal positive adaptation occurs. Furthermore, rates of adaptation are different between individuals under the same training stimulus.

The beat-to-beat variation in resting pulse rates, or heart rate variability (HRV) may be useful tool to monitor individual training adaptation (e.g. is that person positively or negatively adapting to the current training load). The aim of this research is to design a method by which HRV can be effectively used to monitor training adaption in elite rowers. To do this we need to model and analyse HRV data that has been collected for a prolonged period.

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

As a world class athlete and carded athlete training at a High Performance centre, you have been invited to be part of this research.

However you will not be able to take part in this research if:

- ✓ *You have any known heart or cardiovascular condition or if a member of your family died below the age of 50 as a result of a heart condition*

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- ✓ *You have ever had an injury or medical condition that you think may affect your ability to sense pain or discomfort*
- ✓ *You have ever had persistent or regular back pain*
- ✓ *You are taking prescribed medication*
- ✓ *You have cultural or religious sensitivities about human body measurements*
- ✓ *You have any other reason to consider that you are not in good health and of average, or better than average fitness*
- ✓ *You are diabetic*

### **What will happen in this research?**

Your involvement in this research requires you to do nothing more than you currently do as part of your involvement in your high performance programme. Since HRV measures are critical to our investigations, we simply require you to continue to record HRV every morning upon waking as outlined below. We also invite you to give your consent to use your data as part of this research. However as mentioned previously this is entirely your decision.

#### *Heart Rate Variability Assessment*

HRV will be assessed every morning upon waking. It is advised that you leave both heart rate monitor watch and electrode strap by your bedside each evening to ensure minimum disturbances when applying the apparatus. To record HRV successfully follow these steps:

1. Apply the apparatus, lie still and ensure the heart rate monitor (HRM) is reading your heart rate (HR)\*
2. Wait for your HR to stabilise and then press start on the HRM
3. During this time it is very important you **lie still, don't talk** and ensure you're **breathing rate is stable**. Lie still for a duration of 8 min
4. After 8 min press "stop" on the HRM

\*Hint: If you find your HR is not reading leave some water next to your bed and wet the transmitter on the strap before putting it on.

### **What are the discomforts and risks?**

As there are potential conflicts of interest with me as the Rowing Physiologist working for HPSNZ, all the gathered data will be de-identified by a third party individual before being handed back to me for analysis as part of the PhD. This third party individual will be a professional whom is not part of AUT, HPSNZ or Rowing New Zealand.

### **What are the benefits?**

By recording HRV you will gain understanding into how your autonomic nervous system responds to the type of training. This will help you better understand how to guide your training in the future (e.g. when to increase or decrease the training load).

### **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 the data gained during this study will only be available to the researchers involved. If the data is published in the public domain your name as a subject will not be revealed and all subjects will remain anonymous.

**What are the costs of participating in this research?**

Every morning during your time training

**Will I receive feedback on the results of this research?**

As a rower recording HRV you will receive regular updates on your training adaptation and any significant finding of the research.

**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:

Name: Prof Paul Laursen

E-mail: paul.laursen@hpsnz.org.nz

Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTECH, Rosemary Godbold (rosemary.godbold.@aut.ac.nz).

**Whom do I contact for further information about this research?  
Researcher Contact Details:**

Name: Daniel Plews

E-mail: Daniel.plews@hpsnz.org.nz

**Project Supervisor Contact Details:**

Name: Prof Paul Laursen

E-mail: paul.laursen@hpsnz.org.nz

**Approved by the Auckland University of Technology Ethics Committee on 6 November, AUTECH Reference number 12/188.**

<h1>Consent Form</h1>	 <small>TE WĀNANGA ARONUI O TAMAKI MAKAU RAU</small>
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**Project title: The Practical Application of Heart Rate Variability to Monitor Training?**

**Project Supervisor:** Adjunct Prof Paul Laursen

**Researcher:** Daniel Plews

I acknowledge that I have undertaken the tests under contract of Rowing New Zealand (RNZ)

Yes ☐ No ☐

I acknowledge that any data which names me personally as an athlete will remain the property RNZ

Yes ☐ No ☐

I acknowledge that any of my data may be used for the purpose of research at AUT University and will be de-identified prior to analysis

Yes ☐ No ☐

I am agreeable to allow the use of my data for the purpose of research, including journal publications and post-graduate thesis

Yes ☐ No ☐

I understand that any de-identified data will be held for the purpose of research only (by the named researcher and supervisor) for a period of three years

Yes ☐ No ☐

Participants signature:

.....

Participants Name:

.....

Participants contact (if appropriate):

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

Date:

*Note: The Participant should retain a copy of this form.*

## Chapter Ten



AUTEC  
SECRETARIAT

6 November 2012

Paul Laursen  
Faculty of Health and Environmental Sciences

Dear Paul

Re: **12/188 Vagal-related indices as a tool to guide training in world class athletes.**

Thank you for providing evidence as requested. I am pleased to advise that it satisfies the points raised by the Auckland University of Technology Ethics Committee (AUTEC). I have approved your ethics application for three years until 5 November 2015.

As part of the ethics approval process, you are required to submit the following to AUTEC:

- A brief annual progress report using form EA2, which is available online through <http://www.aut.ac.nz/research/research-ethics/ethics>. When necessary this form may also be used to request an extension of the approval at least one month prior to its expiry on 5 November 2015;
- A brief report on the status of the project using form EA3, which is available online through <http://www.aut.ac.nz/research/research-ethics/ethics>. This report is to be submitted either when the approval expires on 5 November 2015 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. If your research is undertaken within a jurisdiction outside New Zealand, you will need to make the arrangements necessary to meet the legal and ethical requirements that apply within their.

To enable us to provide you with efficient service, we ask that you 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](mailto:ethics@aut.ac.nz).

All the very best with your research,

Dr Rosemary Godbold  
Executive Secretary

**Auckland University of Technology Ethics Committee**

CC: Daniel Plews [daniel.plews@hpsnz.org.nz](mailto:daniel.plews@hpsnz.org.nz), Andrew Kilding

## Heart rate variability in elite triathletes, is variation in variability the key to effective training? A case comparison

Daniel J. Plews · Paul B. Laursen ·  
Andrew E. Kilding · Martin BuchheitReceived: 6 October 2011 / Accepted: 9 February 2012  
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**Abstract** Measures of an athlete's heart rate variability (HRV) have shown potential to be of use in the prescription of training. However, little data exists on elite athletes who are regularly exposed to high training loads. This case study monitored daily HRV in two elite triathletes (one male: 22 year,  $\dot{V}O_{2\max}$  72.5 ml kg min<sup>-1</sup>; one female: 20 year,  $\dot{V}O_{2\max}$  68.2 ml kg min<sup>-1</sup>) training 23 ± 2 h per week, over a 77-day period. During this period, one athlete performed poorly in a key triathlon event, was diagnosed as non-functionally over-reached (NFOR) and subsequently reactivated the dormant virus herpes zoster (shingles). The 7-day rolling average of the log-transformed square root of the mean sum of the squared differences between R-R intervals (Ln rMSSD), declined towards the day of triathlon event (slope = -0.17 ms/week;  $r^2 = -0.88$ ) in the NFOR athlete, remaining stable in the control (slope = 0.01 ms/week;  $r^2 = 0.12$ ). Furthermore, in the NFOR athlete, coefficient of variation of HRV (CV of Ln rMSSD 7-day rolling average) revealed large linear reductions towards NFOR (i.e., linear regression of HRV variables versus day number towards NFOR: -0.65%/week and

$r^2 = -0.48$ ), while these variables remained stable for the control athlete (slope = 0.04%/week). These data suggest that trends in both absolute HRV values and day-to-day variations may be useful measurements indicative of the progression towards mal-adaptation or non-functional over-reaching.

**Keywords** Over-training · Monitoring · Cardiac parasympathetic function · Elite triathletes

## Introduction

Training programs of elite athletes typically consist of periods of high training loads with limited periods of rest and recovery (Fiskerstrand and Seiler 2004; Laursen 2010; Seiler 2010). Knowing when to recover and for how long to do so in elite athletes can therefore be difficult. Over-reaching (OR), non-functional over-reaching (NFOR) and over-training (OT) are terms often used to describe a stress-regeneration imbalance, with negative outcomes, such as hormonal changes (Meeusen et al. 2004), disturbed sleep, increased levels of fatigue and reductions in performance commonly reported (Meeusen et al. 2006). While short-term OR is typically an important component of the elite training cycle, prolonged OR pushes an athlete into NFOR or OT, which results in performance impairment and possible negative health consequences (Israel 1976). However, where the point of transition between OR and NFOR/OT has been unsuccessfully sought by researchers and practitioners for decades (Barron et al. 1985; Morgan et al. 1987).

One of the difficulties faced by practitioners attempting to solve this problem is that the symptoms of OR and NFOR are similar, and not necessarily more severe than for OR (Halson and Jeukendrup 2004). For example

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## Evaluating Training Adaptation With Heart-Rate Measures: A Methodological Comparison

Daniel J. Plews, Paul B. Laursen, Andrew E. Kilding, and Martin Buchheit

The aim of this study was to compare 2 different methodological assessments when analyzing the relationship between performance and heart-rate (HR) -derived indices (resting HR [RHR] and HR variability [HRV]) to evaluate positive adaptation to training. The relative change in estimated maximum aerobic speed (MAS) and 10-km-running performance was correlated to the relative change in RHR and the natural logarithm of the square root of the mean sum of the squared differences between R-R intervals on an isolated day (RHR<sub>day</sub>; Ln rMSSD<sub>day</sub>) or when averaged over 1 wk (RHR<sub>week</sub>; Ln rMSSD<sub>week</sub>) in 10 runners who responded to a 9-wk training intervention. Moderate and small correlations existed between changes in MAS and 10-km-running performance and RHR<sub>day</sub> ( $r = .35$ , 90%CI [-.35, .76] and  $r = -.21$  [-.68, .39]), compared with large and very large correlations for RHR<sub>week</sub> ( $r = -.62$  [-.87, -.11] and  $r = .73$  [.30, .91]). While a trivial correlation was observed for MAS vs Ln rMSSD<sub>day</sub> ( $r = -.06$  [-.59, .51]), a very large correlation existed with Ln rMSSD<sub>week</sub> ( $r = .72$  [.28, .91]). Similarly, changes in 10-km-running performance revealed a small correlation with Ln rMSSD<sub>day</sub> ( $r = -.17$  [-.66, .42]), vs a very large correlation for Ln rMSSD<sub>week</sub> ( $r = -.76$  [-.92, -.36]). In conclusion, the averaging of RHR and HRV values over a 1-wk period appears to be a superior method for evaluating positive adaption to training compared with assessing its value on a single isolated day.

**Keywords:** monitoring, cardiac parasympathetic function, heart-rate variability

Athletes, coaches, and sport science practitioners seek useful methods to monitor individual adaptation or maladaptation to training. It has been shown that heart-rate (HR) -derived indices such as morning resting HR (RHR) and HR variability (HRV) decrease and increase, respectively, after endurance-training regimens and may therefore represent an easy-to-use, noninvasive means by which to assess individual adaptation to endurance training.<sup>1</sup>

During the monitoring of an elite triathlete who developed signs of maladaptation to training, we recently identified a number of methodological issues associated with determining adaptation using isolated (ie, single-day) RHR and HRV readings.<sup>2</sup> Factors such as noise, temperature, light,<sup>3</sup> and prior exercise<sup>4</sup> can affect HRV on any given day. In the former article,<sup>2</sup> we showed that RHR and HRV data, displayed as rolling or weekly averages, appeared to provide a more consistent representation of the changes in fatigue and maladaptation. As suggested previously, the use of isolated daily

values to measure changes in HRV has likely led to the equivocal findings reported throughout the HRV training literature.<sup>2</sup>

Given the potential of this alternative methodological approach, we revisited the RHR and HRV data taken from recreationally trained runners who responded positively to a 9-week training intervention.<sup>5</sup> To assess the best practical methodological approach to analyze positive adaptation to a training intervention, we compared the relationship between isolated versus weekly-averaged RHR and HRV values and running performance.

### Methods

#### Participants

Out of the 14 runners described in the original study,<sup>5</sup> 10 of the positive responders (mass  $75.6 \pm 7.4$ , estimated maximum aerobic speed [MAS]  $17.3 \pm 1.7$  km/h, 10-km  $48:34 \pm 7:45$  min:s) were selected for further assessment to allow fair comparison between 2 different methodological approaches. Only 10 responders were included for further analysis (compared with  $n = 11$  in the original paper<sup>5</sup>), due to limited availability of data in the final week of the training intervention for 1 subject, which would make comparison between single-day and weekly-averaged values invalid.

Plews and Laursen are with High Performance Sport New Zealand, Auckland, New Zealand. Kilding is with Sport Performance Research Inst New Zealand, Auckland University of Technology, Auckland, New Zealand. Buchheit is with the Football Performance and Science Dept, Aspire Academy for Sports Excellence, Doha, Qatar.

Plews DJ, Laursen PB, Kilding AE, Buchheit M (2013) Evaluating Training Adaptation with Heart Rate Measures: A Methodological Comparison. International Journal of Sports Physiology and Performance. Epub ahead of Print.



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## CURRENT OPINION

## Training Adaptation and Heart Rate Variability in Elite Endurance Athletes: Opening the Door to Effective Monitoring

Daniel J. Plews · Paul B. Laursen · Jamie Stanley ·  
Andrew E. Kilding · Martin Buchheit

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© Springer International Publishing Switzerland 2013

**Abstract** The measurement of heart rate variability (HRV) is often considered a convenient non-invasive assessment tool for monitoring individual adaptation to training. Decreases and increases in vagal-derived indices of HRV have been suggested to indicate negative and positive adaptations, respectively, to endurance training regimens. However, much of the research in this area has involved recreational and well-trained athletes, with the small number of studies conducted in elite athletes revealing equivocal outcomes. For example, in elite athletes, studies have revealed both increases and decreases in HRV to be associated with negative adaptation. Additionally, signs of positive adaptation, such as increases in cardiorespiratory fitness, have been observed with atypical

concomitant decreases in HRV. As such, practical ways by which HRV can be used to monitor training status in elites are yet to be established. This article addresses the current literature that has assessed changes in HRV in response to training loads and the likely positive and negative adaptations shown. We reveal limitations with respect to how the measurement of HRV has been interpreted to assess positive and negative adaptation to endurance training regimens and subsequent physical performance. We offer solutions to some of the methodological issues associated with using HRV as a day-to-day monitoring tool. These include the use of appropriate averaging techniques, and the use of specific HRV indices to overcome the issue of HRV saturation in elite athletes (i.e., reductions in HRV despite decreases in resting heart rate). Finally, we provide examples in Olympic and World Champion athletes showing how these indices can be practically applied to assess training status and readiness to perform in the period leading up to a pinnacle event. The paper reveals how longitudinal HRV monitoring in elites is required to understand their unique individual HRV fingerprint. For the first time, we demonstrate how increases and decreases in HRV relate to changes in fitness and freshness, respectively, in elite athletes.

**Electronic supplementary material** The online version of this article (doi:10.1007/s40279-013-0071-8) contains supplementary material, which is available to authorized users.

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### 1 Introduction

One of the more promising methods to monitor individual adaptation to training involves the regular monitoring of cardiac autonomic nervous system (ANS) status, through the measurement of resting or post-exercise heart rate variability (HRV) [3–5]. Indeed, non-functional overreaching (NFOR) and/or negative adaptation to training is thought to be generally associated with reductions in vagal-

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Plews DJ, Laursen PB, Stanley J, Kilding AE, Buchheit M (2013b) Training adaptation and heart rate variability in elite endurance athletes - opening the door to effective monitoring. Sports Med 49 (9):773-781.

## Chapter Ten

"Monitoring Training With Heart Rate Variability: How Much Compliance is Needed for Valid Assessment?"  
by Plews DJ et al.  
*International Journal of Sports Physiology and Performance*  
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*Note.* This article will be published in a forthcoming issue of the *International Journal of Sports Physiology and Performance*. The article appears here in its accepted, peer-reviewed form, as it was provided by the submitting author. It has not been copyedited, proofread, or formatted by the publisher.

### Section: Original Report

**Article Title:** Monitoring Training With Heart Rate Variability: How Much Compliance is Needed for Valid Assessment?

**Authors:** Daniel J. Plews<sup>1,2</sup>, Paul B. Laursen<sup>1,2</sup>, Yann Le Meur<sup>3</sup>, Christophe Hausswirth<sup>3</sup>, Andrew E. Kilding<sup>2</sup> and Martin Buchheit<sup>4</sup>

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**Journal:** *International Journal of Sports Physiology and Performance*

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## Chapter Ten

"Heart Rate Variability and Training Intensity Distribution in Elite Rowers"  
by Plews DJ, Laursen PB, Kilding AE, Buchheit M  
*International Journal of Sports Physiology and Performance*  
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**Section:** Original Investigation

**Article Title:** Heart Rate Variability and Training Intensity Distribution in Elite Rowers

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