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**QUANTIFYING TRAINING LOAD
AND ITS RELATIONSHIP
TO HEART RATE RECOVERY**

by

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Dedicated to my mom,

*Whose unconditional love, support and
sacrifice has allowed me to achieve my goals.*

*Your strength, zest for life, integrity,
patience and kindness are my inspiration.*

You mean the world to me!

University of Cape Town

TABLE OF CONTENTS

ACKNOWLEDGEMENTS	6
DECLARATION	7
ACADEMIC PUBLICATIONS	8
THESIS ABSTRACT	9
 CHAPTER 1: LITERATURE REVIEW 	
1.1 INTRODUCTION	14
1.2 QUANTIFYING TRAINING LOAD	16
1.2.1 Questionnaires and diaries	16
1.2.2 Physiological measures	18
1.2.2.1 Oxygen consumption.....	18
1.2.2.2 Lactate.....	20
1.2.2.3 Heart rate.....	21
1.2.3 Rating of Perceived Exertion	23
1.2.4 Indices of training stress	24
1.2.4.1 Training Impulse.....	24
1.2.4.2 Summated Heart Rate Zones.....	25
1.2.4.3 Lucia's TRIMP.....	26
1.2.4.4 Session RPE.....	26
1.2.5 Direct observation	28
1.3 MODELLING THE RELATIONSHIP BETWEEN TRAINING AND PERFORMANCE	30
1.4 MONITORING TRAINING ADAPTATIONS AND RESPONSE TO TRAINING	36
1.4.1 Physiological adaptations to endurance training	37
1.4.1.1 VO ₂ max, Exercise economy, Lactate & Ventilatory thresholds.....	38
1.4.1.2 Lactate.....	38
1.4.1.3 Testosterone and cortisol.....	39
1.4.1.4 Skeletal muscle.....	40
1.4.1.5 Iron status.....	41
1.4.1.6 Summary.....	42
1.4.2 Fatigue	43

1.4.2.1 Recovery.....	43
1.4.2.2 Muscle pain.....	43
1.4.2.3 Profile of Mood States.....	45
1.4.3 Using heart rate to monitor training response.....	46
1.4.3.1 Resting heart rate.....	46
1.4.3.2 Submaximal heart rate.....	48
1.4.3.3 Maximal heart rate.....	48
1.4.3.4 Heart rate variability.....	49
1.4.3.5 Heart rate recovery.....	53
1.5 SUMMARY OF LITERATURE REVIEW.....	59
1.6 OBJECTIVES OF THIS THESIS.....	61

CHAPTER 2: VALIDITY OF SELF-REPORTED TRAINING DURATION

2.1 INTRODUCTION.....	64
2.2 METHODS.....	65
2.2.1 Study Design.....	65
2.2.2 Study Protocol.....	65
2.2.2.1 Anthropometric and physiologic measurements.....	65
2.2.2.2 Exercise monitoring and assessment of training duration.....	66
2.2.2.3 Division of subjects into groups.....	67
2.2.3 Statistical Analyses.....	67
2.3 RESULTS.....	68
2.4 DISCUSSION.....	70
2.5 CONCLUSIONS.....	72

CHAPTER 3: QUANTIFYING TRAINING LOAD - A COMPARISON OF SUBJECTIVE AND OBJECTIVE METHODS

3.1 INTRODUCTION.....	74
3.2 METHODS.....	75
3.2.1 Study Design and Subjects.....	75
3.2.2 Methodology.....	76
3.2.2.1 Anthropometric and physiologic measurements.....	76
3.2.2.2 Quantification of training load.....	76

3.2.3 Statistical Analyses.....	78
3.3 RESULTS.....	79
3.3.1 TRIMP vs. Summated Heart Rate Zones Method.....	79
3.3.2 TRIMP vs. Session RPE Method.....	80
3.3.3 Summated Heart Rate Zone Method vs. Session RPE Method.....	83
3.4 DISCUSSION.....	86
3.5 CONCLUSIONS.....	91

**CHAPTER 4: THE RELATIONSHIP BETWEEN TRAINING LOAD
AND HEART RATE RECOVERY**

4.1 INTRODUCTION.....	94
4.2 METHODS.....	97
4.2.1 Study Design and Subjects.....	97
4.2.2 Methodology.....	98
4.2.2.1 Body composition and physiological measurements.....	98
4.2.2.2 Protocol.....	99
4.2.2.3 Quantification of training load.....	99
4.2.2.4 Heart Rate Interval Monitoring System Test.....	100
4.2.2.5 Quantification of percent heart rate recovery.....	101
4.2.3 Statistical Analyses.....	102
4.3 RESULTS.....	102
4.3.1 Subject characteristics.....	102
4.3.2 Relationship between TRIMP week 1 and TRIMP week 2.....	103
4.3.3 Relationship between Training load and Heart rate recovery.....	104
4.3.4 Relationship between VO_2 max and heart rate recovery.....	106
4.3.5 Changes in heart rate recovery from HIMS 1 to HIMS 2.....	106
4.3.6 Repeatability of HIMS Stage 4 heart rate.....	108
4.4 DISCUSSION.....	109
4.4.1 Relationship between Training load and Heart rate recovery.....	109
4.4.2 Changes in heart rate recovery from HIMS 1 to HIMS 2.....	110
4.4.3 Repeatability of HIMS Stage 4 heart rate.....	112

**CHAPTER 5: THE EFFECT OF ENDURANCE TRAINING
ON HEART RATE RECOVERY**

5.1 INTRODUCTION	116
5.2 10 km RUNNING PROGRAMME	118
5.2.1 METHODS	118
5.2.1.1 Study design and participants	118
5.2.1.2 Methodology	118
5.2.1.2.1 Body composition and physiological measurements.....	118
5.2.1.2.2 10 km Running Programme.....	119
5.2.1.2.3 Daily records.....	120
5.2.1.2.4 HIMS Tests.....	121
5.2.1.2.5 Outcomes.....	121
5.2.1.3 Data analyses	123
5.2.2 RESULTS	124
5.2.2.1 Group Results	124
5.2.2.1.1 Physiology and performance.....	124
5.2.2.1.2 Relationship between TRIMP and heart rate recovery.....	124
5.2.2.2 Individual Results	127
5.2.2.2.1 CASE STUDY 1.....	127
5.2.2.2.2 CASE STUDY 2.....	131
5.2.2.2.3 CASE STUDY 3.....	135
5.2.2.2.4 CASE STUDY 4.....	139
5.2.2.2.5 CASE STUDY 5.....	143
5.2.2.2.6 CASE STUDY 6.....	147
5.2.2.2.7 CASE STUDY 7.....	151
5.2.2.2.8 CASE STUDY 8.....	155
5.2.2.2.9 CASE STUDY 9.....	159
5.2.2.2.10 CASE STUDY 10.....	163
5.2.3 DISCUSSION	167
5.2.3.1 Group Results	167
5.2.3.1.1 Physiology and performance.....	167
5.2.3.1.2 Relationship between TRIMP and heart rate recovery.....	168
5.2.3.2 Individual Results	169
5.2.3.2.1 Physiological and performance changes with training.....	169
5.2.3.2.2 Individual response to training.....	170

5.2.3.2.3 Monotony.....	172
5.3 IRONMAN.....	174
5.3.1 METHODS.....	174
5.3.1.1 Participant.....	174
5.3.1.2 Methodology.....	174
5.3.1.2.1 Body composition and physiological measurements.....	174
5.3.1.2.2 Ironman training programme.....	175
5.3.1.2.3 Daily records.....	175
5.3.1.2.4 HIMS Tests.....	175
5.3.1.2.5 Outcomes.....	176
5.3.2 RESULTS.....	176
5.3.3 DISCUSSION.....	180
5.4 SUMMARY AND CONCLUSIONS.....	181
CHAPTER 6: THESIS SUMMARY AND CONCLUSIONS.....	183
CHAPTER 7: REFERENCES.....	197
CHAPTER 8: APPENDICES.....	219
1 Equations used to quantify exercise intensity and to model the relationship between training and performance.....	220
2 Borg 6-20 Rating of Perceived Exertion (RPE) Scale.....	221
3 Borg Category Ratio (CR-10) Scale and Session RPE Scale.....	222
4 Total Quality of Recovery (TQR) Scale.....	223
5 Scoring for Action Recovery.....	224
6 Muscle Soreness Assessment Analogue Scale.....	225
7 Results from studies investigating the effect of training on heart rate variability, showing the disparity in findings, methodology of recording HRV and prescribed training programmes.....	226
8 Modified Physical Activity Readiness Questionnaire.....	227
9 Injury and Illness Report.....	228
LIST OF TABLES.....	229
LIST OF FIGURES.....	231

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DECLARATION

I, Jill Borresen, do hereby declare that the research presented in this thesis is my own unaided work, both in concept and execution, and apart from the normal guidance from my supervisor I have received no other assistance. Neither the substance nor any part of this thesis has been submitted in the past, or is being, or is to be submitted for a degree in this or any other University.

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This thesis is presented for examination in fulfilment of the requirements for the degree of Doctor of Philosophy.

Signature: _____

Date: _____

ACADEMIC PUBLICATIONS

Autonomic control of heart rate during and after exercise – measurements and implications for monitoring training status. *Sports Medicine* (in press).

Quantifying training load – A comparison of subjective and objective methods. J Borresen, M Lambert. *International Journal of Sports Physiology and Performance* 3(1): 16 - 30, 2008.

Changes in heart rate recovery in response to acute changes in training load. J Borresen, M Lambert. *European Journal of Applied Physiology* 101: 503 - 511, 2007.

Validity of self-reported training duration. J Borresen, M Lambert. *International Journal of Sports Science and Coaching* 1(4): 353 - 359, 2006.

A theoretical basis of monitoring fatigue: a practical approach for coaches. M Lambert, J Borresen. *International Journal of Sports Science and Coaching* 1(4): 371 – 388, 2006.

CONFERENCE PRESENTATIONS

The 2nd Clinical Sports Medicine Conference, Cape Town, 4-6 October 2006. “The theoretical basis of using heart rate recovery in monitoring training status”.

THESIS ABSTRACT

Scientific research is playing an increasingly important role in the development of optimal exercise training programmes that meet specific goals within specified times. Improving the accuracy of training prescription first involves quantifying what the athlete is currently doing. Secondly, it needs to be established whether or not the athlete is adapting favourably to the training programme. This thesis investigated current methods of quantifying training load, and proposed the use of heart rate recovery to monitor the physiological response to training.

The quantification of exercise training may involve athletes self-reporting their exercise. The first study of this thesis investigated the relationship between what athletes say they do in training and what they actually do. Twenty-nine physically active participants (12 male, 17 female) self-reported their average weekly training duration for 3 weeks of *ad libitum* training. During the following 2 weeks exercise duration was recorded. There was a significant relationship ($r = 0.87$) between self-reported and recorded duration, with 24% of athletes over-estimating, 17% under-estimating and 59% accurately estimating their training duration. The margin of error between self-reported and actual training duration may affect the prescription of training if only self-reported data is used to quantify training. It is therefore recommended that this error be accounted for in research and coaching, or where possible physiological measurements be used to corroborate self-reported data.

The second study investigated the relationship between three popular indices for quantifying internal training stress: Session RPE (subjective method); and Training Impulse (TRIMP) and the Summated Heart Rate Zones method (objective methods). Thirty-three participants trained *ad libitum* for two weeks, during which time heart rate, duration and RPE were recorded and used to calculate training load using the three methods. Participants were divided into groups based on whether the regression equations over- (OVER group), under- (UNDER group) or accurately (ACCURATE group) predicted the relationship between the three methods. Training load calculated using TRIMP correlated best with the Summated Heart Rate Zone method ($r = 0.98$) (both heart rate-based equations). The strong correlation suggests that both methods may be suitable for quantifying continuous as well as interval

training sessions. A correlation of $r = 0.76$ occurred between TRIMP and Session RPE training load. When investigating possible characteristics that might explain the variance not accounted for in this relationship it was found that the OVER group spent a greater percentage of training time in heart rate zone 4 (80 - 90%HRmax) than UNDER (46 ± 8 vs. $25 \pm 10\%$ (mean \pm SD), $p = 0.008$). UNDER spent a greater percentage of training time in heart rate zone 1 (50 - 60%HRmax) than OVER (15 ± 8 vs. $3 \pm 3\%$, $p = 0.005$) and ACCURATE (15 ± 8 vs. $5 \pm 3\%$, $p = 0.020$) and more time in heart rate zone 2 (60 - 70%HRmax) than OVER (17 ± 6 vs. $7 \pm 6\%$, $p = 0.039$). A correlation of $r = 0.84$ occurred between the Summated Heart Rate Zones method and Session RPE training load. This comparison also revealed that OVER spent proportionally more training time in heart rate zone 4 than UNDER ($45 \pm 8\%$ vs. $25 \pm 10\%$, $p = 0.018$). UNDER had a lower training heart rate than ACCURATE (132 ± 10 vs. $148 \pm 12 \text{ b}\cdot\text{min}^{-1}$, $p = 0.048$) and spent more time in heart rate zone 1 than OVER (15 ± 8 vs. $4 \pm 3\%$, $p = 0.013$) and ACCURATE (15 ± 8 vs. $5 \pm 3\%$, $p = 0.015$). This study concluded that the subjective Session RPE method remains useful in providing reasonable assessments of training load compared to the objective heart rate-based methods, however the methods deviate in their assessments when proportionally more time is spent training at either very low or high intensities. Since Session RPE is a more global indication of the difficulty of an exercise bout, the variance may represent numerous extraneous factors that contribute towards an individual's personal perception of the difficulty of the session. Conversely, since neither of the objective methods have been validated, it cannot be ruled out that there may be inherent flaws in these equations that may affect their relationship with the Session RPE method.

The third study proposed that the measurement of heart rate recovery, an indirect marker of autonomic function, may offer a practical way of quantifying the physiological effects of training. The relationship between training load (TRIMP) and percent heart rate recovery (HRr%) after a standardized submaximal running (Heart rate Interval Monitoring System: HIMS) test was investigated in 11 participants that kept their training load constant over a 2-week period. This study also assessed whether HRr% changed in response to acute changes in training load from one week to the next. Twenty-eight men and women (mean age 30 ± 5 years) trained *ad libitum* for two weeks during which time heart rate and duration were recorded to calculate TRIMP. Participants were grouped based on whether they increased

(Group I, n = 9), decreased (Group D, n = 8) or kept their training load constant (Group S, n = 11) from week 1 to week 2. At the end of each week the participants completed a HIMS test. Changes in heart rate at the end of the test and HRr% from week 1 to week 2 were compared between groups. A significant inverse relationship was found between average weekly TRIMP and HRr% in participants that kept their training load the same over two weeks ($r = -0.61$). However no correlation was found between $VO_2\text{max}$ and HRr%. Since heart rate recovery relates fairly well to training load, it may provide information about the effects of habitual training loads on autonomic nervous system function, rather than being a measure of cardiorespiratory adaptation. With regard to acute changes in HRr% in response to changes in training load, Group I had a slower HRr% and Group D tended to have a slightly faster HRr% after HIMS 2 than after HIMS 1 (mean percent change $5.6 \pm 8.7\%$ vs. $-2.6 \pm 3.9\%$, $p = 0.03$). Thus a negative effect on heart rate recovery was observed with increases in training load. Submaximal heart rate at the end of the HIMS tests was not affected by acute changes in training load. Whereas heart rate during exercise measures cardiac load, heart rate recovery may reflect the state of the autonomic nervous system, indicating the body's capacity to respond to exercise.

The fourth study expanded on the findings of Chapter 4, with investigations into the chronic response of heart rate recovery to endurance training. Ten previously sedentary females (age range 21 to 47 years) participated in a running programme that provided training guidance towards running a 10 km road race at the end of 12 weeks. Heart rate, Session RPE and training duration were recorded and TRIMP was calculated for every training session. Muscle soreness and total quality of recovery were assessed daily. Every 2 weeks the participants completed a HIMS test, after which heart rate recovery and submaximal heart rate were calculated. Performance was assessed before and after the 12 weeks using time, total number of heart beats and average heart rate during a 2.4 km run/walk assessment. Twelve weeks of endurance running training significantly improved 2.4 km time (18.5 ± 1.8 vs. 16.5 ± 2.3 min, $p = 0.025$) and 1-minute heart rate recovery (34 ± 8 vs. 48 ± 9 beats, $p = 0.004$), and reduced the number of heart beats during the 2.4 km assessment (2944 ± 488 vs. 2583 ± 402 beats, $p = 0.022$) as well as submaximal heart rate (184 ± 13 vs. 176 ± 11 $\text{b}\cdot\text{min}^{-1}$, $p = 0.00004$). When data from all 10 participants were pooled to assess the relationship between 2-weekly TRIMP (irrespective of changes in training load between 2-week periods) and heart rate

recovery, a correlation of $r = -0.15$ was found. When data were divided into groups based on whether TRIMP increased, decreased or remained constant between successive 2-week periods, the mean percent change in HRr% was also not different between groups. Although performance for the group improved after the completion of the programme, each participant's physiological response was different. Some participants responded well to relatively low training loads where others responded well to much higher training loads. It also appeared as though there were "slow responders" and "fast responders" to the programme, highlighting the variability in the way individuals respond to exercise and how this is translated into changes in performance. The relationship between training load and heart rate recovery may also be affected by the phase of training. Thus, each individual's training load threshold and physiological response needs to be identified in order to optimise training adaptation.

In conclusion, this thesis found that the margin of error between self-reported and actual training data should be accounted for in research and coaching, or where possible physiological measurements should be used to corroborate self-reported data. Further research is required to establish the exact cause of the relatively poor correlation between objective and subjective methods of quantifying training load as this may have important implications for their accuracy in quantifying internal training stress. The relationship between heart rate recovery and training load was also investigated, along with the potential of using heart rate recovery to monitor the physiological response to training. The measurement of acute and chronic changes in heart rate recovery may contribute to an understanding of positive and negative training effects on autonomic nervous system function, which may provide important information in the prescription of training. The highly personal physiological response to training and the subsequent effect on performance suggests that a quantifiable relationship between training load and heart rate recovery needs to be established on an individual basis. In addition, since there are many factors that influence an individual's tolerance of exercise, it is recommended that a holistic view be taken in monitoring training adaptations.

CHAPTER 1

<p>LITERATURE REVIEW</p>

Two review articles containing information included in this chapter have been accepted for publication:

Autonomic control of heart rate during and after exercise – measurements and implications for monitoring training status. J Borresen, M Lambert.

Sports Medicine (in press).

A theoretical basis of monitoring fatigue: a practical approach for coaches.

M Lambert, J Borresen. *International Journal of Sports Science*

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1.1 INTRODUCTION

The ultimate goal of any sports coach and athlete is to produce a winning or personal best performance at a specific time, preferably in competition. The prescription of the training required to achieve this goal has historically been largely instinctive, resulting from years of personal coaching experience. As such the ability to achieve peak fitness and performance coinciding with dates of competition is met with varying degrees of success. It is generally believed that increasing training will result in improvements in sporting performance and physical well-being. However, although widely accepted, this vague approach to prescribing training may be tenuous, especially since random increases in training volume may also increase the likelihood of injury and symptoms of overtraining^{1:2a}. The role of scientific research in this process is becoming more important in order to prescribe optimal training programmes that prevent both under training and overtraining and increase the chance of achieving desired performances.

The frequency, duration and intensity of exercise all contribute to the nature and magnitude of the training effect^{4:5}. However, relatively little research has been conducted into the quantification of training programmes and their effects on physiological adaptation and subsequent performance. This is surprising since peaking for sporting performance requires an understanding of the quantifiable effects of training on performance so that optimal training and rest regimens may be planned in preparation for the event. Attempts have been made to model the effects of training on performance so that performance can be predicted. Some of these theoretical models will be discussed briefly in this literature review but a detailed study thereof is beyond the scope of this thesis. It does however present the context in which this thesis exists, since the research conducted for this thesis has the

^a Overtraining has been defined as “an accumulation of training and/or non-training stress resulting in long term decrement in performance capacity with or without related physiological and psychological signs and symptoms of maladaptation in which restoration of performance capacity may take several weeks or months.” In contrast, the phenomenon of Functional Overreaching has been described as follows: “Intensified training can result in a decline in performance however, when appropriate periods of recovery are provided, a supercompensation effect may occur with the athlete exhibiting an enhanced performance when compared to baseline levels”. Furthermore “when this intensified training continues, the athletes can evolve into a state of extreme overreaching or Non-functional Overreaching that will lead to a stagnation or decrease in performance which will not resume for several weeks or months. However, eventually these athletes will be able to fully recover after sufficient rest.”³

potential to contribute to improvements in the accuracy of equations that attempt to model performance.

Optimizing the prescription of training first involves quantifying what the athlete is currently doing. Several methods have been suggested to quantify the intensity of exercise bouts, some of which will be reviewed below. Questionnaires and diaries for example obtain recalled data from the athlete. Physiological measures such as oxygen consumption, blood lactate concentration and heart rate have been used to quantify exercise intensity more objectively. Recently theoretical indices of training stress have been proposed that attempt to quantify a training session into a unit “dose” of physical effort or internal physiological stress.

Secondly, it needs to be established whether or not the athlete is adapting favourably to certain levels of exertion. Ideally this adaptation should be quantifiable. With this knowledge, training can then be titrated to optimize the athlete’s improvement in order to meet a specific goal within a specified time. Some measurements and methods that have been used to facilitate the monitoring of training adaptations are reviewed below. Numerous physiological adaptations that occur in response to prolonged exercise training have been investigated as possible markers to monitor fitness, fatigue, overtraining and recovery. However, to date no single measure has been identified that can accurately assess if an athlete is adapting positively or negatively to training or that can predict overtraining. Recently more global physiological measures of chronic fatigue including recovery, mood states and muscle soreness have been investigated for their potential to contribute information about how a person is responding to training. Finally, measures of heart rate such as resting, submaximal and maximal heart rate, heart rate variability and heart rate recovery are presented for their potential to quantify training adaptations. Since the autonomic nervous system responds to exercise, has a major effect on heart rate, and is interlinked with many physiological systems, the measurement of the responsiveness of the autonomic nervous system to training, using a heart rate measurement, may provide useful information about the functional capacity of the body.

1.2 QUANTIFYING TRAINING LOAD

The ability to quantify the intensity of exercise performed in a training session is important in assessing the effectiveness of training. Several methods have been used to quantify exercise training intensity and have been reviewed by Williams and Eston (1989)¹ and Hopkins (1991)⁶. Hopkins (1991) proposed four method categories, namely retrospective questionnaires, diaries, physiological monitoring and direct observation⁶.

1.2.1 Questionnaires and diaries

Questionnaires and diaries obtain recalled self reported data from the athlete. Diaries are usually completed frequently (daily) whereas questionnaires are used to examine physical activity during the past week, month or even years⁶. The use of questionnaires to assess habitual physical activity and exercise, especially in large populations, is popular since their administration is easy, cost effective and does not impede training; however their weakness is the fact that the athletes' responses are subjective⁶ with the potential of inherent error. Thus the use of data collected by questionnaires to quantify exercise load is limited by inadequate reliability and validity compared to laboratory measures⁷. Reliability, defined as the ability to generate similar results on two separate occasions, decreases as the time between the activity and recall increases, since this is dependant on human memory^{6,7}.

A sports score derived from the Baecke questionnaire assesses the intensity of physical activity and has been used to estimate weekly training load⁸. The sport score is calculated using the duration (h.wk⁻¹), frequency (mo.yr⁻¹) and intensity of the activity (unitless codes based on energy costs)⁸. There are however inherent problems with questionnaires that assess the type, intensity, frequency and duration of exercise, as well as the environmental conditions in which the exercise was performed^{6,7}. Duration and frequency for example, may be over-reported especially if the person is influenced by the response they believe is sought by the investigators. Seasonal variations in duration and frequency of training may also not be taken into consideration. Perceptions of intensity may differ depending on the experience and tolerance of the person, particularly if asked to report intensity as simply light, moderate, hard or very hard. Although absolute measures of intensity are commonly

used, the relative equivalent may be more informative since considerable inter- and intra-individual differences may exist in the way people respond to various modes of exercise. The environmental conditions under which the activity is performed may have important motivational, psychological and physical effects on the person, but are often overlooked. Responses to questionnaires can also be influenced by differences in human understanding, which may be the result of cultural factors or due to the translation of the questionnaire when used in more than one language. The length of the questionnaire and the detail required from participants may also affect results, as people may become bored or confused with exhaustive questioning^{6,7}.

Finally, the response rate to a questionnaire must be reported, since a sample that is not representative of the population may introduce bias to the results⁶. Therefore, although questionnaires may assist with monitoring general changes in population activity, attempts to quantify exercise dosage from data collected with questionnaires appears to remain inadequate⁷. Diaries have an advantage over questionnaires in that they are recorded within a shorter time period after the training session⁶. However, diaries share many of the drawbacks of questionnaires, such as poor compliance, the necessity to keep diaries short and simple, honesty and understanding, to name a few.

The validity of self-reported data should therefore be considered an important factor contributing to the accuracy of training load quantification, particularly if subsequent changes in the training programme are made based on these results. The question therefore arose: "Do people actually do what they say they do in exercise training?" Chapter 1 of this thesis investigates the relationship between self-reported and recorded training duration (considered one of the easiest training factors to quantify) in a group of self-coached athletes.

1.2.2 Physiological measures

Physiological variables that have been used to quantify exercise intensity have included relative and absolute measures of oxygen consumption (VO_2), blood lactate concentrations and heart rate^{1:6}.

1.2.2.1 Oxygen consumption

Since it is generally accepted that the relationship between oxygen consumption and steady state work rate is linear⁹ (Figure 1.1) VO_2 has been promoted as a valid measure of exercise intensity during steady state exercise but not interval, supra-maximal exercise bouts.

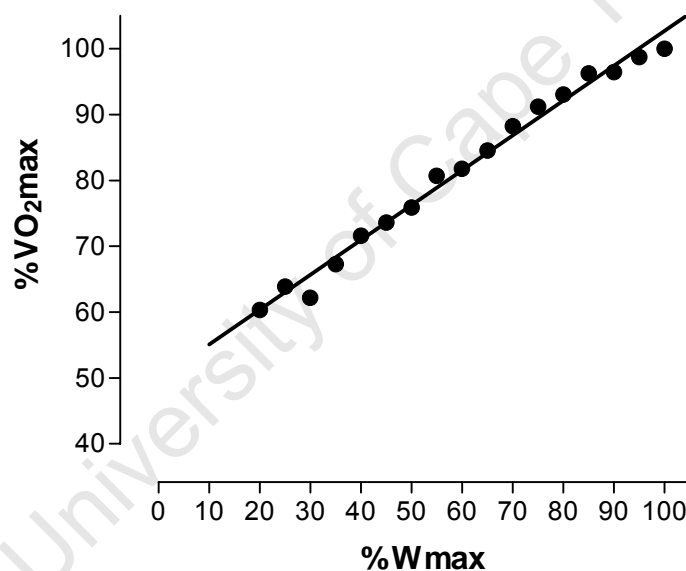


Figure 1.1: The relationship between percent maximal oxygen consumption ($\% \text{VO}_2\text{max}$) and percent maximal workload ($\% \text{Wmax}$)

In a review of the oxygen kinetics during exercise Xu and Rhodes (1999) point out that when exercising at a work rate below the lactate threshold VO_2 increases exponentially to a steady-state level, but when exercising above the lactate threshold VO_2 kinetics become more complex¹⁰. Relative ($\% \text{VO}_2\text{max}$) rather than absolute values of VO_2 have been used to compare the exercise intensities of athletes of differing physiological and performance characteristics⁶. However maximal oxygen

consumption (VO_2max)^b is exercise mode specific. For example the same person may have a lower VO_2max after performing a maximal test on a cycle ergometer or during swimming, than on a treadmill¹². Therefore VO_2max needs to be determined for the specific mode of exercise before exercise can be prescribed using relative VO_2 values. Oxygen consumption reserve (VO_2R) has been suggested as a more accurate means with which to prescribe exercise intensity than $\% \text{VO}_2\text{max}$ ¹³. In this equation the average VO_2 during a training session is combined with maximum VO_2 and resting VO_2 in order to relate a relative VO_2 value to the intensity of workload. In this way a target workload can be established at a given percentage of the difference between maximal and resting VO_2 ¹³ (Appendix 1, Equation 1.1).

It has been shown in cycling and running, that whereas calculating exercise intensity using heart rate reserve (HRres, explained in 1.2.2.3 below and Appendix 1, Equation 1.2) and VO_2R give similar results, the exercise intensities calculated using percent heart rate reserve ($\% \text{HRres}$) and $\% \text{VO}_2\text{max}$ differ^{14;15}. Baldwin et al (2000) found that heart rate and plasma markers of exercise stress such as lactate, ammonia and hypoxanthine at $70\% \text{VO}_2\text{peak}$ ^c were different between trained and untrained individuals, but were similar when exercise was undertaken at 95% of lactate threshold¹⁶. This supports the suggestion that using $\% \text{VO}_2\text{peak}$ does not necessarily produce the same physiological response in different people. It has also been found that the VO_2 kinetics at the onset of exercise may differ with the level of physical training, age and disease, and as such may be inappropriate as a means with which to prescribe relative exercise intensity^{16;17}. Swain et al (2000) point out however, that although the use of $\% \text{VO}_2\text{R}$ is preferable over $\% \text{VO}_2\text{max}$, exercise prescriptions based on $\% \text{VO}_2\text{max}$ may still be used for highly trained athletes since the difference between $\% \text{VO}_2\text{R}$ and $\% \text{VO}_2\text{max}$ for these individuals is minimal at exercise intensities typically used in training¹³.

^b VO_2max is defined as the maximal rate of oxygen uptake and utilization by the body, and is the product of maximum cardiac output and maximum arteriovenous oxygen consumption difference¹¹. The measurement of VO_2max must satisfy certain objective criteria, one of which is that maximal exercise capacity is reached. This is usually accepted if two of the following criteria are met: (i) a rating of 8-10 on the Borg CR-10 RPE Scale (ii) a peak heart rate similar to an age-predicted maximal heart rate (iii) a respiratory exchange ratio (RER) of > 1.10 ¹¹.

^c VO_2peak is the maximal rate of oxygen uptake and utilization by the body at voluntary volitional fatigue. The measurement of VO_2peak does not need to satisfy the objective criteria necessary in the measurement of VO_2max .

1.2.2.2 Lactate

The use of lactate to prescribe training intensity usually requires an initial incremental test in which a variety of speeds are correlated with measurements of blood lactate concentration. At subsequent training sessions the exercise speed is prescribed based on the desired lactate level according to these correlations. Particular attention has been paid to determining the lactate threshold, defined as the exercise intensity at a fixed blood lactate level or the maximal lactate steady-state¹⁸. It has been proposed as a measure of endurance fitness¹⁸, but also a means with which to standardise training intensity¹⁹. The steady-state exercise intensity that elicits a lactate concentration of approximately 4 mmol.L⁻¹ has been suggested as the most favourable intensity for inducing optimal physiological adaptations for endurance events¹⁹. However, Stegmann et al (1981) warn that this “optimal” lactate level may range from 2.0 to 7.5 mmol.L⁻¹ among athletes²⁰. Eniseler (2005) used blood lactate concentrations to quantify the intensity of various soccer training activities²¹. He found that about half of the activity during “match play” elicited blood lactate concentrations above 4 mM, and that most of the activity during “tactical training” and “technical training” occurred below 2 mM lactate. During the “modified game” training activity approximately 45% of activity was <2 mM, 30% of the activity was between 2 and 4 mM, and about 25% was above 4 mM lactate²¹.

Majumdar et al (1997) investigated using the magnitude of the lactate response to estimate the training stress during specific on-court badminton training regimes aimed at improving speed endurance²². This training consisted of five segments of differing intensities, which are imposed by manipulating the work-rest ratio. They compared the blood lactate concentrations of each training segment with measurements taken during simulated match play, and found much higher levels of lactate during the training segments than during match play (8 – 10.5 mmol.L⁻¹ vs. 4.7 (±1.9) mmol.L⁻¹ respectively)²². The lower lactate readings during match play may have been due to the characteristic short intermittent type of exercise, whereas the higher lactate response during on-court training may be due to a lower technical requirement during these segments, allowing the player to exercise at a greater intensity than is possible during matches²². The authors suggest that such intense workouts could have a negative effect on the co-ordination and technical skill that is

essential in badminton. As such monitoring the lactate response and controlling the training intensity below 6 mmol.L^{-1} may be useful²².

The measurement of blood lactate concentrations has become easier with the development of portable measurement instruments that require the collection of only one drop of blood from a finger prick. Nevertheless, it remains impractical to measure lactate frequently during every training session to prescribe intensity. Furthermore, the inherent inter- and intra-individual differences in the extent to which lactate accumulates during exercise are two of many limitations in the use of lactate to prescribe exercise intensity^{19,23}. Extraneous factors such as ambient temperature and dehydration may influence the interpretation of lactate variables. The mode of exercise can also influence interpretation, particularly when the muscle mass recruited during exercise is different²³. During running and cycling for example, the same lactate concentration occurs at different levels of VO_2 ²⁴.

Exercise duration, intensity and the rate of change in exercise intensity may also influence lactate concentration. Prior exercise, diet and muscle glycogen content may also affect the relationship between lactate concentration and exercise intensity^{19,23}. Exercising with damaged muscles has also been shown to cause an increase in blood lactate levels²³. Improvements in training status as well as overtraining have both been associated with decreases in maximal and submaximal blood lactate concentration²⁴⁻²⁶, which may lead to erroneous interpretations of lactate measurements and incorrect exercise prescription²³. The interpretation of lactate concentration may further be affected by sampling and measurement procedures such as the time and site of blood sampling, measurement techniques and dilution volume^{19,23}. The extent to which the abovementioned factors affect the way lactate accumulates, independent of exercise intensity, makes the importance of the lactate threshold less definitive²⁰, thus limiting its usefulness in monitoring and prescribing training intensity.

1.2.2.3 Heart rate

Heart rate monitoring has become a popular way to measure exercise intensity²⁷. This method is based on the principle that there is a linear relationship between heart rate and steady state work rate^{6,9,28}. Pharmacological blockade and measures of

heart rate variability have been used to assess the contributions of the autonomic nervous system to the changes that occur in heart rate from rest through increasing intensities of exercise in healthy participants^{27;29-33}. Parasympathetic tone appears to predominate at rest²⁹, with the initial increase in heart rate at the onset of exercise being primarily due to parasympathetic withdrawal³¹⁻³³ (Figure 1.2). As the intensity of exercise increases to more vigorous levels and heart rate increases above 100 b.min⁻¹ the sympathetic system is activated which causes further increases in heart rate³⁴. At maximal exercise intensity it is the combination of parasympathetic withdrawal and sympathetic predominance that contribute to the observed tachycardia³¹⁻³³ (Figure 1.2). Kannankeril et al (2004) discovered that *total* parasympathetic withdrawal does not occur even during high intensity exercise³⁰.

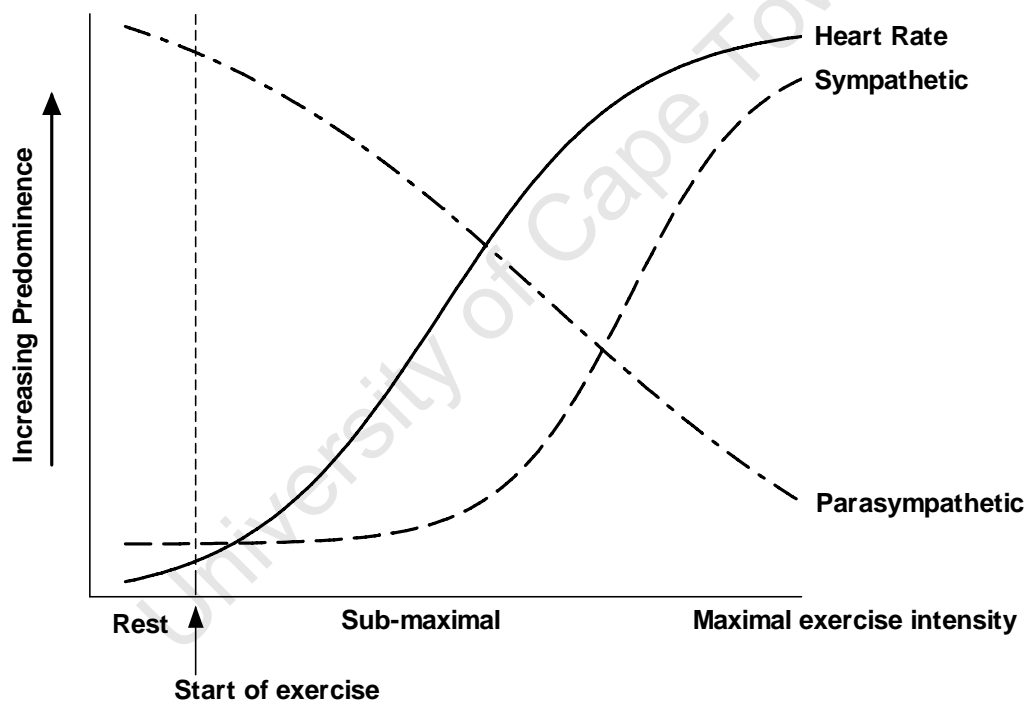


Figure 1.2: Schematic representation of the contributions of the parasympathetic and sympathetic nervous systems to heart rate at rest and during increasing intensities of exercise.

Eniseler (2005) investigated heart rate measurements during various soccer training activities to quantify the intensity of each²¹. He found that average heart rate was highest during “match play” (157 ± 19 b.min⁻¹), followed by “modified game” (135 ± 28 b.min⁻¹) and “tactical training” (126 ± 21 b.min⁻¹), and lowest during “technical training” (118 ± 21 b.min⁻¹)²¹. Percent maximum/competition heart rate has also been used to prescribe exercise intensity⁶, but Karvonen and Vuorimaa (1988)

suggest that the use of percent heart rate reserve (%HRres) is a more accurate means of quantifying and prescribing intensity, since this method takes into account the fact that resting heart rate increases and maximal heart rate decreases with age³⁵. In this equation the average heart rate during a training session is combined with maximum heart rate and resting heart rate in order to relate a relative heart rate (%HRmax) to the intensity of workload³⁵ (Appendix 1, Equation 1.2).

Although heart rate monitors have been found to measure heart rate accurately during physical activity, many factors may influence the relationship between work load and heart rate. The day-to-day variation in heart rate is approximately 6 beats per minute³⁶, or <6.5%³⁷. However, if the factors affecting heart rate, such as state of training, environmental conditions, diurnal changes²⁸, exercise duration, hydration status, altitude^{27:28} and medication are controlled, the accuracy with which heart rate can be used as a marker of exercise intensity improves³⁶.

1.2.3 Rating of Perceived Exertion

A rating of perceived exertion (RPE) is based on the assumption that the total physiological stress experienced during exercise can be represented by a subjective score²⁸ between 6 and 20 in the Borg 6-20 RPE Scale (Appendix 2). This range of scores was initially validated against heart rate, such that $HR = RPE \times 10^{38}$. This principle was demonstrated in a study by Robinson et al (1991) who found that during steady state exercise the athletes' reported RPE correlated well to their average heart rate recorded during the sessions²⁸. They concluded that it may be possible for runners to adjust their training intensity using their own perceptions of effort. Green et al (2006) also found that acute changes in heart rate and RPE measures during high intensity interval cycling were closely associated, showing that both respond similarly and rapidly to alterations in workload³⁹. Little and Williams (2007) however disagreed, as they found poor correlations between heart rate and RPE responses during short duration, high intensity soccer drills⁴⁰. The validity of RPE in regulating exercise intensity during step dance sessions was also found to be poor when compared to heart rate measures⁴¹. The authors suggest that the use of the Borg 6 – 20 RPE Scale is not suitable for the nonlinear structure of step dance sessions, and that it may be more appropriate for estimating the intensity of progressive increases in power output⁴¹.

A meta analysis of the literature concluded that, although the Borg scale has been shown to be a valid measure of exercise intensity, the validity coefficients between the Borg 6 – 20 RPE Scale and physiological criterion variables are not as high as previously thought⁴². For example, the weighted mean validity coefficients were 0.62 for heart rate, 0.57 for blood lactate, 0.64 for %VO₂max, 0.63 for VO₂, 0.61 for ventilation and 0.72 for respiration rate. Gros Lambert and Mahon (2006) also point out that the Borg RPE Scales have only been validated in adults, not in children⁴³. The relationship between RPE and heart rate in adolescents and younger children appears to be affected by mode of exercise, and by cardiorespiratory and other developmental factors in even younger children. In middle-aged and elderly individuals however, RPE can be associated with heart rate as a useful tool to control exercise intensity, providing training status is taken into account. In their review Gros Lambert and Mahon (2006) found that training may alter the HR-RPE relationship by increasing the person's ability to detect muscular sensations and to utilise sensory cues in the perception of effort. They point out that it is still not clear how the brain interprets afferent feedback to produce a perception of effort⁴³. Thus further research is required to ascertain the physiological mechanisms behind our cognitive perception of effort, which may clarify exactly what RPE represents.

1.2.4 Indices of training stress

1.2.4.1 Training Impulse

Eric Banister and co-workers proposed a method of quantifying a training session into a unit "dose" of physical effort⁴⁴. They suggested that a person's heart rate response to exercise, along with the exercise duration, collectively called a Training Impulse (TRIMP), may be a plausible measure of physical effort as it is based on the extent to which exercise increases heart rate between resting and maximal levels⁴⁴. A TRIMP is calculated using training duration, maximal heart rate, resting heart rate and the average heart rate during the exercise session. A weighting factor (Y) that emphasizes high intensity exercise is also applied to the equation to avoid giving disproportionate importance to long-duration low intensity exercise compared to intense short-duration activity⁴⁴. The Y factor is based on the lactate profiles of trained men and women relative to increases in exercise intensity (Figure 1.3).

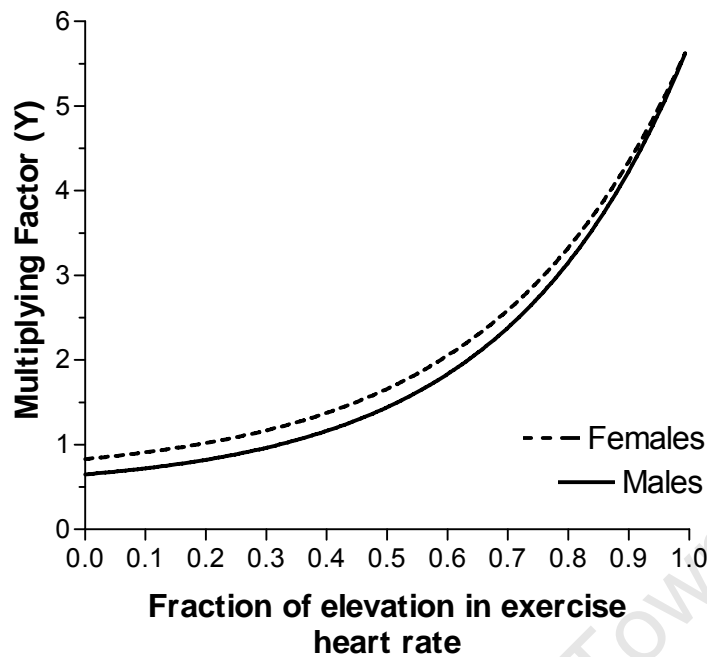


Figure 1.3: The relationship of the multiplying factor (Y) to increases in heart rate based on the pattern of increased blood lactic acid accumulation during exercise. Adapted from Banister (1991)⁴³.

The ability to quantify training to a single figure/factor, as is possible with this equation, is particularly appealing in terms of its practical application. However, the use of this method of quantification is limited by the necessity to use heart rate monitors throughout training and requires steady state heart rate measurements, thus limiting the accuracy with which exercise of an interval nature can be quantified. A further practical limitation of TRIMP as a measure of training load is the inability to quantify non-aerobic modes of exercise such as resistance training. This is because heart rate increases disproportionately during resistance exercise and the heart rate responses required for the calculation of TRIMP are not elicited.

1.2.4.2 Summated Heart Rate Zones

The Summated Heart Rate Zone Score is a modification to the calculation of training load that facilitates the quantification of interval training⁴⁵. The accumulated duration (min) spent in each of five heart rate zones is calculated (i.e. 50-60%, 60-70%, 70-80%, 80-90%, 90-100% of maximal heart rate) and then multiplied by a multiplier factor for each zone (50-60% = 1, 60-70% = 2, 70-80% = 3, 80-90% = 4, 90-100% = 5). The results are then summated. After an extensive review of the literature, there appears to be no evidence that this method of quantification has been validated. The

Summated Heart Rate Zones equation may therefore have been derived theoretically and not through experimentation.

1.2.4.3 Lucia's TRIMP

Recently a modified version of the Summated Heart Rate Zone equation has been used by Earnest et al (2004) and Lucia et al (2003) and referred to as "Lucia's TRIMP" by Impellizzeri et al (2004)⁴⁶⁻⁴⁸. In this method the duration spent in each of three heart rate zones (zone 1: below the ventilatory threshold; zone 2: between the ventilatory threshold and the respiratory compensation point; zone 3: above the respiratory compensation point) is multiplied by a coefficient (k) relative to each zone (k = 1 for zone 1, k = 2 for zone 2, and k = 3 for zone 3) and the adjusted scores are then summated. The original source of this equation however was not referenced in these papers.

1.2.4.4 Session RPE

Foster et al (1996) introduced a Session RPE measure to calculate training load instead of using heart rate data, in an attempt to simplify the quantification of training load⁴⁹. To calculate Session RPE the athlete's perceptions of the intensity of training are included in the quantification of training load. The intensity, heart rate and the type of exercise being performed do not need to be measured. The Session RPE scale is a rating of the overall difficulty of the exercise bout, obtained 30 minutes after the completion of the exercise⁴⁹. It has been adapted from the Borg Category Ratio (CR-10) RPE Scale (Appendix 3) which translates the athlete's perception of effort into a numerical score between 0 and 10⁵⁰. The assessment of Session RPE is designed to encourage the athlete to respond to a simple question "*How was your workout?*" with the goal of getting an uncomplicated response that reflects the athlete's global impression of the workout⁴⁹.

According to Foster et al (1998, 2001) a daily session load can be calculated by multiplying Session RPE (Appendix 3) by the duration of aerobic exercise (in minutes), or the number of repetitions performed in resistance exercise^{51;52}. The use of Session RPE to quantify training load has potential in being a mode- and intensity-independent method that can be used for multiple types of exercise such as

high intensity, or non-steady state exercise like resistance training⁵³⁻⁵⁵, high-intensity interval training or plyometric training⁵¹. However, there remain limitations to its use in both aerobic and resistance training.

RPE has been shown to be influenced more by resistance load than by volume, so that performing more repetitions with a lighter load was perceived as being easier than performing fewer repetitions against a heavier load^{53;54}. Sweet et al (2004) and McGuigan et al (2004) found that the RPE varied significantly when using different muscle groups during resistance exercise that was performed at the same percent of 1-repetition maximum^{53;55}. They explained this phenomenon by proposing that perceived exertion increases as muscle mass (and hence metabolic demand), range of motion and the number of joints involved in a movement increase. They further suggested that the order in which the exercises are performed, the fibre type of the muscle used, the mode of exercise for which the athlete is trained (i.e. the level of experience the athlete has in resistance training), as well as the time at which RPE is reported may also affect RPE⁵³. For example, a study by Day et al (2004) found no difference between session RPE and mean RPE recorded after one set of resistance exercise⁵⁴ at equivalent exercise intensities, whereas Sweet et al (2004), who recorded mean RPE after two sets, did report differences. It was subsequently found that RPE increased significantly between the first and second sets of resistance exercise⁵³.

Foster et al (1998, 2001) have proposed Session RPE to be a valid and reliable measure of exercise intensity in aerobic exercise when compared to heart rate-based methods^{51;52}. They compared the Session RPE method with the Summated Heart Rate Zone Score (described in 1.4.3 below) and found that although the Summated Heart Rate Zone Score gave lower scores than the Session RPE method, the pattern of differences between the two methods was very consistent⁵¹. However, no correlation coefficients were provided in Foster et al (2001)⁵¹, and although individual correlations between the two methods ranged between $r = 0.75$ and 0.90 in Foster et al (1998), statistical methods were not explained⁵². Impellizzeri et al (2004) studied the relationship between the Session RPE method and the three objective methods (Banister's TRIMP, Edwards' Summated Heart Rate Zone method and Lucia's TRIMP) in soccer players while training and playing matches⁴⁶. Individual correlations between the Session RPE method and Banister's TRIMP method ranged between $r =$

0.50 and 0.77; individual correlations between the Session RPE method and Edward's Summated Heart Rate Zone method ranged from $r = 0.54$ to 0.78 ; and individual correlations between the Session RPE method and Lucia's TRIMP methods ranged between $r = 0.61$ and 0.85^{46} . They suggest that the Session RPE-based score cannot yet replace the heart rate-based methods as a valid measure of exercise intensity as only 50% of the variation they measured in heart rate could be explained by the session RPE⁴⁶.

The complex interaction of many factors that contribute to the personal perception of physical effort, including hormone concentrations (e.g. catecholamines), substrate concentrations (e.g. glucose, glycogen, lactate), personality traits, ventilation rate, neurotransmitter levels, environmental conditions or psychological states may limit the use of RPE in accurately quantifying or prescribing exercise intensity¹. Although using objective physiological measurements like heart rate may be a more accurate way of calculating training load, the subjective measure of RPE remains useful. Thus if heart rate monitors are not available, or an easier means of reporting and calculating training load is required, the RPE method may still give reasonably accurate assessments of training load.

The Training Impulse (TRIMP), Session RPE and the Summated Heart Rate Zone methods are becoming popular methods of quantifying training load. The accuracy of these methods in assessing internal training stress is important particularly if changes in training programmes are to be made based on these results. Therefore Chapter 2 of this thesis assesses the relationships between the subjective method (Session RPE) and two objective methods (TRIMP and Summated Heart Rate Zone method) of quantifying *ad libitum* training. In addition an attempt was made to identify characteristics that might explain the variance not accounted for in the relationship between these objective and subjective methods.

1.2.5 Direct observation

Direct observation is usually carried out by a coach during the training session and may record training components such as exercise mode, duration and absolute or relative intensity⁶. Speed, for example, may be a useful measure of intensity in swimming or on a flat measured, indoor running track. However, in other modes

such as cycling, skiing and cross country running, the influence of factors such as terrain, environmental conditions and equipment may alter the accuracy with which speed reflects intensity⁵⁶. Direct observation may also include subjective measures such as the coach's perception of whether or not an athlete is overtraining⁶.

Perceptions of training by coaches and athletes have been studied by Foster et al (2001) in which they found significant differences between the training coaches prescribed and the training athletes actually did⁵⁷. They initiated a study to find contributing factors to negative training outcomes such as overtraining syndrome or sub-optimal performance. Moderate relationships were found between the training load ($r = 0.72$) and the exercise intensity ($r = 0.75$) that coaches had prescribed compared to what the athletes actually did, whereas a low relationship was found for training duration ($r = 0.65$). On days that the coach prescribed a hard training session the RPE reported by the athlete was significantly less than intended by the coach. Conversely, on days prescribed by the coach to be days of easy training or even rest, it was found that several athletes trained at moderate to high levels⁵⁷. This dissociation may have a significant impact on the effectiveness of the training the coach subsequently prescribes. The extent to which training can be quantified based on direct observations may therefore also be limited. Since this method requires the presence of an observer at every training session, which may be impractical or impossible, the amount of data able to be collected in order to monitor training accurately may be inadequate⁶. Where possible physiological measurements should corroborate direct observation and should be used to report the validity of the data used so that it can be considered when assessing the results.

New technology using global positioning system (GPS) offers innovative ways to track distance covered and speed during training⁵⁸⁻⁶⁰. The accuracy of these techniques has improved so that for distance mean errors of 0.04 – 0.7% have been found, and for position mean errors of 1.94 – 2.13 m have been found⁶¹. Schutz and Herren (2000) found that the accuracy of speed prediction had a standard deviation of 0.08 km.h⁻¹ for walking and 0.11 km.h⁻¹ for running, yielding coefficients of variation of 1.38% and 0.82% respectively⁶². Studies have shown that GPS can be used to quantify training load in horses^{63;64}. Although there are to our knowledge no published studies using this technology to quantify training load in humans there does appear to be potential for its use under certain circumstances.

The accurate prescription of training towards the attainment of a desired performance not only requires the accurate quantification of training loads, but also knowledge of the physiological mechanisms involved in the response to training. In addition, the ability to accurately predict future performance requires the development of a quantifiable measure of the effect of training loads and training response on performance.

1.3 MODELLING THE RELATIONSHIP BETWEEN TRAINING AND PERFORMANCE

Attempts have been made to correlate quantified exercise loads with subsequent performance in order to facilitate the prediction of future performances. Foster et al (1996) presented quantified observations of the performance response of competitive athletes to changes in training load but found no significant relationships between the improvements in time trial performance and training time, duration of high intensity training, training intensity (reflected in RPE ratings) or training load (calculated using Session RPE)⁴⁹. These findings further emphasize the complex relationship between a number of training variables that may contribute to training load, the body's adaptive response and subsequent performance. More complex mathematical models have been developed that attempt to describe the effect of training bouts (impulses) on subsequent performance as a dose-response relationship. These models consider the athlete as a system in which the training load is the input and performance the system output. The systems models are attractive in their potential to allow more accurate prediction of performance at specific times, or conversely to enable the design of optimal training programmes towards a specific performance goal⁶⁵⁻⁶⁷.

Banister and co-workers proposed an equation to assess the training effect (dose) on performance (response) thus allowing the establishment of a quantifiable relationship between these two variables⁴⁴. It is suggested that performance could be defined by two components, a 'fitness impulse' and 'fatigue impulse' and that at any time their difference (fitness – fatigue) can predict an athlete's performance^{44;66}. Thus, in its simplest form the equation comprises two functions in which one represents a positive influence on performance (Fitness) and the other represents a negative

response to training (Fatigue) such that the predicted performance at any time is the difference between these two factors (Figure 1.4, Appendix 1, Equation 1.3)⁴⁴.

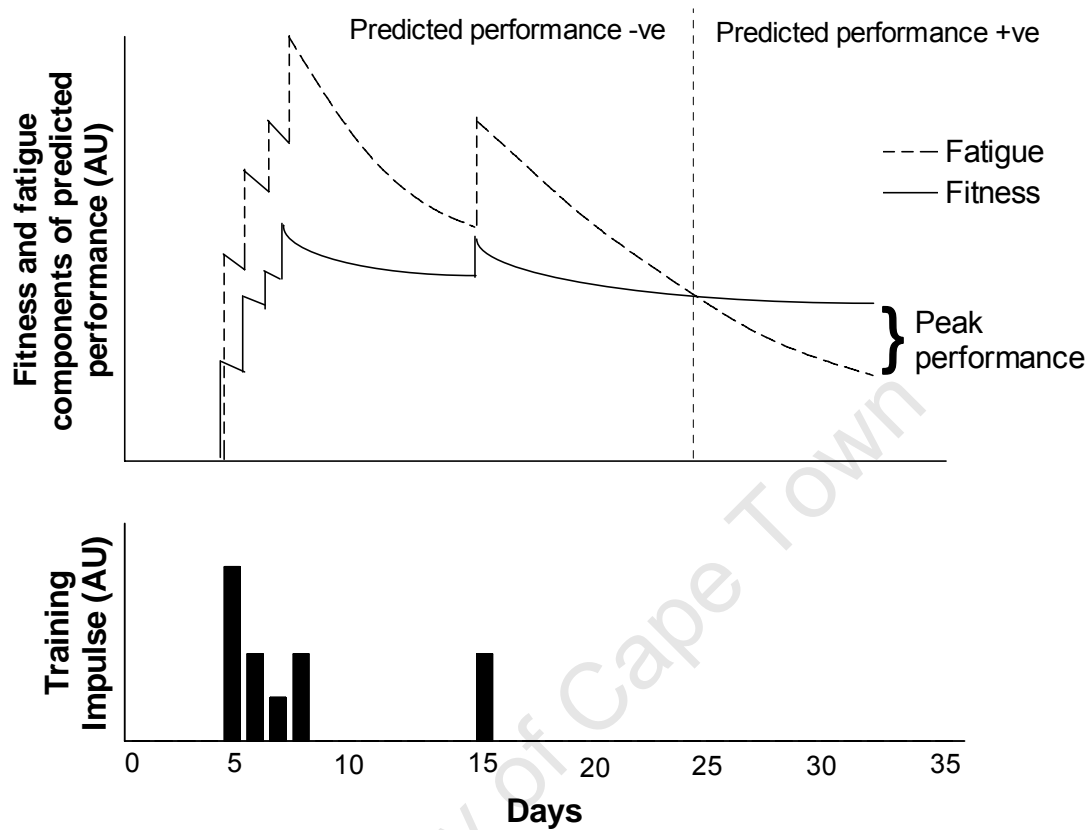


Figure 1.4: Schematic representation of the accumulation and decay of the fitness impulse and fatigue impulse in response to training impulses. The contribution of a training impulse to fatigue is shown proportionally larger than to fitness however the decay time constant of fitness is longer. The difference between the fitness impulse and the fatigue impulse represents predicted performance. Adapted from Banister (1991)⁴³.

Between training sessions the fitness and fatigue variables decline exponentially but at different rates. Banister (1991) suggested that the fitness decay time constant may be estimated initially as 45 days and the fatigue decay time constant as 15 days (Figure 1.4). These values are only estimates that allow initial estimates to be made of future performance. As data are collected thereafter these estimates are compared to real performance measures, and the constants adjusted if discrepancies occur between the estimated and real performance⁴⁴.

Busso et al (1991) subsequently tested the accuracy of a simplified form of the above model, comprising only the fitness impulse⁶⁷. They found that incorporating this variable in the model produced a similar fit of estimated and real performances, accounting for 61% to 87% of the total variation in estimated and actual

performances. However, they pointed out that the fatigue effect may have been under-estimated due to the low intensity endurance training of the subjects in the study, and as such did not contribute substantially to changes in performance. They suggested that future studies should include more strenuous and varied training programmes. The researchers further acknowledged the low precision of the performance measures, with the standard error (SE) of the estimated performances ranging from 3.6 to 5.9 performance units (PU) and the real performances SE ranging between 97 and 152 PU. They ascribed this low precision to external factors like daily stress⁶⁷ which cannot be controlled, but need to be recognized as an integral part of performance.

Wood et al (2005) explored possible physiological and psychological correlates of the positive and negative components of the Banister model in an attempt to validate the parameters with physiological markers⁶⁸. They found that running speed at ventilatory threshold and running economy correlated with the fitness parameter ($r = 0.94$ and $r = -0.61$ respectively), whereas a fatigue subset of the POMS (Profile of Mood States questionnaire, discussed in section 1.4.2.3 below) correlated moderately ($r = 0.75$) with the fatigue parameter of the equation. In an attempt to understand this relationship the authors suggest that the fatigue subset of POMS might be reflecting a more global fatigue (comprising various stressors of occupation, lifestyle, illness etc) whereas the fatigue component of the equation may be representing only exercise-induced fatigue. The validity of the fatigue component will thus remain unclear until an accurate measure/marker of exercise-induced fatigue is found. Either that or the fatigue component does not accurately represent exercise-induced fatigue⁶⁸.

Fitz-Clarke et al (1991) proposed the use of influence curves which shows conceptually how each consecutive day's training would affect subsequent performance⁶⁵. The influence curve is a line representing the effect of a training impulse on performance at a specific future time (Appendix 1, Equation 1.4). Performance on a particular day may be considered as a summation of the contributions of each day's training impulse prior to the day of competition and decayed over the time between the training impulse and competition day. Each training impulse adds a contribution to performance according to its initial magnitude⁶⁵.

The impulse response encompasses both the negative and positive influences of each day's training from the start of a programme until competition day. The influence curve thus allows for the calculation of the time point before competition at which training load needs to be reduced, since training after that day would contribute more to the fatigue impulse than to the fitness impulse⁶⁵. The influence curve can also identify a time point in days prior to competition at which training is most beneficial to performance on competition day⁶⁵. This model thus has the potential to design an optimal training programme able to produce a specific performance at a particular time. Influence curves can be used effectively when giving training advice for one event, however analysis of training and performance for several competitive events during a season, as is common for elite athletes, is more complex. Influence curves show that the prescription of optimal training for each event becomes a challenge as the training and rest periods ideal for one performance will impact sub-optimally on subsequent performances⁶⁵.

In the equation of Banister et al (1991) (Appendix 1, Equation 1.3) the model parameters τ_1 (time constant of decay of fitness), τ_2 (time constant of decay of fatigue), k_1 (fitness multiplying factor) and k_2 (fatigue multiplying factor) are initially estimates that are adjusted to suit the individual after fitting a predicted performance to a real performance, and subsequently kept constant (i.e. time-invariant) for the duration of the study period^{44;66}. Busso et al (1997) investigated using a recursive least squares algorithm incorporating parameters that are free to vary over time to more accurately illustrate varying changes in performance after training⁶⁹. They suggested that each training response may be influenced by previous training bouts and that the day-to-day variation in the model parameters may provide important information on the cumulative effects of training^{69;70}. The relationship between predicted and real performances was better using a time-varying model than the time-invariant model, with coefficients of variation for the former being 0.875 and 0.879 for the 2 subjects, compared with 0.682 and 0.666 for the time-invariant model⁶⁹. However, since the parameters in the time-varying model are estimated at any given time from the previous and present data, this type of model would be limited in its ability to predict performance in response to future training, unless the parameters themselves change in a predictable way⁷¹.

Using their recursive least squares algorithm, Busso et al (2002) later studied the effect of an increase in training frequency on exercise-induced fatigue and found that the time needed to regain a previous performance level increased as training frequency increased⁷⁰. The positive effect of a given training load on performance also decreased when training frequency was increased. Thus reducing recovery time between training bouts resulted in an increase in accumulated fatigue⁷⁰. Subsequently they proposed a model that would account for greater fatigue resulting from increased training frequency⁷². The model comprises a fatigue component that varies over time and with intensity of past training bouts. It offers the possibility to more accurately describe the dose-response relationship between cumulative training loads and training response, and thus to study training periodisation⁷².

Hellard et al (2005) proposed a model that includes the concept of a threshold saturation, in which the impact of training on performance is nonlinear and has an upper limit⁷³. This method introduces the possibility of identifying an upper limit to the training stimulus of an athlete so that training intensity and duration can be kept below this threshold in order to optimize physiological adaptations. Training maintained above this level may induce excessive chronic fatigue and lead to a decline in performance. Hellard et al (2005) studied Olympic swimmers over 4 years and found the modified model improved the fit between training and performance compared to the Banister model, however the training variables still explained only 30% of the variation in performance⁷³. They suggested a number of reasons for this discrepancy: 1) an individual's response to the same exercise load may differ between seasons, 2) there may be indirect effects of training, for example adaptations to one form of exercise may influence/change the way in which the body responds to another exercise mode, 3) variations in technique, or 4) the fact that swimmers react differently to the same training stimulus⁷³. The model parameters were also assumed to remain constant throughout the duration of the study, whereas regular adjustment of these parameters may have improved the fit between predicted and real performances.

The proposed mathematical models attempt to describe the effect of training bouts on performance as a dose-response relationship comprising fitness and fatigue impulses. However, although attractive in concept, the accuracy of these theoretical models has proven poor, as is evident in weak correlations between the predicted

and measured performances in response to training^{67,69}. In a study examining the effect of training on performance, Mujika et al (1996) noted significant variance among elite swimmers in a number of systems model variables⁷⁴. The time constants of decay of the fitness and fatigue impulses (τ_1 and τ_2 of the Banister model) ranged between 30 and 70 days, and 0-25 days respectively, and the fitness and fatigue multiplying factors (k_1 and k_2) ranged from 0 to 13.34. The time frame before competition when training load contributed negatively to performance ranged from 0 to 27 days, and the time point prior to competition when training has the most beneficial effect on performance varied between 0 and 56 days. Lastly, the model explained between 45% and 85% of the variations in real performance. This emphasizes a limitation in the design of the performance models, which do not take into account the fact that individuals respond and adapt differently to training. However a limitation in the study was the assumption that the model parameters were constant throughout the study (44 weeks) which ignores the possibility that adaptations to training may alter how an athlete responds to subsequent exercise training⁷⁴. Indeed, Banister suggested that the period within which the model parameters may be assumed to be constant is 60 to 90 days, after which the parameters need to be reset by comparing predicted performance to real performance⁴⁴.

The research conducted for this thesis has the potential to contribute to improvements in the accuracy of equations that attempt to model performance, such as those described above. The first step towards this goal is to more accurately quantify training load. However, it is not only the quantification of training load, but knowledge of the physiological mechanisms involved in the exercise response and the ability to quantify and monitor training induced adaptations, that will allow more accurate prescription of training and hence produce predictable performances.

1.4 MONITORING TRAINING ADAPTATIONS AND RESPONSE TO TRAINING

At the biological level, an exercise training session may be interpreted as a stimulus that causes a disturbance in homeostasis and transient physiological and metabolic changes⁵. These changes are restored to their pre-exercise resting levels during the recovery period after the training session. After several training sessions, the efficiency with which the physiological systems underlying homeostatic control function is altered, so that subsequent exercise at the same intensity may cause less disturbance to homeostasis. With continued training chronic adaptations occur which are often associated with an improvement in physical performance. The progression of changes that occur to the human body following the start of an exercise training programme can be explained by a theoretical concept called the General Adaptation Syndrome. This concept was developed by Dr Hans Selye⁷⁵, a clinical endocrinologist and experimental biologist who was particularly interested in the physiological response of an organism to an imposed stress. He considered any “noxious stimulus” a stressor and the organism’s response to that stimulus the stress response⁷⁶. He divided the stress response into three stages: Stage 1 - the Alarm Reaction, Stage 2 - the Stage of Resistance and Stage 3 - the Stage of Exhaustion. The stress response, including these stages, is represented graphically in Figure 1.5. According to Selye the organism will progress through each of these stages providing the exposure to the stress stimulus is maintained⁷⁵.

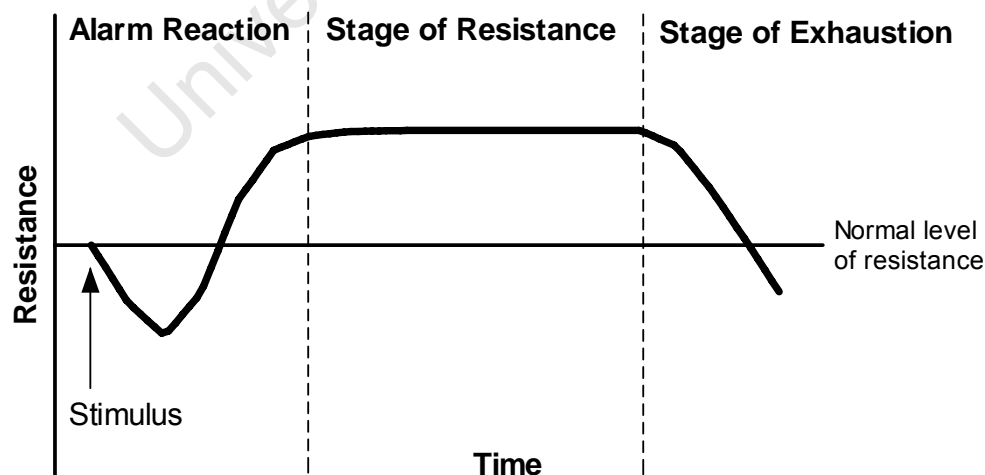


Figure 1.5: Schematic representation of the General Adaptation Syndrome, depicting three stages of change in the degree of resistance of an organism to a stressor. Adapted from Selye (1974)⁶⁷.

According to this concept the initial phase of the Alarm Reaction Stage may be equated with the disturbance in homeostasis and diminished resistance that occurs when a stress such as exercise training is initially imposed on the body¹¹. The physiological response is that the body enters an adaptation phase to restore its homeostatic state⁵. If the exposure to the stress is prolonged the body enters the Stage of Resistance, Stage 2 of the General Adaptation Syndrome, in which the body has adapted to the applied level of stress and has possibly reached a new homeostatic state or increased functional capacity. Finally, if the stress (exercise training) is continued at the same or even higher levels for an extended time, without adequate rest, the Stage of Exhaustion is reached⁷⁵. This stage is particularly relevant in sport and exercise training as this stage may be associated with a decline in performance or an inability to maintain a training load. This is a well described phenomenon known as the overtraining syndrome^{2:77-79}.

Recreational athletes generally have a positive relationship between increases in training load and the physiological adaptations that result in improvements in performance. However, for elite athletes already training at very high volumes, there is a fine line between doing too little or too much training, making this relationship much more critical⁷⁹⁻⁸¹. Insufficient training will not induce appropriate adaptations and will result in suboptimal performance. Too much training however may cause chronic fatigue and a subsequent decline in performance^{77;78}. In order to produce optimal adaptations, training load and recovery must be balanced so that the athlete's physiological systems are sufficiently stimulated to adapt. This can only occur in the presence of adequate recovery between training sessions^{52;79;82;83}. The ability to measure this physiological stimulus and to monitor how an athlete is responding to it would greatly facilitate the accuracy of training prescription.

1.4.1 Physiological adaptations to endurance training

Physiological adaptations that occur in response to prolonged exercise training have been well documented in the literature^{16;34;84-92}. These include changes in muscle morphology, altered metabolism⁵, changes in neuromuscular recruitment patterns during exercise⁹³, as well as improvements in endocrine, cardiovascular and respiratory function. Some have been investigated as possible markers to directly

measure and monitor fitness, fatigue, overtraining and recovery. These include serum iron and ferritin concentrations^{87;94-99}, circulating testosterone and cortisol^{2;100-102}, intramuscular enzymes involved in fatty acid and glycolytic metabolism^{11;80;88;92;103-107} and lactate metabolism^{18;19;23;108} to name a few. A few commonly used “markers” will be discussed below with reference to their relationship with performance.

1.4.1.1 VO₂max, Exercise economy, Lactate & Ventilatory thresholds

Jones and Carter (2000) in their review of the effects of endurance training, identify four key parameters of aerobic fitness, namely VO₂max, exercise economy, lactate and ventilatory thresholds and critical power (which influences oxygen uptake kinetics)⁸⁶. They suggest that an improvement in any one or more of these parameters will produce an improvement in performance. An increase in left ventricular volume after endurance training increases stroke volume which, along with increased myocardial contractility and increased oxygen extraction by the muscle, results in a greater VO₂max^{86;109}. In trained individuals, the exponential increase in VO₂ at the onset of exercise to a steady state level of oxygen uptake is reached sooner. Lactate and ventilatory thresholds are also reported to shift to higher power outputs or running speeds after endurance training. However the relationship between endurance training and exercise economy, defined as the oxygen uptake required at a given absolute exercise intensity, remains unclear⁸⁶. The lactate and ventilatory thresholds in particular have been suggested to be good predictors of endurance performance. However, it must be acknowledged that many other factors such as environmental conditions, race tactics or psychological factors may also influence the outcome of a competitive performance⁸⁶.

1.4.1.2 Lactate

As mentioned in section 1.2.2.2 blood lactate profiling, and in particular the determination of the lactate threshold, has become a popular way to prescribe exercise intensity. In addition, the measurement of blood lactate concentrations during submaximal exercise has been proposed as a means with which to monitor changes in endurance fitness. The background to this is that blood lactate concentration decreases at the same absolute and relative intensity after endurance training¹⁹, and that the absolute work rate at which the onset of blood lactate

accumulation (OBLA) occurs increases after 6 weeks of training¹⁰⁸. A study by Pyne et al (2001) monitored lactate in elite swimmers through a competitive season and found that lactate profiling, derived from a 7 x 200m incremental step test, correlated well with improvements in endurance fitness over the 20-week period¹⁸. However, although there was a direct relationship between improvements in lactate parameters and improvements in maximal 200m test time, they were unrelated to international competition performance¹⁸. A number of factors independent of training have been identified as having an affect on blood lactate concentrations. These have been reviewed by Swart and Jennings (2004)²³ and were presented in section 1.2.2.2. The usefulness and accuracy with which lactate profiling can be used to monitor training adaptations thus remains questionable due to its dependence on standardized testing conditions including well controlled diet, prior training, warm up routine, levels of fatigue and glycogen depletion amongst other factors¹⁸.

1.4.1.3 Testosterone and cortisol

The role of hormones in monitoring or detecting overtraining is not yet fully understood, and is further complicated by conflicting findings. Circulating cortisol concentrations have been found to be higher in overtrained athletes², whereas short-term (6 days) high volume training has produced a decrease in resting serum cortisol levels¹⁰². Bell et al (2000) found that urinary cortisol concentrations increase in women that do concurrent endurance and strength training and decrease in endurance-trained men¹¹⁰. Serum cortisol concentrations increase with intensity, duration and muscle mass involved in exercise, but may also be affected by non-training-induced factors such as genetics, age, diet and time of blood sampling¹⁰¹ which may decrease the precision of the measurement. It has been found that endurance-trained men have lower resting total plasma testosterone levels than untrained men¹⁰⁹, however Bell et al (2000) found that 12 weeks of training had no affect on resting serum testosterone¹¹⁰.

A review of training and overtraining studies by Urhausen et al (1995) found no significant change in circulating testosterone and cortisol at rest during an overtraining period¹⁰¹. They suggest that circulating cortisol and testosterone may be markers of the physiological response to acute exercise rather than to chronic training¹⁰¹. In their attempt to find a reliable marker for distinguishing between

trained and over-reached athletes Meeusen et al (2004) investigated the plasma cortisol response to two bouts of maximal exercise¹⁰⁰. They found that although plasma cortisol concentrations did not differ between the groups after the first bout, cortisol concentrations increased more in the trained group than in the over-reached group after the second maximal exercise bout¹⁰⁰.

The free circulating testosterone:cortisol ratio has been proposed to be an indicator of physiological anabolic/catabolic balance and has been found to decrease during periods of intense training and competition¹⁰¹. A low free testosterone:cortisol ratio (<30%) has thus been suggested to be a marker of a catabolic (overtrained or over-reached) state². The circulating testosterone:cortisol ratio has also been proposed as a predictor of performance; however consensus has not yet been reached on how testosterone and cortisol concentrations change in response to training and how this relates to performance. Filaire et al (2001) found that a decrease in the salivary testosterone:cortisol ratio did not coincide with a decrease in performance, suggesting that changes in the ratio may not be a reliable predictor of performance and as such the implications of observed changes in this ratio remain inconclusive¹¹¹.

1.4.1.4 Skeletal muscle

Hawley and Stepto (2001) presented a theoretical model of training-induced adaptations in skeletal muscle that are likely to influence performance in elite cyclists⁹¹. It comprises three main factors, the first of which includes changes in muscle morphology such as an increase in neural recruitment, capillary density, enzyme activity and type I fibre content⁹¹. The second involves the decrease in muscle glycogen utilization and the increase in intramuscular triglyceride oxidation that occurs during endurance exercise as a result of, amongst other things, an increase in muscle respiratory capacity⁹¹. The concentration and activity level of a number of intramuscular enzymes has been shown to increase after exercise training. Muscle GLUT-4 content for example is greater in endurance-trained muscle, resulting in an improved capacity for glucose uptake¹¹. Endurance training induces an increase in the activity of enzymes like carnitine transferase and fatty acid binding proteins involved in intramyocellular fatty acid transport, hormone-sensitive lipase involved in the mobilization of fatty acids from intramyocellular lipid stores and cytochrome c oxidase in oxidative phosphorylation¹⁰⁴. In this way intra-muscular

triglycerides are hydrolysed during moderate exercise and plasma free fatty acid flux¹⁰⁶ and working muscle free fatty acid uptake⁸⁸ is increased. Total fat oxidation and whole-body lipolysis are unaffected when measured at the same absolute or relative exercise intensities¹⁰⁷. Conversely intramuscular enzymes of glycolytic metabolism such as 6-phosphofructokinase decrease after training so that intramuscular triglyceride metabolism is favoured over the use of muscle glycogen stores and plasma glucose during extended submaximal exercise^{92;104;105}. Endurance exercise training thus appears to enhance the capacity of human skeletal muscle to accumulate glycogen and to spare its use during exercise¹⁰³. Bergman et al (1999) however suggest that after training the use of free fatty acids and intramuscular triglycerides as fuel is of secondary importance in moderate and high intensity exercise⁸⁸. Such contradictory findings cast doubt over the usefulness of serum intramuscular enzyme concentration in the detection of overtraining syndrome⁸⁰.

The third factor in Hawley and Stepto's (2001) theoretical model considers acid-base status in terms of an increased lactate threshold and transport capacity⁹¹. Hawley and Spargo (2007) also reviewed the cellular and metabolic adaptations in human skeletal muscle resulting from intense endurance training, many of which have been presented above¹¹². Hawley and Stepto (2001) conclude that knowledge of the physiological adaptations that occur in response to training regimes, and their subsequent effect on performance, is limited and that further research is required⁹¹. Thus, although much has been reported on the correlations between endurance training and physiological adaptations, specific markers that facilitate the quantification of a dose-response relationship between training, adaptation and performance remain to be identified.

1.4.1.5 Iron status

Iron status has been proposed as a marker of both long term training adaptations, such that it may be used to monitor training and to detect the early stages of overtraining, as well as a measure of the body's acute response to exercise. For example it has been suggested that the measurement of serum iron, serum ferritin and transferrin may be used to identify the inflammatory response to muscle damage as well as the state of acute and chronic recovery⁹⁷. Serum iron and ferritin

concentrations have been found to be reduced in chronically exercising individuals, particularly those training at higher intensities⁹⁴⁻⁹⁶. Such decreases may have a negative affect on performance^{94,95}. However conflicting results have been found. For example Bourque et al (1997) and Balaban et al (1995) found no significant change in the concentration of selected iron variables such as serum ferritin⁸⁷ after moderate-intensity endurance training in previously untrained women with normal iron stores⁹⁹, and in runners⁸⁷. Dickson et al (1982) studied the effect of acute exercise on iron metabolism and reported subnormal resting serum ferritin levels in 14% of well-trained runners, possibly a consequence of “dilutional anaemia”⁹⁸. Elevated serum ferritin levels were recorded after ultra-endurance running events, which returned to normal 6 days post-race. A haemoconcentration was evident immediately post-race, followed by peak haemodilution 48 hours after the event⁹⁸. Extraneous factors such as hydration, stress and muscle damage may be partly responsible for the conflicting results concerning changes in selected iron variables in response to acute or chronic exercise. This limits the use of serum iron and related biochemical markers as a means with which to detect overtraining and highlights the need for further research in this area.

1.4.1.6 Summary

Despite years of research many conflicting opinions exist about the effects of training and overtraining on each of the many physiological “markers” that have been investigated. As such, no single measure has been identified that can accurately assess how an athlete is responding to training or predict overtraining. The usefulness of the physiological marker depends on the precision, ease and frequency with which it can be measured, the speed with which results can be interpreted so that frequent monitoring is possible with little inconvenience to the athlete, and how well the marker tracks (or predicts) changes in performance. The correlation between training and the observed changes in these physiological variables is highly personal and dependant on individual tolerance of an exercise load, which may be a culmination of many internal and external factors. Thus attention needs to be directed towards the measurement of markers that reflect an individual’s overall responsiveness or adaptability to training rather than an absolute measure of the changes in physiological variables in response to exercise.

1.4.2 Fatigue

Some information about how the athlete is responding to training may be obtained from assessments of chronic fatigue. Chronic fatigue results from prolonged exposure to excessive training loads⁷⁸ with inadequate recovery and may be exacerbated by psychological and social stressors¹¹³. The condition persists long after the training session and may seriously affect the athlete's performance. Thus, if the symptoms of fatigue can be detected before they become serious, appropriate adjustments can be made to facilitate recovery and maintain performance. The following section discusses some potential markers of chronic fatigue.

1.4.2.1 Recovery

Factors which may affect recovery include nutrition, hydration, sleep, rest, relaxation, emotional support and stretching^{113;114}. Information about each factor needs to be gathered when assessing the recovery status of an athlete¹¹³. A scale for monitoring recovery, analogous to the Borg 6-20 RPE Scale³⁸ has been proposed¹¹³ (Appendix 4). According to Kenttä and Hassmén (1998) recovery can be assessed under two conditions: (i) perceived recovery and (ii) action recovery¹¹³. For the assessment of perceived recovery the athlete is asked before bedtime to rate his/her recovery for the previous 24 hours, including the previous night's sleep using the scale in Appendix 4. For the assessment of action recovery the athletes score themselves for each of four categories, namely (i) Nutrition and hydration, (ii) Sleep and rest, (iii) Relaxation and emotional support and (iv) Stretching and active rest (Appendix 5) for the preceding 24 hours. Although there are no published data using this scale it has potentially practical value as it is easy to administer, can be done on a daily basis and educates the athletes about the nuances of recovery.

1.4.2.2 Muscle pain

Delayed onset muscle soreness or stiffness (DOMS) is commonly experienced after an unusually strenuous training session and usually peaks 24 to 48 hours after the activity^{115;116}. Eccentric exercise in particular appears to induce more severe structural damage to the contractile elements of the muscle than other types of muscle actions^{116;117}. The duration and intensity of eccentric exercise may augment

the sensation of DOMS¹¹⁷. Many mechanisms behind the development of DOMS have been proposed, including high concentrations of lactic acid, muscle spasm and connective tissue damage, however a contemporary explanation is that it is a form of soft tissue injury which includes inflammation, altered membrane permeability and intra-muscular enzyme efflux¹¹⁷.

Muscle pain can be measured objectively or subjectively. An example of the objective method involves the use of a round-ended pressure probe that is pressed into the muscle and is calibrated so that a depression of 1 cm is equivalent to a force of 4 N¹¹⁸. The pain score classification is as follows: a score of 4 = 0 cm depression causing pain; a score of 3 = 1 cm depression causing pain, a score of 2 = 2 cm depression causing pain, a score of 1 = 3 cm depression causing pain, and a score of 0 = 4 cm depression causing pain. The sum of the scores represents the objective pain measurement¹¹⁸. The subjective measurement is based on a rating of general perceived pain and may range on a scale from 0 to 10, where 0 represents no pain, and 10 represents maximal, unbearable pain^{118;119}. Specific muscles are identified for the assessment based on the mode of exercise, and should be done every day at the same time of day. An example of an electronic interface developed in this laboratory to collect the subjective assessments of muscle soreness is presented in Appendix 6. This analogue scale assesses three lower limb muscles (the calf, hamstring and quadriceps) at rest, during activities of daily living and during stretch. The participant is asked to click on each of the pointers on the left and drag it across each line to a point between the cues "No pain" (on the extreme left) and "Unbearable pain" (on the extreme right) that they think represents their muscle sensation at that time.

Lambert et al (2002) found similar increases in objective and subjective reports of muscle pain between 12 and 120 hours after inducing muscle damage in the biceps with eccentric exercise¹¹⁸. Likewise, Semark et al (1999) found that both subjective and objective assessments of muscle pain in the quadriceps muscle were increased at 24 and 48 hours after an exercise protocol involving drop jumps¹¹⁹. Thus either form of pain assessment can be used to accurately report measures of muscle pain^{118;119}.

1.4.2.3 Profile of Mood States

The use of ratings of RPE and mood states have been proposed as possible detectors of impending overtraining¹²⁰. Rietjens et al (2005) studied various physiological, biochemical and psychological markers to determine whether strenuous training-induced fatigue (possibly leading to an overreached state) could be diagnosed early¹²¹. Training load was increased by 100% and maintained for 2 weeks in an attempt to induce overreaching. However, the intensified cycling training resulted in no change in performance and only a trend towards an increased average mood score on the POMS questionnaire, reflecting a decline in mood states. After finding significant differences in cognitive speed tests they suggested that central fatigue may be the first and most sensitive parameter with which to detect overreaching, with an increase in the POMS score (abbreviated version) and an increase in RPE being secondary markers of this state¹²¹. However, since the generally accepted definition of overreaching is a transient decline in performance that is restored after a few weeks of recovery^{79;83;94;122} it can be argued that these athletes did not reach an overreached state, in which case the researchers' conclusions may be misleading.

Changes in mood states, assessed with POMS have however been shown in other studies in which performance decrements did occur, supporting the use of the POMS scores as an early indicator of overreaching^{83;122}. Filaire et al (2001) found that positive mood states (i.e. lower scores for Tension, Depression, Anger, Fatigue and Confusion and higher scores for Vigour) coincided with an increase in winning performances in a soccer team, despite an increase in the intensity of training¹¹¹. They also observed an increase in Depression and Tension during a period of poor performance, where relationships between players and coach, financial and family problems, and levels of fatigue appeared to be unchanged. They therefore suggested that the changes in POMS during this period may have been affected by factors other than those relating to training and external influences and that psychological as well as physiological changes should be considered when monitoring training stress in relation to performance¹¹¹. As such, the use of POMS scores alone in the assessment of cumulative training stress should be done cautiously.

Recently the validity of a single-item measure of daily fatigue was tested against the Profile of Mood States (POMS)¹²³. In the single-item measure participants rate their current state of fatigue (“How fatigued do you currently feel?”) with a report mark from 1 (“not at all”) to 10 (“extremely”). A distinct advantage of developing single-item measures is that the inclusion of short, simple and comprehensible questions will create user-friendly questionnaires. This makes completing them less intimidating and time-consuming and may reduce the drop-out of respondents. van Hooff et al (2007) found the single-item measure to be a valid measure of fatigue and thus may have important practical applications in the assessment of training response¹²³.

1.4.3 Using heart rate to monitor training response

The physiological systems of the body, in particular the autonomic nervous system and adrenal glands¹²⁴, respond during and after an exercise bout to maintain homeostasis. The autonomic nervous system is interlinked with many other physiological systems and thus the responsiveness of the autonomic nervous system may provide useful information about the functional adaptations of the body^{11;125}. It is well known that the autonomic nervous system has a major effect on the regulation of heart rate and the development of heart rate monitors¹²⁶ has allowed further research into the mechanisms behind heart rate responses to exercise and adaptations to training.

1.4.3.1 Resting heart rate

There have been many reports that resting heart rate decreases slightly after endurance training¹²⁷⁻¹³⁴. The mechanism behind the observed resting bradycardia in trained athletes was investigated by Smith et al (1989)¹³⁵. They suggested that this phenomenon was due to a decrease in the intrinsic rhythmicity of the heart and an increase in the predominance of parasympathetic control. There was also a slight decrease in the sympathetic contribution to heart rate¹³⁵. Many studies showing a difference in resting heart rate between trained and untrained individuals are cross-sectional in nature, thus limiting ones ability to discern whether the difference is due to training or due to an inherent variation in the two populations^{132;135}.

Melanson and Freedson (2001) found no significant difference in the resting heart rate of previously sedentary individuals after 16 weeks of training¹³⁶. A possible explanation for this is that the training load in this study may have been less than in studies which did observe slight decreases in resting heart rate. In these studies training duration ranged from 6 weeks to 5 months, the frequency of exercise bouts ranged from 3-7 times per week and duration of each exercise bout ranged from 30 minutes to 4 hours^{127-130;132;133}. In the study by Melanson and Freedson (2001) participants trained 3 times a week, 30 minutes per session and only increased the intensity of exercise from 70%HRreserve in the first 2 weeks to 80%HRreserve from week 3¹³⁶. In the other studies, either the duration of each exercise session was increased substantially (high intensity training at 70-90%VO₂max was increased by 130% and low intensity training at <70%VO₂max was increased by 100%¹²⁷) or the intensity was increased (from 50-60%HRreserve to 80-85%HRreserve¹³⁰) or both were increased (30 minutes of exercise at 55%VO₂max in the first 2 weeks was increased to 50 minutes of exercise at 75%VO₂max^{129;132}). In addition, since changes in resting heart rate may be influenced by many environmental factors, the use of resting heart rate as a marker of endurance training may be of limited value³⁶.

To overcome the difficulties of measuring an accurate resting heart, it has been suggested that measuring heart rate during sleep may be a marker for training status¹³⁷ and monitoring cumulative fatigue. Waldeck and Lambert (2003) investigated the repeatability of monitoring heart rate during sleep, since it can be assumed that the relationship between heart rate and training status should be more evident when the influence of extraneous factors is reduced¹³⁸. Sleeping heart rate was assessed over a three week period during which the subjects kept their daily training constant in order to determine if the measurement was precise enough to be used as a marker of training status. The intra-subject day-to-day variation of minimum heart rate during sleep was about 5 b.min⁻¹. The authors concluded that this variation needs to be considered when interpreting training-induced changes in heart rate¹³⁸. Longitudinal studies have found varying results with regard to changes in sleeping heart rate, with some finding a reduced¹³⁹ or unchanged¹⁴⁰ heart rate after several weeks of exercise training, while others found an increased¹³⁷ heart rate after two weeks of intensive training. With this amount of variation, it is unlikely that changes in heart rate measured during sleep will have any practical prognostic value in identifying fatigue in athletes.

1.4.3.2 Submaximal heart rate

It is widely accepted that, under controlled conditions, heart rate at the same submaximal intensity decreases after endurance training and is proposed to be due to a decrease in sympathetic activity to the heart^{83;102;129;141-144}. However, Swaine et al (1994) found that a non-significant decrease in heart rate (HR) at a respiratory exchange ratio (R) of unity ($HR/R_{=1}$) was reversed after 8 weeks of moderate-intensity training suggesting that training-induced bradycardia occurs during the early stages of training and is subsequently reversed¹⁴⁵. Uusitalo et al (1998) also detected non-significant changes in intrinsic heart rate and autonomic modulation (measured as the sympathovagal balance index after pharmacological blockade with atropine and propranolol) in female endurance athletes after 6-9 weeks of intense training¹²⁷. Swaine et al (1994) proposed that training elicits different effects on various indices of fitness and that a decrease in submaximal heart rate may be a weak marker of cardio-respiratory fitness¹⁴⁵. What has not been mentioned is whether submaximal heart rate responds to acute changes in training load as well. This may have implications for its use as a tool to monitor acute physiological responses to imposed training loads. This question was investigated as part of a larger study in Chapter 4 of this thesis.

1.4.3.3 Maximal heart rate

Some studies report that maximal heart rate changes very little^{11;146-149} with endurance training whereas other studies show decreases in maximum heart rate with training¹⁵⁰⁻¹⁵² and increases with detraining^{153;154}. However a comprehensive review of studies that investigated the effects of endurance training on maximal heart rate, involving a total of 314 subjects, concluded that maximum heart rate can decrease by between 5 and 13 $b \cdot min^{-1}$ with aerobic training, and increase by 4 to 10 $b \cdot min^{-1}$ with tapering or detraining in some individuals¹⁵⁵. Proposed mechanisms explaining decreases in maximal heart rate with training include plasma volume expansion¹⁵¹, enhanced baroreflex function¹⁵⁶ (abstract) and decreased β -adrenergic receptor number and density^{157;158} (abstracts). However further research is required to explain this phenomenon fully¹⁵⁵.

1.4.3.4 Heart rate variability

Recently particular attention has been focused on heart rate variability (HRV), largely as a means with which to evaluate cardiac autonomic control. Heart rate variability is a non-invasive measurement of the variation in the R-R intervals on an electrocardiogram¹²⁵. The indices of HRV can be represented in a time domain in which R-R intervals (in milliseconds) are plotted against time (in seconds) (Figure 1.6).

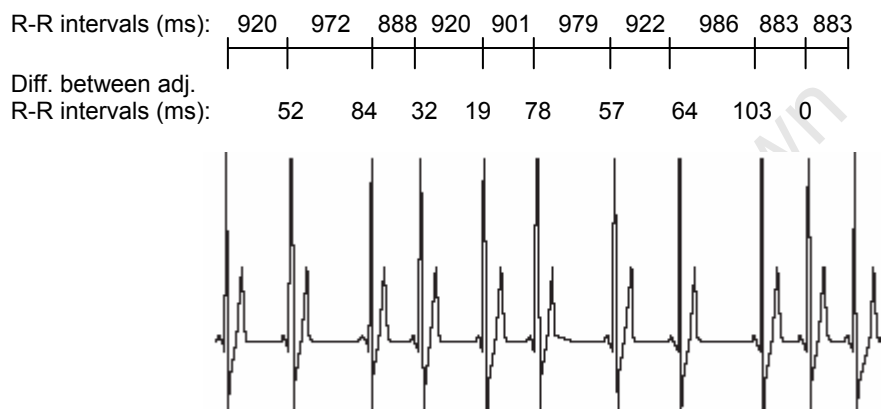


Figure 1.6: Heart rate variability can be represented in a time domain in which R-R intervals (ms) and the differences between adjacent R-R intervals are calculated. Adapted from Achten and Jeukendrup (2003)²⁶.

Heart rate variability can also be represented in the frequency domain, which is the frequency at which the length of the R-R interval changes²⁷ (Figure 1.7). The frequency domain is the power spectral density and corresponds to the power of different oscillations present in the electrocardiogram. Peaks at different frequencies represent differing contributions by the parasympathetic and sympathetic nervous systems.

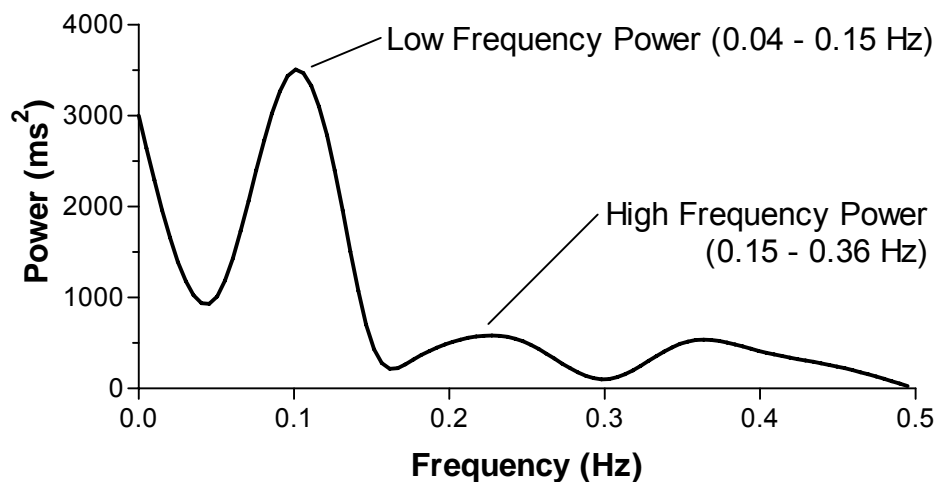


Figure 1.7: Heart rate variability represented in the frequency domain, which is the frequency at which the length of the R-R interval changes. Adapted from Achten and Jeukendrup (2003)²⁶.

The parasympathetic activity contributes substantially to high frequency power (HF), the energy in the heart rate power spectrum between 0.15Hz and 0.36Hz^{27;128}. Both sympathetic and parasympathetic activity contribute to low frequency power (LF), 0.04 - 0.15Hz. Ultra low frequency power (ULF) between 0.00066Hz and 0.0033Hz and very low frequency power (VLF) between 0.0033 and 0.04Hz can also be measured^{27;128;159}. The ratio LF : HF represents sympatho-vagal balance, allowing HRV to be used as an index of autonomic responsiveness, with high values reflecting sympathetic predominance²⁷.

- *Effects of endurance training*

A cross-sectional study using HRV in the time domain found that trained individuals have increased variability in R-R intervals (during a 24 hour recording period which included normal daily activities and sleep) compared to untrained individuals¹²⁸. However in longitudinal studies or in cross-sectional studies using the frequency domain, differences in total power, HF and LF due to training seem less conclusive. Some studies found that indices in the time domain^{128;136;160} or HF^{136;160} and LF^{161;162} in the frequency domain increased after training, where others did not see a change^{141;163} or reported a decrease in LF¹⁶⁰. These inconclusive results may be explained by the use of inconsistent methodologies such as the duration of the HRV recording (6 minutes to 24 hours) and body positioning during recording

(supine/semi-supine, during daily activities or sleep). Differences in training, such as the duration of the programme (6 weeks to 1 year), exercise session frequency (3 to 7 days per week), intensity (70% to 90% of HR_{res} or HR_{max} that either remained constant or increased over the duration of the trial) and duration (25 minutes to 4 hours) may also account for different results between studies^{128;130;136;160-164}. This diversity in methodologies makes it difficult to compare results to form a consensus regarding the effects of training on the various indices of HRV (Appendix 7).

Bonaduce et al (1998) compared 24-hour HRV in detrained athletes vs. untrained individuals, and also studied the effect of 5 months of vigorous training on HRV in the same detrained athletes¹²⁸. Variability was higher in both the time and frequency domains, and LF:HF was lower in detrained compared to untrained participants, whereas the same variables were unchanged after 5 months of training in the detrained athletes. There was however a decrease in resting heart rate after 5 months of training, suggesting that the change in resting heart rate after training may be due to mechanisms other than changes in sympatho-vagal balance. The authors suggest that, since the high frequency component represents “*fluctuations in autonomic inputs to the heart rather than the mean level of autonomic inputs*”, the possibility that training had increased parasympathetic influence without changing the modulation of efferent vagal activity cannot be excluded¹²⁸.

A similar phenomenon was observed in a study by Buchheit et al (2004) that investigated HRV during slow-wave sleep in sedentary, moderately trained and highly trained young subjects¹⁶⁵. They found that moderately trained subjects had increased vagal-related HRV indices compared to sedentary individuals but that this increase was not perpetuated in highly trained athletes, despite the latter group having lower heart rate¹⁶⁵. Studies comparing habitually active elderly subjects to their sedentary counterparts revealed that those with a long-term active lifestyle had higher global and vagal-related indices of HRV^{166;167}. In addition, greater improvements in vagal control were gained when more time was spent in moderate to high intensity activities or when daily energy expenditure was increased to a moderate level of 600 kcal.d⁻¹¹⁶⁷. These results may contribute to an improved understanding of the direct effect of training duration, frequency and intensity on HRV. However further investigation is required to confirm the findings and to assess their application when considering the use of HRV to monitor training status.

- *Detecting fatigue and overtraining*

HRV has also been proposed as a means with which to detect cumulative fatigue and overtraining. Portier et al (2001) investigated the differences in spectral indices during a relative rest period and during intensive training in elite runners¹⁵⁹. They found resting HRV and LF spectral power to be lower, while HF power was higher, and changes in the LF:HF ratio showed sympathetic control to be decreased during the intensive training period. The researchers suggest that the shift towards vagal predominance may indicate an increase in training-induced fatigue, and since autonomic balance seems to be affected by training, that spectral analysis of HRV may be useful in the early detection of accumulated fatigue¹⁵⁹. Conversely, Pichot et al (2000 and 2002) found that 3 weeks of intensive training and 1 month of overload training induced a shift towards sympathetic predominance during night-time recordings^{168;169}. A relative increase in parasympathetic and decrease in sympathetic tone occurred after a subsequent week of recovery^{168;169}. While some aspects of HRV may have the potential to be used to monitor autonomic control in athletes, it is premature as this stage to use it diagnostically with confidence¹²⁵.

- *Limitations*

Besides the disparate findings of various studies due to the use of vastly different methodologies (Appendix 7) a number of other factors may influence the interpretation of HRV and thus limit its use in monitoring training status. It has been found that HRV at rest¹⁷⁰ and 24 hr HRV¹⁷¹ decreases with age and that there is less withdrawal of parasympathetic tone during submaximal and maximal exercise¹³⁰ in the elderly. However Buchheit et al (2004) found that regular, long-term participation in sporting activities may counteract the age-related decline in cardiac autonomic control¹⁶⁶. Carter et al (2003) found that younger subjects had a larger autonomic adjustment to training than older participants of similar initial training status, suggesting that age may influence the adaptability of the cardiovascular system¹⁶⁴. Conversely, Levy et al (1998) found that the older subjects in their study, who had a 47% lower HRV than the younger subjects before training, experienced a greater increase in HRV (+68%) than the younger subjects (+17%) after 24 weeks of aerobic training¹³⁰.

Tulppo et al (1998) conducted a cross-sectional study assessing the effects of age and physical fitness on HRV at rest and during exercise¹⁷⁰. They found that aging resulted in a reduction in HRV at rest, but there were less profound age-related differences in HRV during exercise. In addition, although there were no differences in HRV at rest among three levels of fitness (based on VO₂ max), the subjects with higher VO₂ max values had higher HRV during exercise¹⁷⁰. The dissociation between changes in heart rate and changes in vagal-related HRV with training also reported by Bonaduce et al (1998) and Buchheit et al (2004), may limit the use of HRV as a marker of cardiac vagal control when vagal activity is already high (i.e. in highly-trained athletes)^{128;165}. Buchheit et al (2004) suggest further research is required to establish the mechanism behind the regression of vagal-related HRV indices in highly-trained athletes to levels comparable to sedentary individuals¹⁶⁵. Thus age, training status and their combined effects need to be considered when interpreting changes in the indices of HRV.

Other factors that affect HRV include respiration as heart rate increases during inspiration and decreases during expiration¹⁶². Therefore respiratory frequency, for example, needs to be controlled to ensure that spectral sympathetic indices are unaltered¹²⁵. Tidal volume, environmental and body temperature¹⁶⁴, time of day²⁷, sampling frequency and method of normalization¹⁶⁰ may all influence HRV. Heart rate variability needs to be measured during controlled rest or exercise¹⁶⁰ as the spectral analysis of heart rate requires steady state conditions¹²⁵. Thus, in order for the results of various studies to be compared and consensus reached regarding the specific training effects on HRV, standardized methodologies need to be established for short and long-term training involving untrained and trained individuals along with the relevant analysis^{27;160;162}. A review of the literature revealed that prospective, randomised, controlled, long-term studies using validated measurements are required before HRV can be used with confidence in monitoring training status¹²⁵.

1.4.3.5 Heart rate recovery

Heart rate recovery is the rate at which heart rate decreases (or the time taken for heart rate to recover) after moderate to heavy exercise and is dependant on the relationship between parasympathetic and sympathetic nervous activity¹⁷².

- *Mechanisms*

The parasympathetic and sympathetic control of heart rate deceleration following the cessation of cardiovascular exercise has been investigated. Savin et al (1982) proposed that sympathetic withdrawal contributed more to the decrease in heart rate immediately after exercise, with parasympathetic re-activation playing a greater role later in the recovery of heart rate¹⁷³. However, more recent studies have observed a coordinated interaction of parasympathetic re-activation and sympathetic withdrawal, with parasympathetic re-activation occurring faster, and therefore playing the more important role in the early deceleration of heart rate^{30;174-176}. The parasympathetic effect on heart rate recovery (measured as the difference in heart rate with and without atropine) was also investigated by Kannankeril et al (2004) who found that parasympathetic reactivation occurred rapidly in the first minute of recovery. Parasympathetic stimulation then increased until 4 minutes and remained fairly constant until 10 minutes after the cessation of exercise³⁰.

Heart rate recovery after maximal exercise is slower than after submaximal exercise in healthy individuals, and is attributed to the sympathetic nervous system being stimulated significantly more during maximal exercise. Thus, after high intensity exercise it is also the withdrawal of sympathetic stimulation that contributes to the deceleration of heart rate after the cessation of exercise³⁰. Pierpont et al (2000) have investigated the time course of autonomic change during immediate recovery from exercise and in particular the validity of using a first-order exponential decay curve to model the heart rate response after exercise¹⁷⁷. They found this curve to be an imprecise depiction of heart rate recovery after maximal exercise but acceptable for modelling recovery after submaximal exercise. They suggest that after low levels of exercise, when the sympathetic nervous system has not been entirely stimulated, heart rate recovery, which is governed mainly by the re-activation of the parasympathetic system, may follow a first order exponential decay. However after high intensity exercise, during which sympathetic stimulation dominates, the sympathetic drive may continue well into the recovery phase and contribute to sustained tachycardia despite the re-activation of the parasympathetic system. This would alter or delay the first order exponential decay that can be applied to heart rate recovery after submaximal exercise¹⁷⁷. When comparing heart rate recovery in boys and girls Mahon et al (2003) noted gender differences after submaximal exercise

performed at an absolute work rate¹⁷⁸. However, after exercise that was performed at a relative intensity heart rate recovery responses were similar. They therefore suggest that it is important to control the intensity of exercise that is performed directly before the assessment of heart rate recovery in order to more accurately evaluate changes in heart rate recovery within or between subjects¹⁷⁸.

- *Relationship to all-cause mortality*

A delayed heart rate recovery has been found to be a strong predictor of subsequent mortality^{172;179}, especially where coronary heart disease and cardiovascular disease is concerned^{180;181}. Delayed heart rate recovery is defined in this context as a reduction of 12 beats or less in the first minute following symptom-limited maximal treadmill exercise using standard and modified Bruce protocol¹⁷⁹. Desai et al (2001) found no correlation between abnormal heart rate recovery and increased incidence of myocardial infarction or ischaemia¹⁸². However they found strong correlations between heart rate recovery and the chronotropic variables of peak heart rate, percent peak heart rate and heart rate reserve percent in both healthy individuals and groups with coronary artery disease¹⁸³. This suggests that heart rate recovery depends significantly on the chronotropic response, such that chronotropic incompetence will produce an abnormally slow heart rate recovery¹⁸³.

Gaibazzi et al (2004) found no prognostic value in the use of 1-minute heart rate recovery after symptom-limited maximal exercise on a cycle ergometer¹⁸⁴, however a retrospective study of 2193 men who were followed-up after 7 years, found that the first 2 minutes of heart rate recovery after treadmill testing did predict mortality¹⁸⁰. Gaibazzi et al (2004) suggest that the different modes of exercise performed prior to measuring heart rate recovery may account for the opposing results¹⁸⁴. They propose that the difference in the characteristics of each exercise mode may alter the absolute value of heart rate recovery chosen as a prognostically significant cut-off, as well as changing the prognostic power of the parameter itself¹⁸⁴. Lipinski et al (2004) also found that there was an increased risk of coronary artery disease in the subjects who had a slower recovery of heart rate during the second minute of recovery after symptom-limited graded treadmill exercise to 85% of their estimated maximal heart rate¹⁸⁰. One mechanism proposed to explain the relationship between delayed heart rate recovery (delayed parasympathetic re-activation) and mortality is that during

delayed recovery the protective effect that parasympathetic tone affords the cardiovascular system is diminished¹⁷⁵. Kannankeril and Goldberger (2002) suggest that parasympathetic tone may produce an “antiarrhythmic” effect by prolonging ventricular refractoriness, but this mechanism requires further investigation during exercise and recovery in healthy and diseased populations¹⁷⁵.

Seshadri et al (2004) suggested that a delayed 1-minute heart rate recovery after exercise may be a useful prognostic tool for chronic obstructive pulmonary disease (COPD) patients, as obstructive, restrictive and mixed-type lung disease may be associated with autonomic imbalance¹⁸⁵. If this is the case it may become possible to differentiate between all-cause mortality that is due to underlying pulmonary disease from mortality due to cardiovascular causes¹⁸⁵. Kizilbash et al (2006) investigated the relationship between heart rate recovery and the metabolic syndrome and found that slower heart rate recovery does not precede development of the metabolic syndrome, but rather appears after syndrome components are present¹⁸⁶.

The effect of cardiac rehabilitation programmes on heart rate recovery has also been investigated. Tiukinhoy et al (2003) found that 12 weeks of rehabilitation improved 1-minute heart rate recovery by 18 b.min⁻¹ in patients that had experienced a cardiac event¹⁸⁷. Another 12-week aerobic-based cardiac rehabilitation programme produced an improvement of 5 b.min⁻¹ in 1-minute heart rate recovery in heart failure patients that had a low functional capacity and an abnormal heart rate recovery (<12 b.min⁻¹)¹⁸⁸. This training however had no effect on patients with a higher initial functional capacity and normal heart rate recovery (>12 b.min⁻¹)¹⁸⁸. Two months of exercise rehabilitation significantly improved 1-minute heart rate recovery (by 5 b.min⁻¹) in heart failure patients, however even greater improvements (13 b.min⁻¹) were observed up to 6 minutes into recovery after exercise testing¹⁸⁹.

Studies by Giallauria et al (2006) confirm the improvement in heart rate recovery after participation in cardiac rehabilitation programmes^{190;191}. A 3-month rehabilitation programme improved 1-minute heart rate recovery by 5 b.min⁻¹ in elderly acute myocardial infarction patients¹⁹⁰. In addition, long-term home-based exercise training after acute myocardial infarction was found to be useful for maintaining or improving the beneficial results of the 3-month exercise training programme on cardiovascular capacity and heart rate recovery¹⁹¹. In this study, a 6-month follow-up observed a

further $4 \text{ b}\cdot\text{min}^{-1}$ improvement in heart rate recovery¹⁹¹. They concluded that heart rate recovery, a simple means with which to assess autonomic tone, may be used to identify patients at higher risk for cardiac events, to evaluate patient outcomes after cardiac rehabilitation programmes and to investigate possible mechanisms behind the beneficial prognostic effects of exercise training in cardiac patients^{187;190-192}. MacMillan et al (2006) also found significant improvements in heart rate recovery after exercise training during cardiac rehabilitation and noted that heart rate recovery improved similarly in older and younger males and females¹⁹³. An interesting question that has not been resolved is whether exercise training alone improves mortality, or whether the improvement in heart rate recovery, as a result of an adjustment between sympathetic and parasympathetic control, has an independent role in improving mortality.

- *Relationship to training status*

Endurance trained athletes have a faster heart rate response at the start of exercise and after the cessation of exercise at similar absolute intensities compared to sedentary subjects^{143;194}. Bunc et al (1988) studied highly trained rowers¹⁹⁴ and Short and Sedlock (1997) compared untrained subjects (that had been participating in two hours of walking or low-impact aerobics per week for four to six months) with trained runners, cyclists and swimmers that had been participating in more than five hours of training per week for four to six months before the study began¹⁴³. Short and Sedlock (1997) found that the heart rate recovery kinetics after exercise that elicited the same relative intensity ($70\% \text{VO}_2\text{peak}$) were similar for trained and untrained participants. However, after the same absolute workload ($1.5 \text{ L}\cdot\text{min}^{-1} \text{VO}_2$) heart rate recovery was faster for the trained group¹⁴³. In contrast Hagberg et al (1980) found that the time course of heart rate recovery after submaximal exercise was faster after either an absolute or relative workload in participants that had trained for nine weeks¹⁹⁵.

As such heart rate recovery may be useful as an indicator of the functional status of the cardiovascular system. In support of this assertion Sugawara et al (2001) found that post-exercise heart rate recovery was significantly accelerated after only eight weeks of moderate intensity training and that it returned to baseline levels after four weeks of subsequent detraining¹⁹⁶. Yamamoto et al (2001) associated an increase in

parasympathetic tone with the accelerated heart rate recovery they observed within just seven days of a 6-week endurance training period¹⁹⁷. In this study post-exercise heart rate recovery remained accelerated after the first week whereas resting heart rate continued to decrease. They concluded that a training-induced change in the autonomic control of heart rate recovery occurs sooner than it does for resting heart rate¹⁹⁷. The effect that an early training-induced increase in plasma volume may have had on the observed adaptations to heart rate was not considered in this study.

Although most of the research investigating training-induced changes in heart rate recovery has focused on endurance training Otsuki et al (2007) found that heart rate recovery is accelerated in both strength- and endurance-trained athletes¹⁹⁸. In their study the time constant of post-exercise heart rate decay was lowest in endurance-trained athletes followed by strength-trained athletes, with the heart rate decay of both these groups being significantly lower than that of the controls¹⁹⁸. Brown and Brown (2007) studied the HRV during six minutes of recovery after high-intensity exercise in trained masters runners and showed that HRV decreased in both time and frequency domains¹⁹⁹. These findings suggested an increased parasympathetic withdrawal during heart rate recovery after high intensity exercise in trained masters runners¹⁹⁹. Giallauria et al (2005) studied the effect of 8 weeks of aerobic training in the healthy elderly and found that heart rate recovery improved, suggesting that exercise training improves parasympathetic - sympathetic balance in older subjects¹⁹².

Heart rate recovery is therefore known to change with endurance training^{143;194;196;197;200} and in diseased states^{172;175;179-181;183;185}. In addition, it has been suggested that overtraining causes a disturbance in autonomic control⁸² which may be reflected in recovery heart rate. However no research has been found that indicates whether heart rate recovery can be used as a marker representing the body's capacity to respond to training. This thesis explores this possibility. The first question that arises when considering this potential marker is how heart rate recovery relates to training load and whether heart rate recovery responds to acute changes in training load. Chapter 4 assesses heart rate recovery after a standardized submaximal exercise test in participants that vary in the amount of training they do each week, but who are accustomed to that amount of training. The next question to be addressed is what effect progressive increases in training load,

as in a periodised endurance training programme, has on the relationship between training load and heart rate recovery. This is discussed in Chapter 5 along with reports of the chronic adaptation of heart rate recovery to progressive training. Whether heart rate recovery responds in a predictable way to acute changes in training load under these dynamic conditions, or whether individuals respond and adapt differently to training is also examined in Chapter 5 of this thesis.

1.5 SUMMARY OF LITERATURE REVIEW

Despite years of research no single factor has been identified that can quantify how an individual is responding to training or can predict overtraining with accuracy. There is thus no quantitative means with which to assess whether the training response is optimal in terms of producing advantageous physiological adaptations and improving performance. The usefulness of a physiological marker also depends on the ease and frequency with which it can be measured and on the speed with which the results can be interpreted so that frequent monitoring is possible with little inconvenience to the athlete. The correlation between training load and the observed changes in these physiological variables is highly individualised and may be a culmination of many internal and external factors. Thus attention needs to be directed towards the identification of markers that reflect an individual's overall responsiveness or adaptability to training rather than an absolute measure of the changes in specific physiological variables in response to exercise. The functional state of the autonomic nervous system may provide useful information about the overall functional adaptation of the body in response to a training stimulus as it is a system interlinked with many other physiological systems in the body.

This literature review therefore began by examining the changes that occur in heart rate with endurance training and assessed the usefulness of heart rate based physiological measurements as tools with which to monitor training status. There is general consensus that resting and submaximal heart rates decrease with endurance training, although changes in maximal heart rate are less clear. All of these measurements however are affected by extraneous factors that limit their use as sensitive markers of training status. More insight into the autonomic control of heart rate (in particular the contributions of the parasympathetic and sympathetic systems

to heart rate) has become possible with investigations involving HRV. The effect of endurance training on the various indices of HRV however remains inconclusive. This can be attributed to the use of inconsistent methodologies that makes it difficult to compare results between different studies and to reach consensus.

The autonomic control of heart rate recovery following the cessation of cardiovascular exercise has also been investigated. Since the rate of recovery is determined by the coordinated interaction of parasympathetic and sympathetic systems, heart rate recovery may provide a measure of the disturbance in autonomic control in response to endurance training. It has been found that heart rate responses both at the onset and cessation of exercise are faster in endurance-trained athletes compared to untrained individuals and that a delayed heart rate recovery is associated with mortality. Thus heart rate recovery may represent a continuum of autonomic nervous system function with delayed heart rate recovery, compromised autonomic nervous system function and mortality at the one extreme and accelerated heart rate recovery, optimal autonomic function and enhanced cardiovascular condition on the other end of the continuum. Whereas the analysis of heart rate during exercise may provide a measure of cardiac load, the measurement of heart rate recovery may reflect training-induced disturbances in the autonomic nervous system that provide useful information about how an athlete is responding to endurance training. The rate of decay of heart rate during the first minute after the cessation of a standardized cardiovascular test may be unique to each individual and may be an easily measurable index of an athlete's current cumulative state of fatigue. It may provide useful information on cardiac and autonomic nervous system adaptations that may contribute to a more comprehensive understanding of how an individual is responding to training. This method of quantifying an individual's physiological responsiveness to training and the potential practical application thereof in the prescription of optimal training programmes was investigated in this thesis.

1.6 OBJECTIVES OF THIS THESIS

Scientific research is playing an increasingly important role in the development of optimal training programmes that prevent under training and overtraining and increase the chance of achieving winning or personal best exercise or sporting performances at the desired time. Optimizing the prescription of training firstly involves quantifying what the athlete is currently doing. Secondly, it needs to be established whether or not the athlete is adapting favourably to the training programme. With this knowledge training can be titrated to optimize the athlete's improvement in order to meet a specific goal within a specified time. This thesis therefore attempts to answer a number of research questions that arose when investigating these first two steps.

Quantifying training loads:

Chapter 2

- Do athletes actually do what they say they do with regard to exercise training duration?

Chapter 3

- How are different methods of quantifying training related?
- What physiological characteristics may explain the variance not accounted for in the relationship between objective and subjective methods of quantifying training load?

Monitoring the training response:

Chapter 4

- How is training load (TRIMP) related to heart rate recovery?
- Does heart rate recovery respond to acute changes in training load?
- Does submaximal heart rate reflect acute changes in training load?

Chapter 5

- Does heart rate recovery reflect chronic adaptation to increases in training load during a periodised endurance training programme?
- Does the relationship between training load and heart rate recovery persist in a periodised training programme, when training load is not held constant?
- Does heart rate recovery respond in a predictable way to acute changes in training load during a periodised training programme?
- Do individuals adapt in a similar way (acutely and chronically) after exposure to an endurance training programme?

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CHAPTER 2

<p>VALIDITY OF SELF-REPORTED TRAINING DURATION</p>

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2.1 INTRODUCTION

Training adaptations are a consequence of, amongst other factors, an athlete's response to a training stimulus. The training stimulus depends on the interaction between the frequency, duration and intensity of the training session and the recovery period before the next training session²⁰¹. If the training stimulus is not prescribed appropriately the athlete will under-perform either as a result of not doing sufficient training or of doing too much¹¹³. The first step in producing an effective, customised training programme is to quantify what the athlete is currently doing. Thereafter training can be adjusted to optimize the athlete's improvement. One method of quantifying/assessing training requires that the athlete self-report their training using questionnaires or diaries⁶. The use of questionnaires to assess training habits is popular among coaches since their administration is easy, inexpensive and does not interfere with training. A weakness however, is that the athletes' responses are subjective and therefore may be inaccurate⁶.

This may affect the quantification of training and influence the subsequent prescription of training intensity, duration and frequency by the coach. The difference between the training the coaches prescribe and the training the athletes actually do has been studied by Foster et al (2001) who initiated a study in an attempt to find contributing factors to negative training outcomes such as overtraining syndrome or sub-optimal performance⁵⁷. They found moderate relationships between the training the coaches prescribed and what the athletes actually did in terms of training load ($r = 0.72$) and exercise intensity ($r = 0.75$). A low relationship was found for training duration ($r = 0.65$) which was surprising, particularly as duration is a relatively easy component of training to quantify⁵⁷. The relationship between what athletes say they do in training compared to what they actually do has not been studied in athletes who prescribe their own training without the assistance of a coach. This is an important and substantial group from which participants will be chosen in future studies. Accordingly the aim of this study was to assess the relationship between self-reported and recorded training in a group of self-coached athletes. We chose training duration as the measured variable in this study as this is an easily quantifiable factor.

2.2 METHODS

2.2.1 Study Design

Fifteen men and nineteen women ($n = 34$), who trained regularly (range 82 – 853 min.wk⁻¹), were recruited from the local community, gyms and running clubs with the use of flyers and direct communication with club committees. The study was approved by the University of Cape Town Ethics and Research Committee. A Physical Activity Readiness Questionnaire (PAR-Q) (Appendix 8) was administered to all participants prior to their enrolment in the study, to ensure that they were not at any health risk while performing exercise during the study. Participants who answered “Yes” to two or more of the questions on the PAR-Q were not included in the study. The participants gave their informed consent after the testing protocol and potential risks of the study were explained to them. They were asked to maintain the same type and quantity of training for five weeks. For the first three weeks the participants trained *ad libitum*. During week 3 body composition, maximal oxygen consumption and maximal heart rate of the participants was measured and they self-reported their average weekly training duration for this period. During the following two weeks their *ad libitum* training duration was monitored. Participants were excluded from the trial if they were injured or ill or had other reasons which prevented them from training to their normal capacity during the study ($n = 5$), thus the data of 29 subjects were analysed.

2.2.2 Study Protocol

2.2.2.1 Anthropometric and physiologic measurements

Body mass was recorded on a calibrated scale (Seca model 708 Germany) and recorded to the nearest 100 g. The stature of each athlete was recorded to the nearest mm using a stadiometer (Seca model 708 Germany). Body fat percent was assessed with a near infrared (NIR) measurement on the right bicep using the Futrex-6100A/ZL (Kett Electric Laboratory, Futrex Inc. Gaithersburg, MD USA). A reliability study in this laboratory showed that the relationship between body composition using dual-photon x-ray absorptiometry and NIR was $r = 0.84$, and the

limits of agreement were -2.8 to 5.0% (n = 59) (unpublished data). Resting heart rate, recorded immediately after waking in the morning with a Vantage XL heart rate monitor (Polar Electro, Kempele, Finland), was taken at least three times during the trial.

An incremental exercise test to exhaustion was conducted on a motor driven treadmill (Quinton Instruments, Seattle, WA, USA) according to the protocol of Noakes et al (1990), to determine the subject's maximum oxygen consumption (VO_2max , $\text{ml.kg}^{-1}.\text{min}^{-1}$), peak treadmill running speed (PTRS, km.h^{-1}) and maximum heart rate (HRmax , b.min^{-1})²⁰². A face mask was secured over the athlete's nose and mouth to collect expired O_2 and CO_2 and the Oxycon Alpha (Jaeger, Germany) was used to measure VO_2 during the test. A 5-minute warm up at 8-10 km.h^{-1} and 0% gradient preceded the start of the test. The subject began the test running at 10-12 km.h^{-1} , after which the speed was increased by 0.5 km.h^{-1} every 30 seconds, until the subject felt he/she could not continue. During the last 5 seconds of each 30-second interval, heart rate was recorded using a heart rate monitor (Vantage XL, Polar Electro, Kempele, Finland), and the subjects were asked to rate their perceived exertion (RPE), using the numerical Category Ratio (CR-10) Borg scale (Appendix 3). The test was terminated at volitional exhaustion, and maximal achievement was accepted if the subject reached two of the following criteria: (i) a rating of 8-10 on the Borg CR-10 RPE scale (ii) a peak heart rate similar to their age-predicted maximal heart rate (iii) a respiratory exchange ratio (RER) of > 1.10. VO_2max ($\text{ml.kg}^{-1}.\text{min}^{-1}$), maximum heart rate (b.min^{-1}) and PTRS (km.h^{-1}) were defined as the highest respective measurements recorded during the last full 30-second interval completed.

2.2.2.2 Exercise monitoring and assessment of training duration

Prior to the start of the study subjects were asked to maintain a constant training load for five weeks. They were not told that they were going to be asked to recall their training duration during the first three weeks as we did not want them to prepare specifically for this. During the subsequent two weeks participants wore a Vantage XL Polar heart rate monitor (Polar Electro, Kempele, Finland) during all of their exercise training sessions, allowing duration to be recorded and the average duration per week calculated (min.wk^{-1}). The relationship between self-reported and recorded average weekly duration of exercise was then assessed.

2.2.2.3 Division of subjects into groups

Participants were divided into three groups based on whether they over-estimated, under-estimated or accurately estimated the duration of training they did during the first three weeks compared to during the last two weeks of the study. That is, those subjects that increased their training (Group I, n = 5) had under-estimated their training duration during the first three weeks; those that decreased their training duration (Group D, n = 7) had over-estimated the amount of training they did in the first three weeks; and those that kept their training the same (Group S, n = 17) had correctly estimated the training they were doing. This was done by calculating the percent difference in average weekly training duration for the self-reported training (first three weeks) and the recorded training (last two weeks) (Equation 1).

$$\% \text{ difference} = \frac{(\text{min.wk}^{-1} \text{ self-reported} - \text{min.wk}^{-1} \text{ recorded})}{\text{min.wk}^{-1} \text{ self-reported}} \times 100 \dots \dots \dots (1)$$

A variation of 20% was selected as a reasonable range with which to account for normal intra-individual variation, as it is unrealistic to expect athletes, when asked to maintain their current training load, to keep their training *exactly* the same. As such, those subjects that maintained their training within a 20% range were regarded as having kept their training duration constant throughout the trial. The baseline measurements for each group were then compared to assess if there might be characteristics particular to athletes that either over-estimated, under-estimated or accurately estimated their training duration.

2.2.3 Statistical Analyses

A Pearson's Product Moment correlation was performed to assess the relationship between self-reported and recorded average weekly training duration. The 95% confidence intervals around the correlation coefficient were calculated using a spreadsheet for this purpose downloaded from www.sportsci.org²⁰³. The limits of agreement between self-reported and measured training duration were calculated using a Bland-Altman plot²⁰⁴. A Kruskal-Wallis test was used to compare differences in anthropometric and physiologic measurements between the groups that under-

estimated training (Group I), over-estimated training (Group D), or accurately assessed training (Group S). Statistical analysis was performed using STATISTICA 7.0 (StatSoft, Inc (2004), data analysis software system) and statistical significance was accepted at $p < 0.05$.

2.3 RESULTS

Baseline measurements are shown in Table 2.1 for all athletes ($n = 29$) and the 3 groups based on whether athletes had over-estimated (Group D, $n = 7$), under-estimated (Group I, $n = 5$) or accurately self-reported (Group S, $n = 17$) the average weekly duration of endurance exercise they were doing during the first 3 weeks.

Table 2.1: Subject characteristics ($n = 29$; 12 males, 17 females). Group I are the subjects who under-estimated training, group D over-estimated training and group S accurately assessed their training. Data are expressed as mean \pm SD

Subject characteristics	Group I ($n = 5$)	Group D ($n = 7$)	Group S ($n = 17$)	All ($n = 29$)
Age (years)	29 \pm 7	30 \pm 7	30 \pm 4	30 \pm 5
Mass (kg)	58 \pm 8	64 \pm 10	68 \pm 14	65 \pm 12
Stature (cm)	164 \pm 6	168 \pm 10	171 \pm 9	169 \pm 8
Body fat (%)	18 \pm 7	22 \pm 7	20 \pm 8	20 \pm 7
VO ₂ max (ml.min ⁻¹ .kg ⁻¹)	56 \pm 8	55 \pm 5	56 \pm 9	56 \pm 8
Heart rate max (b.min ⁻¹)	191 \pm 10	198 \pm 9 *	187 \pm 6 *	190 \pm 9
Peak treadmill running speed (km.h ⁻¹)	16 \pm 3	17 \pm 1	17 \pm 2	17 \pm 2
Average resting HR during trial (b.min ⁻¹)	56 \pm 7	59 \pm 7	54 \pm 7	56 \pm 7

* Group D significantly different from Group S ($p = 0.025$)

When assessing how accurately the participants self-reported the duration of training they were doing it was found that 17 (9 female, 8 male) of the 29 athletes (59%) kept their training duration constant, suggesting they had accurately self-reported their current average weekly endurance training duration. Seven athletes (5 female, 2 male; 24%) decreased their training duration, i.e. they had over-estimated their average weekly training when self-reporting after the first three weeks. Five athletes

(3 female, 2 male; 17%) increased their training, indicating they had under-estimated the duration of cardiovascular training they had been doing in the first three weeks. There was a significant relationship ($r = 0.87$; 95% CI: 0.74 – 0.94) between self-reported and recorded average weekly duration (Figure 2.1).

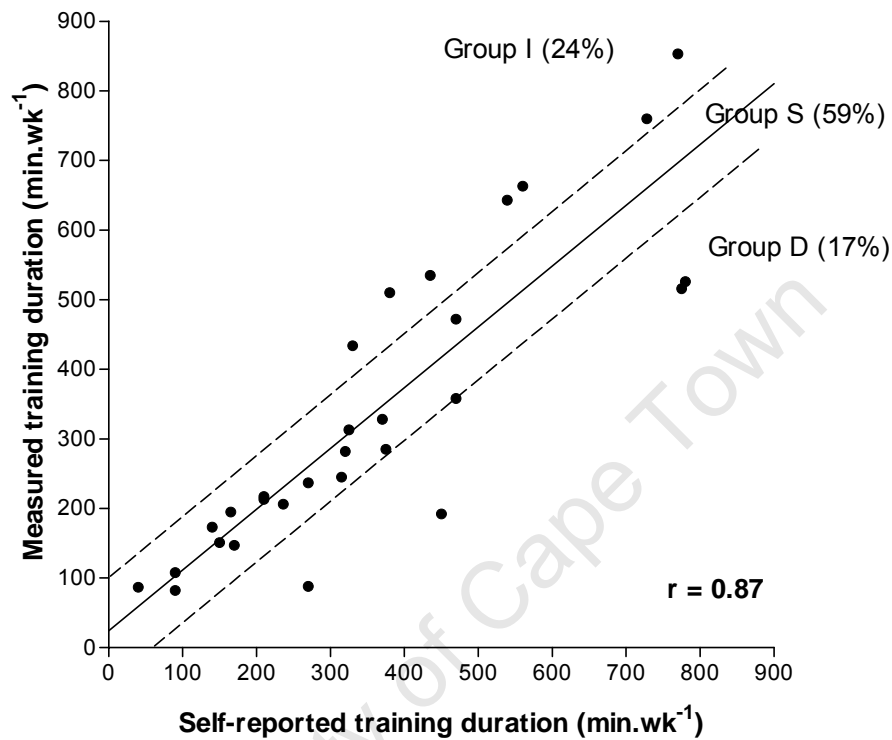


Figure 2.1: Relationship between self-reported training duration (min.wk⁻¹) and measured training duration (min.wk⁻¹) (n = 29).

Figure 2.2 shows the Bland-Altman plot of self-reported and measured training duration²⁰⁴. The limits of agreement were -193 to 235 min.wk⁻¹. The variation around the mean difference (21 min.wk⁻¹) appeared to be random and without bias.

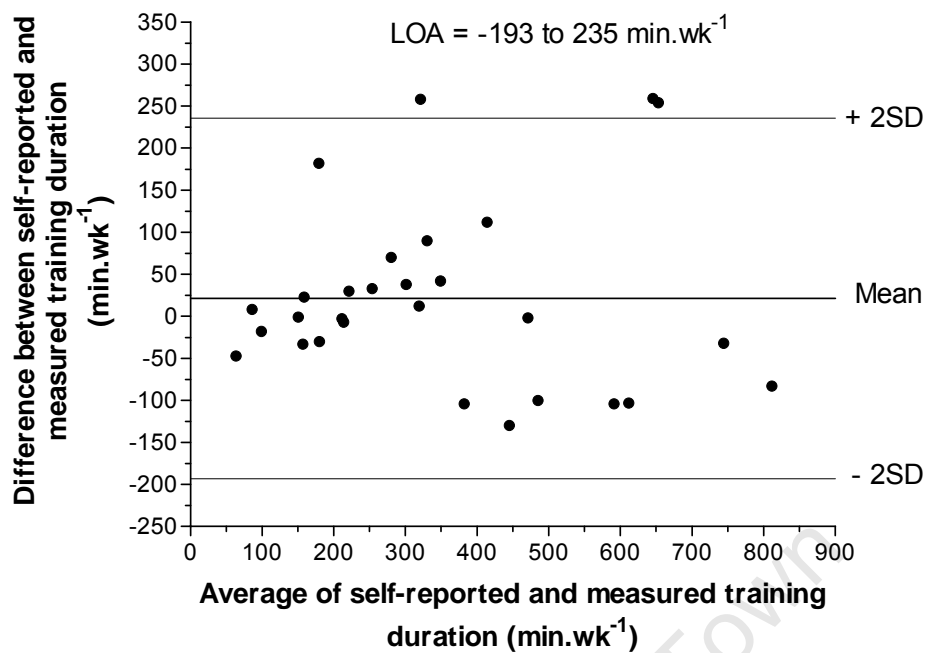


Figure 2.2: Bland Altman plot of self-reported and measured training duration (n = 29)

There were no significant differences in subject characteristics between the three groups except for maximal heart rate measured during the maximal treadmill test ($p = 0.030$). The maximal heart rate of Group S ($187 \pm 6 \text{ b.min}^{-1}$) was significantly lower than the maximal heart rate of Group D ($198 \pm 9 \text{ b.min}^{-1}$) ($p = 0.025$), despite the age of the two groups being similar (Table 2.1).

2.4 DISCUSSION

The first finding of this study was that more than half the athletes (59%) accurately self-reported the average weekly duration of their endurance training they had done for three weeks. Approximately one quarter (24%) of the athletes over-estimated their average weekly training duration and only 17% under-estimated the duration of training they were doing. Although the correlation between self-reported and recorded average weekly training duration may at first glance appear to be good ($r = 0.87$), this correlation means that 24% of the variance in self-reported duration cannot be accounted for by the measured duration. This margin of error may have a significant impact if the data are used to prescribe exercise training. One also needs to consider that duration is probably one of the most easily quantifiable components

of training. As such it is reasonable to expect even greater variance in self-reported data involving other more complex components of training such as intensity. Similar discrepancies between self-reported and actual training data were found by Gilman and Wells (1993)²⁰⁵. In this study athletes reported having completed three sessions of easy intensity training, less than one session of moderate intensity training, and one or two sessions of hard intensity training per week. However, heart rate data revealed that 45% of their training had been of moderate intensity, and only 9% of their training time had been in the hard intensity zone. The authors concluded that the runners over-estimate the amount of time they spend performing high intensity training, making it necessary to monitor training intensity with more objective measures such as heart rate²⁰⁵.

The accuracy with which an athlete reports their training may be influenced by external factors. For example, duration and frequency may be over-reported if the athlete is influenced by the response they believe is sought by the coach or investigator. Perceptions of training intensity may differ depending on experience or tolerance of the person, particularly if asked to report intensity as simply light, moderate, hard or very hard^{6:7}. Therefore, although questionnaires may assist with monitoring general trends in population activity, attempts to quantify exercise dosage from data collected with questionnaires remain inadequate⁷. Where possible physiological measurements should corroborate self-reported data, or should be used to report the validity of the data used so that it can be considered when assessing the results.

In an attempt to identify specific physiological characteristics of people who over-estimate, under-estimate or accurately estimate the amount of training they are doing, maximal heart rate was the only characteristic measured that differed among the three groups. Specifically, the athletes that over-estimated their training duration had a significantly higher maximal heart rate during the maximal treadmill test compared to the subjects that accurately estimated their training, despite the age of the two groups being similar. The reason behind this is unclear. While it has been suggested that maximal heart rate decreases with endurance training¹⁵⁵ there were no apparent differences in the average state of training of the three groups, certainly based on the mean VO_2 max of each group (Table 2.1). It is not known whether there may be other factors, not tested in this study, which may affect HRmax and which

may be common to the athletes in Group D. Thus, whether this characteristic can be used to predict which subjects are prone to over-estimating their training duration requires further investigation.

2.5 CONCLUSIONS

This study therefore highlights that the quantification of an athlete's training may be inaccurate when relying exclusively on self-reported data, which may in turn influence the effectiveness of customised training programmes. It is recommended that the margin of error introduced by the use of self-reported data in research studies and coaching prescription should be accounted for, or where possible physiological measurements should be used to corroborate self-reported data.

As previously mentioned, important weaknesses in the use of questionnaires are the subjective nature of the data collected, and the dependence on human memory to supply the data. Recently theoretical indices of training stress have been developed that may reduce the error introduced by using self-reported data in the quantification of training load. Most of these methods incorporate objective measures such as heart rate, but one method has introduced the subjective measure of RPE in an attempt to simplify the calculation and measurement of training load. The relationship between two popular objective equations and one subjective equation are discussed in Chapter 3.

CHAPTER 3

<p>QUANTIFYING TRAINING LOAD – A COMPARISON OF SUBJECTIVE AND OBJECTIVE METHODS</p>
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Data in this chapter has been accepted for publication in the
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3.1 INTRODUCTION

The ability to accurately measure and monitor training load has the potential to provide valuable information for the prescription of individualized exercise programmes. Banister and co-workers (1991) proposed that an athlete's heart rate response to exercise, combined with exercise duration, may represent an objective measure of physical effort which allows the quantification of a training session into a unit "dose" of physical effort⁴⁴. This concept has been named a "Training Impulse" (TRIMP), the equation for which is explained in detail by Banister (1991)⁴⁴. The ability to quantify training to a single figure/factor is appealing in terms of its practical application. The use of the TRIMP equation requires steady state heart rate measurements⁴⁴, which may limit the quantification of exercise consisting of alternating bouts of high intensity exercise and recovery. Further methods of quantifying training load have therefore evolved and are currently used in research. For example the Summated Heart Rate Zone method, proposed by Edwards (1993), may facilitate the quantification of interval training since it divides the training session into duration spent in each of five heart rate zones (50-60%, 60-70%, 70-80%, 80-90% and 90-100% of maximal heart rate)⁴⁵. Duration in each zone is multiplied by a different factor, which weights the higher intensity zones more than the lower intensities. Thereafter the adjusted scores are summated⁴⁵. Recently a modified version of the Summated Heart Rate Zone equation has been used by Earnest et al (2004) and Lucia et al (2003) and referred to as "Lucia's TRIMP" by Impellizzeri et al (2004)⁴⁶⁻⁴⁸. In this method the duration spent in each of three heart rate zones (zone 1: below the ventilatory threshold; zone 2: between the ventilatory threshold and the respiratory compensation point; zone 3: above the respiratory compensation point) is multiplied by a coefficient (k) relative to each zone (k = 1 for zone 1, k = 2 for zone 2, and k = 3 for zone 3) and the adjusted scores are then summated. The original source of this equation however was not referenced in these papers.

Foster et al (1996) simplified the quantification of training load by substituting a Session RPE measure for the use of heart rate data⁴⁹. An exercise Session Load is calculated by multiplying Session RPE (Appendix 3) by the duration of the aerobic exercise bout⁴⁹. The Session RPE method is the only subjective measure of training load that has been developed and popularised. It was developed with the goal of simplifying, yet remaining equivalent to the objective measures of assessing training

load (i.e. TRIMP and Summated HR Zones methods). Foster et al (1998, 2001) evaluated the relationship between the subjective Session RPE method and the objective Summated Heart Rate Zone method and found the individual correlations between the two techniques ranged from $r = 0.75$ to $0.90^{49;51}$. The authors concluded that the Session RPE method was a valid and reliable measure of exercise intensity in aerobic exercise when compared to heart rate-based methods^{49;51}.

Impellizzeri et al (2004) studied the relationship between the Session RPE method and three objective methods: Banister's TRIMP, Edwards' Summated Heart Rate Zone method and Lucia's TRIMP in soccer players while training and playing matches⁴⁶. Individual correlations between the Session RPE method and Banister's TRIMP method ranged between $r = 0.50$ and 0.77 ; individual correlations between the Session RPE method and Edward's Summated Heart Rate Zone method ranged from $r = 0.54$ to 0.78 ; and individual correlations between the Session RPE method and Lucia's TRIMP methods ranged between $r = 0.61$ and 0.85^{46} . However, there appears to be no evidence that either the TRIMP, Summated Heart Rate Zones score or Lucia's TRIMP equations have been validated, posing the question of the legitimacy of validating the Session RPE method against these heart rate-based methods. Collectively the data from these studies suggest that the relationship between the different methods of quantifying training load varies. The reasons for the variations have not been clearly explained. Therefore the aim of this study was to assess the relationships between the Session RPE method and the TRIMP and Summated Heart Rate Zone method in the quantification of *ad libitum* training and to identify characteristics that may explain the variance not accounted for in the relationship between the objective and subjective methods of quantifying training load.

3.2 METHODS

3.2.1 Study Design and Subjects

Thirty-three habitually physically active men ($n = 15$) and women ($n = 18$) were recruited for this two-week trial from the local community, gyms and running clubs. A

Physical Activity Readiness Questionnaire (PAR-Q) (Appendix 8) was administered to all participants prior to their enrolment in the study, to ensure that they were not at any health risk while performing exercise during the study. Before the start of the trial the body composition, maximal oxygen consumption and maximal heart rate of the participants was measured. Thereafter the participants trained *ad libitum* for two weeks during which time their training was quantified simultaneously using the Session RPE, TRIMP and Summated Heart Rate Zone methods.

This prospective observational cohort study was approved by the University of Cape Town Ethics and Research Committee and carried out in accordance with the principles outlined in the Declarations of Helsinki²⁰⁶. The participants gave their informed consent after the testing protocol was explained to them.

3.2.2 Methodology

3.2.2.1 Anthropometric and physiologic measurements

Body mass was recorded to the nearest 100 g and the stature of each athlete was recorded to the nearest mm (Seca model 708, Germany). Body fat percent was assessed with a near infrared (NIR) measurement on the right bicep using the Futrex-6100A/ZL (Kett Electric Laboratory, Futrex Inc. Gaithersburg, MD, USA). Resting heart rate, recorded immediately after waking in the morning with a Vantage XL heart rate monitor (Polar Electro, Kempele, Finland), was taken at least three times during the trial. An incremental exercise test to exhaustion was conducted on a motor driven treadmill, as described in Chapter 2, to determine the subject's maximum oxygen consumption (VO_2max , $\text{ml}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$), peak treadmill running speed (PTRS, $\text{km}\cdot\text{h}^{-1}$) and maximum heart rate (HRmax , $\text{b}\cdot\text{min}^{-1}$).

3.2.2.2 Quantification of training load

All the training data collected and analysed in this study was cardiovascular in nature. The training of 19 of the 33 participants consisted exclusively of running, whereas four other runners also engaged in some gym training. Gym training consisted mainly of interval-type training which combined cycling, stepping, spinning

and treadmill/interval running. Subjects also reported using elliptical machines, the super-circuit, cross-trainers or rowing machines. Five participants engaged in gym training exclusively. Four subjects did approximately equal amounts of running and cycling, and one subject only cycled. During the two weeks of the trial each participant wore a heart rate monitor for each exercise training session, to record heart rate ($\text{b}\cdot\text{min}^{-1}$) and exercise duration (min). Subjects completed a daily training diary in which they recorded exercise mode and Session RPE (Appendix 3) for each training bout. Three equations were used to calculate training load for each of the training sessions performed during the trial:

- *Training Impulse (TRIMP)* ⁴⁴:

$$\text{TRIMP (w(t))} = \text{duration of training (min)} \times \Delta\text{HR ratio} \times Y \dots\dots\dots(2)$$

- $\Delta\text{HR ratio} = \frac{\text{HR}_{\text{ex}} - \text{HR}_{\text{rest}}}{\text{HR}_{\text{max}} - \text{HR}_{\text{rest}}}$

Where: HR_{rest} = resting heart rate,
 HR_{ex} = average heart rate during exercise,
 HR_{max} = maximum heart rate.

- $Y = 0.64e^{1.92x}$ (for males) and $Y = 0.86e^{1.67x}$ (for females),

Where: $e = 2.712$ and $x = \Delta\text{HR ratio}$.

- *Summated Heart Rate Zone Method* ⁴⁵:

$$\text{Training load} = (\text{duration (min) in Zone 1} \times 1) + (\text{duration in Zone 2} \times 2) + (\text{duration in Zone 3} \times 3) + (\text{duration in Zone 4} \times 4) + (\text{duration in Zone 5} \times 5) \dots\dots\dots(3)$$

Where: Zone 1 = 50-60% of maximum heart rate,
 Zone 2 = 60-70% HR_{max} , Zone 3 = 70-80% HR_{max} ,
 Zone 4 = 80-90% HR_{max} , Zone 5 = 90-100% HR_{max}

- *Session RPE Method* ⁴⁹:

$$\text{Session load} = \text{Duration (min)} \times \text{Session RPE} \dots\dots\dots(4)$$

In this method the athlete's perception of the overall difficulty of the training bout was recorded 30 minutes after the completion of the exercise⁴⁹ according to the scores in Appendix 3. The Session RPE scale is based on the Borg Category Ratio (CR-10) RPE Scale which

translates the athlete's perception of effort into a numerical score between 0 and 10 (Appendix 3). The athlete is asked to respond to the simple question: "*How was your workout?*" with the goal of getting an uncomplicated response that reflects the athlete's global impression of the workout⁴⁹.

Session training loads were summated for each subject for the two weeks to calculate total training load for each method. The equations and the standard error of the estimate (SEE) defining the relationship between the total training loads for the three methods were calculated using a linear regression equation (Graphpad Prism v3 for Windows, GraphPad Software, San Diego California USA). The data points that occurred outside the SEE of the regression equation were identified and grouped according to whether they were above (OVER) or below (UNDER) the SEE of the regression line. The ACCURATE group consisted of those subjects within the SEE around the regression line. The anthropometric, physiological and training characteristics of the three groups were then compared.

3.2.3 Statistical Analyses

A Pearson's Product Moment correlation was performed to assess the relationship between the objective (heart rate-based) and subjective (RPE-based) methods of quantifying Training Load (for each session for each subject and with pooled data for each subject). The 95% confidence intervals around the correlation coefficient were calculated using a spreadsheet for this purpose downloaded from Hopkins (2006)²⁰³. A Levene's test was used to check for the homogeneity of variance between the groups and a Kolmogorov-Smirnov test was used to test for normality. These tests showed unequal variance and that the data were not normally distributed, thus requiring the use of non-parametric tests thereafter. A Kruskal-Wallis test was used to compare the three groups (OVER, UNDER and ACCURATE) that were identified based on their position relative to the SEE of the regression equation. Statistical analysis was performed using STATISTICA 7.0 data analysis software system (StatSoft, Inc. Tulsa, OK, USA) and statistical significance was accepted at $p < 0.05$.

3.3 RESULTS

The 33 participants in this study had a mean (\pm SD) age of 30 ± 5 years, mass of 67 ± 13 kg, height 171 ± 10 cm, body fat $20 \pm 7\%$, $VO_{2\max}$ 56 ± 8 ml.min⁻¹.kg⁻¹ and resting heart rate of 55 ± 7 b.min⁻¹. The subjects had an average of 11 ± 4 training sessions in the two weeks. The variation in training load was large for each subject. For example the average coefficient of variation for total training load for each subject determined over the 2 weeks was 75% (95% CI: 63 to 87%) for the Session RPE method, 78% (95% CI: 68 to 89%) for TRIMP and 74% (95% CI: 63 to 85%) for the Summated Heart Rate Zones method. Relationships between the three different methods were then evaluated to establish whether the two heart rate-based and the RPE-based methods provided similar assessments of training load.

3.3.1 TRIMP vs. Summated Heart Rate Zones Method

There was a correlation of $r = 0.98$ (95% CI: 0.96 to 0.99) between total training load calculated using the TRIMP and the Summated Heart Rate Zones method (pooled data) (Figure 3.1).

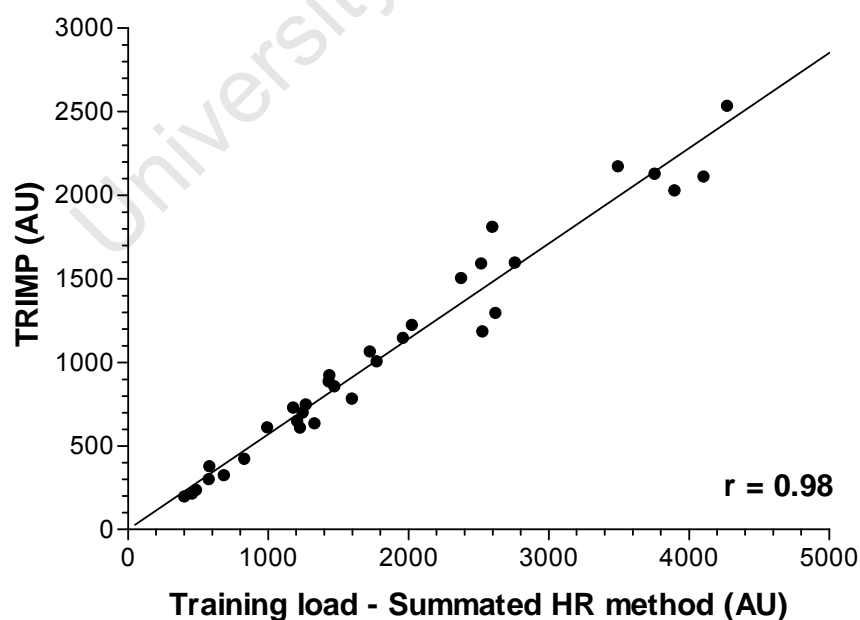


Figure 3.1: Relationship between Total Training Load calculated using TRIMP and the Summated HR Zones method.

3.3.2 TRIMP vs. Session RPE Method

There was a correlation of $r = 0.76$ (95% CI: 0.56 to 0.88) between total training loads calculated using TRIMP and the Session RPE method (Figure 3.2) (pooled data). The 95% CI for the correlation coefficients determined for the session training loads of each subject was $r = 0.84$ to 0.93 (i.e. within-subject data) (Table 3.1).

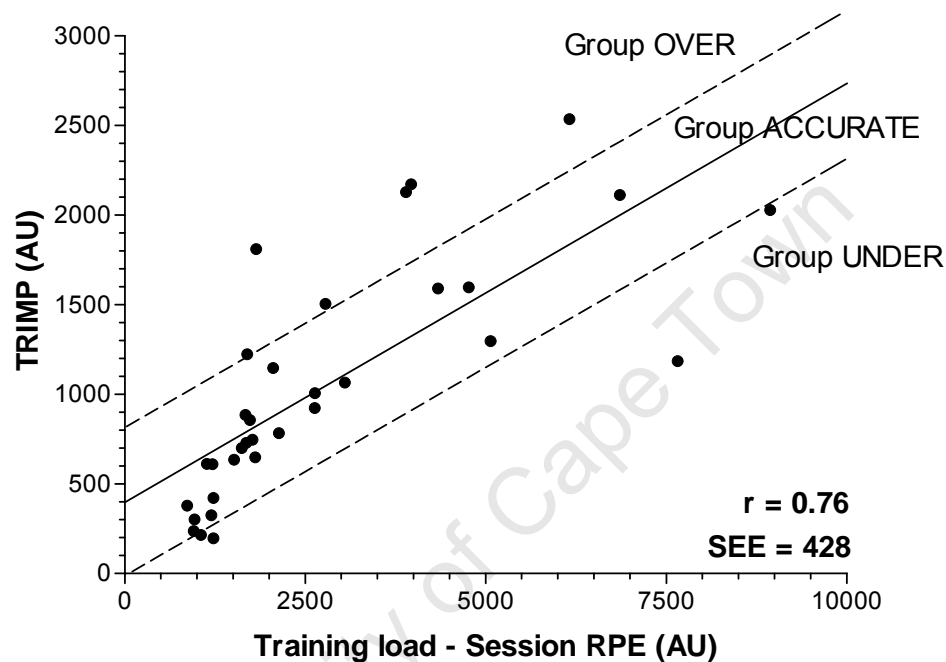


Figure 3.2: Relationship between Total Training Load calculated using TRIMP and the Session RPE method. Groups were based on whether the regression equations over-predicted (OVER), under-predicted (UNDER) or accurately predicted (ACCURATE) the relationship between the methods.

The position of the data points for the OVER group lying above the regression line in Figure 3.2 indicates that the TRIMP method over-estimated the calculation of training load when compared to the Session RPE method for the OVER group. Conversely, the data points of the UNDER group lying below the regression line in Figure 3.2 indicates that the TRIMP method under-estimated the calculation of training load compared to the Session RPE method for the UNDER group.

Table 3.1: Within-subject correlations between TRIMP and Session RPE method, and between Summated HR Zone and Session RPE method. The number of exercise sessions included in the calculation of the correlation coefficient (r) for each subject is presented.

Participant	TRIMP vs. RPE (r)	Summated HR vs. RPE (r)	No. of sessions
1	0.84	0.88	12
2	0.97	0.97	14
3	0.98	0.99	14
4	0.90	0.93	12
5	0.91	0.90	7
6	0.94	0.95	7
7	0.90	0.93	8
8	0.93	0.83	13
9	0.75	0.80	8
10	0.95	0.94	7
11	0.98	0.97	10
12	1.00	1.00	10
13	0.84	0.81	7
14	0.94	0.95	19
15	0.95	0.94	7
16	0.96	0.96	13
17	0.51	0.74	11
18	0.94	0.93	11
19	0.99	0.99	6
20	0.72	0.76	11
21	0.75	0.78	5
22	0.83	0.89	9
23	0.99	1.00	6
24	0.54	0.53	5
25	0.98	0.98	9
26	0.97	0.99	14
27	0.78	0.82	10
28	0.93	0.96	15
29	0.90	0.92	16
30	0.97	0.96	11
31	0.69	0.73	14
32	0.95	0.96	7
33	0.98	0.97	19
Range	0.51 - 1.00	0.53 - 1.00	5 - 19

Baseline anthropometric, physiological and training characteristics are shown in Table 3.2 for the OVER group (n = 6), UNDER group (n = 4) and ACCURATE group (n = 23), i.e. the three groups identified based on their position relative to the SEE of the regression equation for the correlation between the Session RPE method and TRIMP.

Table 3.2: Subject characteristics for athletes in OVER (n = 6), UNDER (n = 4) and ACCURATE (n = 23) groups, in the correlation between the Session RPE method and the TRIMP method. Groups were based on whether the regression equations over- (OVER), under- (UNDER) or accurately predicted (ACCURATE) the relationship between the methods (pooled data). Data are expressed as mean \pm SD.

Subject characteristics	OVER	UNDER	ACCURATE
Age (years)	31 \pm 6	27 \pm 5	30 \pm 5
Mass (kg)	58 \pm 10	73 \pm 14	68 \pm 13
Stature (cm)	170 \pm 9	175 \pm 12	170 \pm 10
Body fat %	19 \pm 6	17 \pm 3	20 \pm 7
VO ₂ max (ml.min ⁻¹ .kg ⁻¹)	57 \pm 10	61 \pm 7	55 \pm 8
HRmax (b.min ⁻¹)	181 \pm 10	193 \pm 9	192 \pm 9
Peak treadmill running speed (km.h ⁻¹)	17 \pm 3	18 \pm 1	17 \pm 2
Resting heart rate (b.min ⁻¹)	50 \pm 4	52 \pm 10	57 \pm 7
Total training duration (2 wks) (min)	894 \pm 325 #	698 \pm 614	461 \pm 300
Ave training HR (b.min ⁻¹)	144 \pm 11	132 \pm 10	148 \pm 13
Percent time in Zone 1 (%)	3 \pm 3 \$	15 \pm 8 @	5 \pm 3
Percent time in Zone 2 (%)	7 \pm 6 \$	17 \pm 6	13 \pm 6
Percent time in Zone 3 (%)	22 \pm 6	30 \pm 9	27 \pm 11
Percent time in Zone 4 (%)	46 \pm 8 \$	25 \pm 10	36 \pm 9
Percent time in Zone 5 (%)	17 \pm 10	3 \pm 1 @	17 \pm 14
Training load (RPE method, AU) *	3383 \pm 1671	4717 \pm 4164	2298 \pm 1549
Training Impulse (TRIMP, AU)	1896 \pm 480 #	907 \pm 879	853 \pm 461
Training load (Summated HR Zone method, AU)	3084 \pm 881 #	1819 \pm 1701	1520 \pm 840

OVER significantly different from ACCURATE

\$ OVER significantly different from UNDER

@ UNDER significantly different from ACCURATE

* This comparison should be interpreted with caution because the interpretation of RPE scales may vary between individuals.

OVER had a higher total training duration over two weeks (894 ± 325 min vs 461 ± 300 min, $p = 0.031$), and training load (using the Summated Heart Rate Zone method, 3084 ± 881 AU vs. 1520 ± 840 AU, $p = 0.015$) than ACCURATE (Table 3.2). OVER spent 21% more of their total training time in heart rate zone 4 than UNDER ($46 \pm 8\%$ vs. $25 \pm 10\%$, $p = 0.008$). OVER had a higher total TRIMP than both ACCURATE (1896 ± 480 AU vs. 853 ± 461 AU, $p = 0.004$) and UNDER (1896 ± 480 AU vs. 907 ± 879 AU, $p = 0.045$). UNDER spent 12% more of their total training time in heart rate zone 1 than OVER ($15 \pm 8\%$ vs. $3 \pm 3\%$, $p = 0.005$) and 10% more of their training time in heart rate zone 1 than ACCURATE ($15 \pm 8\%$ vs. $5 \pm 3\%$, $p = 0.020$). UNDER also spent 10% more of their total time training in heart rate zone 2 than OVER ($17 \pm 6\%$ vs. $7 \pm 6\%$, $p = 0.039$). ACCURATE spent 14% more of their total training time in zone 5 than UNDER ($17 \pm 14\%$ vs. $3 \pm 1\%$, $p = 0.031$). UNDER had a non-significantly lower average training heart rate than ACCURATE ($p = 0.052$). All groups spent similar proportions of their training time in heart rate zones 2 and 3.

3.3.3 Summated Heart Rate Zone Method vs. Session RPE Method

A correlation of $r = 0.84$ (95% CI: 0.70 to 0.92) was found when comparing the Summated Heart Rate Zone method with the Session RPE method in the calculation of total training load (Figure 3.3) (pooled data). The 95% CI for the correlation coefficients determined for the session training loads of each subject was $r = 0.86$ to 0.94 (i.e. within-subject data) (Table 3.1).

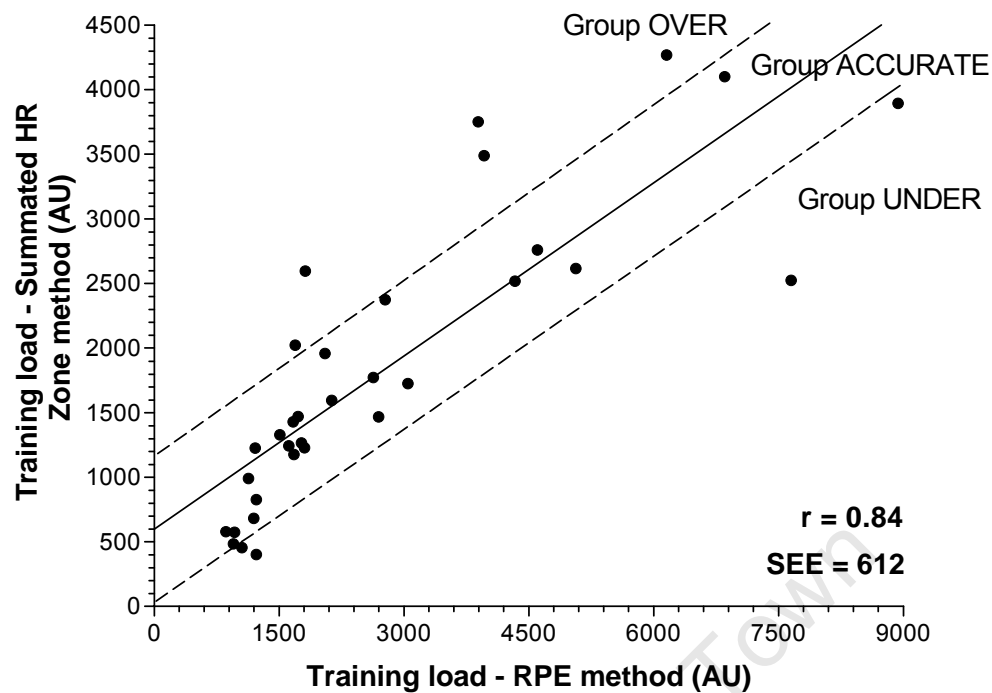


Figure 3.3: Relationship between Total Training Load calculated using the Summated Heart Rate Zone method and the Session RPE method. Groups were based on whether the regression equations over-predicted (OVER), under-predicted (UNDER) or accurately predicted (ACCURATE) the relationship between the methods.

The position of the data points for the OVER group lying above the regression line in Figure 3.3 indicates that the Summated Heart Rate Zone method over-estimated the calculation of training load when compared to the Session RPE method for the OVER group. Conversely, the data points of the UNDER group lying below the regression line in Figure 3.3 indicates that the Summated Heart Rate Zone method underestimated the calculation of training load compared to the Session RPE method for the UNDER group.

Baseline anthropometric, physiological and training characteristics are shown in Table 3.3 for OVER ($n = 5$), UNDER ($n = 4$) and ACCURATE ($n = 24$), the three groups identified based on their position relative to the SEE of the regression equation for the correlation between the Session RPE method and the Summated Heart Rate Zone method.

Table 3.3: Subject characteristics for OVER (n = 5), UNDER (n = 4) and ACCURATE (n = 24) groups, for the correlation between the Session RPE method and the Summated Heart Rate Zones method. Groups were based on whether the regression equations over- (OVER), under- (UNDER) or accurately predicted (ACCURATE) the relationship between the methods (pooled data). Data are expressed as mean \pm SD.

Subject characteristics	OVER	UNDER	ACCURATE
Age (years)	32 \pm 6	27 \pm 5	30 \pm 5
Mass (kg)	55 \pm 9	73 \pm 14	68 \pm 13
Stature (cm)	167 \pm 8	175 \pm 12	171 \pm 10
Body fat %	21 \pm 3	17 \pm 3	20 \pm 8
VO ₂ max (ml.min ⁻¹ .kg ⁻¹)	54 \pm 6	61 \pm 7	56 \pm 9
HRmax (b.min ⁻¹)	179 \pm 11	193 \pm 9	191 \pm 9
Peak treadmill running speed (km.h ⁻¹)	16 \pm 3	18 \pm 1	17 \pm 2
Total training duration (2 wks) (min)	944 \pm 336 #	698 \pm 614	469 \pm 296
Ave training HR (b.min ⁻¹)	143 \pm 11	132 \pm 10 @	148 \pm 12
Resting HR (b.min ⁻¹)	50 \pm 5	52 \pm 10	56 \pm 7
Percent time in Zone 1 (%)	4 \pm 3 \$	15 \pm 8 @	5 \pm 3
Percent time in Zone 2 (%)	8 \pm 6	17 \pm 6	12 \pm 6
Percent time in Zone 3 (%)	22 \pm 7	30 \pm 9	26 \pm 11
Percent time in Zone 4 (%)	45 \pm 8 \$	25 \pm 10	37 \pm 9
Percent time in Zone 5 (%)	17 \pm 11	3 \pm 1 @	17 \pm 13
Training load (RPE method, AU)*	3504 \pm 1839	4717 \pm 4164	2314 \pm 1508
Training Impulse (TRIMP, AU)	1974 \pm 492 #	907 \pm 879	880 \pm 470
Training load (Summated HR Zone method, AU)	3226 \pm 905 #	1819 \pm 1701	1555 \pm 839

OVER significantly different from ACCURATE

\$ OVER significantly different from UNDER

@ UNDER significantly different from ACCURATE

* This comparison should be interpreted with caution because the interpretation of RPE scales may vary between individuals.

The OVER group had a higher Training load (Summated HR Zone method, 3226 \pm 905 AU vs. 1555 \pm 839 AU, $p = 0.020$), TRIMP (1974 \pm 492 AU vs. 880 \pm 470 AU, $p = 0.007$) and total training duration (944 \pm 336 min vs. 469 \pm 296 min, $p = 0.044$) than ACCURATE; and higher TRIMP than UNDER (1974 \pm 492 AU vs. 907 \pm 879 AU, $p = 0.043$) (Table 3.3). OVER spent 20% more of their total training time in Zone 4 than UNDER (45 \pm 8% vs. 25 \pm 10%, $p = 0.018$). UNDER had a lower average training heart rate than ACCURATE (132 \pm 10 b.min⁻¹ vs. 148 \pm 12 b.min⁻¹, $p = 0.048$).

UNDER spent 11% more of their total training time in zone 1 than OVER ($15 \pm 8\%$ vs. $4 \pm 3\%$, $p = 0.013$) and spent 10% more of their total training time in zone 1 than ACCURATE ($15 \pm 8\%$ vs. $5 \pm 3\%$, $p = 0.015$). ACCURATE spent 14% more of their total training time in zone 5 than UNDER ($17 \pm 13\%$ vs. $3 \pm 1\%$, $p = 0.028$). All three groups spent a similar amount of their total training time in heart rate zones 2 and 3.

3.4 DISCUSSION

An extensive review of the literature confirmed that the current trend is towards the calculation of global measures of training load such as TRIMP, Summated Heart Rate Zones method and Session RPE. For this reason these methods were chosen for this investigation. The first finding of this study was that training load calculated for *ad libitum* training using the TRIMP equation correlated best with the Summated Heart Rate Zone method. This is not surprising since both methods use the direct physiological measure of heart rate as a fundamental part of the calculation. It has been mentioned previously however, that the use of the TRIMP equation requires only steady state heart rate measurements⁴⁴, which would limit its use in the quantification of interval training. It has also been suggested that the Summated Heart Rate Zone method⁴⁵ may better facilitate the quantification of this type of training. However, the strong correlation found between these two methods in this study suggests that both may be suitable for the quantification of continuous as well as interval training sessions.

The current study found that the Summated Heart Rate Zone method correlated well with the Session RPE method explaining approximately 71% of the variance. The relationship between TRIMP and the Session RPE method was the weakest of the three correlations, accounting for only 58% of the variance. Foster et al (1998, 2001) evaluated the relationship between the subjective Session RPE method and the objective Summated Heart Rate Zone method of quantifying training load during different forms of exercise^{51;52}. They found that although the Summated Heart Rate Zone method gave lower scores than the Session RPE method (due to different units), the pattern of differences between the two methods was very consistent, however no correlation coefficients were provided in Foster et al (2001). Individual correlations between the two methods ranged from $r = 0.75$ to 0.90 in Foster et al

(1998) however no statistical methods were explained in the paper. They concluded that Session RPE was a valid and reliable measure of exercise intensity in aerobic exercise when compared to heart rate-based measures^{51;52}. Impellizzeri et al (2004) studied the relationship between the Session RPE method and three objective methods of quantifying training load during soccer training and matches⁴⁶. In their study individual correlations between the Session RPE method and TRIMP method ranged between $r = 0.50$ and 0.77 ; whereas individual correlations between the Session RPE method and the Summated Heart Rate Zone method ranged from $r = 0.54$ to 0.78 ⁴⁶.

The content of the 50 training sessions performed by seven subjects in the study of Foster et al (1998) was not explained, but the 12 subjects in the study of Foster et al (2001) performed three steady state and five interval sessions^{51;52}. Most training in the study of Impellizzeri et al (2004) consisted of “small-sided” games, with sprint and plyometric exercises being performed once a week⁴⁶. The increased use of the oxygen-independent energy system in the more intermittent-type exercise involved in soccer may contribute to an increase in RPE, which may explain some of the discrepancy in the correlations reported in each study⁴⁶. Impellizzeri et al (2004) suggested that the RPE-based method can not yet replace heart rate-based methods as a valid measure of exercise intensity as only about 50% of the variation they measured in the heart rate-based method could be explained by the session RPE method⁴⁶. In the current study 71% of the variance between training load measured using the Session RPE and Summated Heart Rate Zone method could be explained in subjects that engaged in two weeks of *ad libitum* training.

Since the absolute value or score of training load for each of the methods cannot be compared directly due to differences in units as explained by Foster et al (2001), conclusions in the current study are based on the position of the data points relative to the regression line in Figures 3.2 and 3.3. The data points for the OVER group lie above the regression line and thus indicate that the TRIMP method over-estimated the calculation of training load when compared to the Session RPE method for this group. Conversely, the data points of the UNDER group lying below the regression line indicates that the TRIMP method under-estimated the calculation of training load compared to the Session RPE method for the UNDER group.

There are many limitations to each of the three methods that may partially explain the unaccounted variation between the objective and subjective methods of quantifying training load. The complex interaction of many factors contributing to one's personal perception of physical effort may be something to consider when assessing the variation between Session RPE and heart rate-based methods. These interactions may include hormone and substrate concentrations, personality traits, ventilation rate, neurotransmitter levels, environmental conditions and psychological states¹. Limitations such as these therefore need to be considered when quantifying training using RPE-based methods. A further limitation was expressed by Robinson et al (1991) who observed no correlation between mean effort ratings and mean relative training speed between subjects²⁸. They suggest that current RPE scales may not be useful in comparing or prescribing training intensities for *different* runners²⁸. However RPE scales may still be useful within individuals. Although the limitations for using the Session RPE do need to be acknowledged, the practical value of this measure in quantifying training load is emphasised in situations when the measurement and monitoring of training load is necessary but where heart rate monitors are not available, or when an easier means of reporting and calculating training load is required. In these cases the Session RPE method may still give reasonably accurate assessments of training load. RPE may also be a more valid measure of training intensity than the heart rate-based methods when both oxygen-dependent and oxygen-independent metabolic systems are activated⁴⁶.

Foster et al (2001) suggest that the accuracy of the TRIMP equation may be limited by the inability of heart rate data to quantify high intensity or non-steady state exercise like resistance training, high-intensity interval training or plyometric exercise⁵¹. This is because heart rate usually increases disproportionately during resistance exercise and the cardiac responses required for the calculation of TRIMP are not elicited, thus objective measurements of heart rate can not be used to quantify resistance exercise intensity⁵³. The use of average heart rate in the TRIMP equation may thus not adequately represent very high-intensity exercise⁵¹. Results from the current study may assist in evaluating these suggestions. The correlation between the TRIMP method and the Session RPE method of quantifying training load revealed significant differences among the three groups, which were identified based on their position relative to the SEE of the regression equation. Differences in total TRIMP and training volume were found between groups, along with a non-

significant difference in average heart rate. Since the TRIMP equation includes average heart rate during exercise, maximum heart rate, resting heart rate and exercise duration, significant differences in the first three of these factors (since exercise duration is a common factor in both equations) may suggest a quantifiable reason for the poor relationship between TRIMP and the Session RPE method in groups OVER and UNDER in the current study. In addition, endurance training has an effect on resting^{127;129;132}, submaximal^{102;129;142} and possibly maximal¹⁵⁵ heart rate. Overtraining has also been found to decrease heart rate at the same submaximal intensity²⁰⁷. These changes may influence the usefulness of heart rate-based methods for quantifying training loads, especially if these are going to be used to monitor an athlete's training over time.

The TRIMP equation also includes a weighting factor (Y) that emphasizes high intensity exercise in an attempt to avoid giving disproportionate importance to higher volumes of low intensity exercise compared to low volumes of intense activity. Results from the current study suggest that, compared to the training load calculated using the Session RPE method, the TRIMP equation may in fact be giving disproportionate importance to high intensity exercise for those participants that spent a greater percentage of their total training time exercising in higher intensity heart rate zones (Group OVER), and under-estimating (compared to the Session RPE method) the effect of low intensity exercise on training load for those that spent a greater percentage of their total training time exercising in low intensity heart rate zones (Group UNDER). The Y weighting factor in the TRIMP equation is based on the lactate profiles of trained men and women as exercise intensity increases⁴⁴. However, it has been shown that the exponential relationship between lactate and work load may change with training¹⁹. As such, the use of a standard weighting factor (Y) in the TRIMP equation, which is based on a fixed lactate-work load relationship, may be inappropriate for quantifying training load in subjects that differ in training status. It is possible that a different weighting factor may be required for different training states. Thus, the use of the generic TRIMP equation for all the subjects in the current study (who varied in training status) may have contributed to the low correlations observed between the Session RPE method and the TRIMP method. In addition, the lactate response to exercise can also be affected by many external factors such as ambient temperature, dehydration, mode of exercise, exercise duration, intensity and the rate of change in exercise intensity; prior

exercise, diet and muscle glycogen content^{19;23}. Improvements in training status *and* overtraining have both been associated with decreases in maximal and submaximal blood lactate concentration^{24;25} which may lead to erroneous interpretations of lactate measurements²³. For these reasons the fact that the TRIMP weighting factor is based on a fixed lactate-work load relationship may introduce error in the quantification of training load in circumstances where either an athlete's training status changes over time, or when comparing training loads of subjects that differ with respect to training status.

The correlation between the Summated Heart Rate Zone method and the Session RPE method revealed comparable differences among the three groups to those that occurred between the TRIMP and Session RPE methods. The Summated Heart Rate Zones method focuses on the duration spent in five heart rate zones, and weights each zone such that zone 1 is weighted the least and zone 5 is weighted the most. This weighting system may limit the accuracy of this equation. Since a weighting factor is applied to each zone comprising of a range of heart rates, the lowest heart rate and the highest heart rate in each zone will be weighted the same despite a difference in the physiological load. Under certain circumstances a change in heart rate of only 1 b.min⁻¹ will change the weighting factor of the zone thereby increasing or decreasing the calculated load disproportionately. It may be speculated from these results that, similar to the TRIMP method, the Summated Heart Rate Zone method may disproportionately over-estimate the impact that high intensity exercise may have on training load, and under-estimate the effect of low intensity exercise on training load compared to training load calculated using the Session RPE method. Another potential source of error in this equation is that the time spent below 50% of HRmax is not included in the calculation. This may only affect the accuracy of the calculation marginally (if at all) in high intensity workouts, but it is nonetheless worth noting, especially when quantifying training load for submaximal or interval training bouts.

The limitations of this study were that the subject numbers varied in each of the three groups (OVER, UNDER and ACCURATE). Furthermore the numbers were low in the OVER and UNDER groups and the variances between groups were unequal thus requiring the use of less robust non-parametric statistics. Although differences were found between groups using these statistics, there is a risk of making a Type 1 error

(i.e. finding differences that are not true). However, this risk is low as the magnitude of the differences was in most cases large. Although the large variation in training load of the subjects may be viewed as a weakness, a heterogeneous sample may in fact strengthen the research design and reduce the risk of forming the wrong conclusions, which may happen with a homogeneous or bias sample.

3.5 CONCLUSIONS

In conclusion, the training load calculated using the TRIMP equation correlated best with training load calculated with the Summated Heart Rate Zone method. This is understandable as both methods are based on the same physiological measure. However, the Session RPE method also correlated well with the heart rate-based methods, suggesting that the subjective Session RPE Method of calculating training load remains useful. Further results suggest that in athletes that spend a greater percentage of their training time doing high-intensity exercise the TRIMP and the Summated Heart Rate Zone equations may over-estimate training load compared to the Session RPE method. Conversely, in athletes that spend proportionally more of their training time doing low intensity exercise, these heart rate-based methods may under-estimate training load when compared to the training load calculated using the Session RPE method.

An alternative interpretation is that for those athletes spending more time doing low-intensity exercise, the Session-RPE method may over-estimate training load, whereas for athletes participating in proportionally more high intensity exercise the Session-RPE method may under-estimate training load compared to the objective methods. This will have to be confirmed in future studies. Further investigation is also required to establish the exact cause of the poor correlation between objective and subjective quantification methods found in some athletes. Since the Session RPE method represents a more global indication of the difficulty of an exercise bout than the heart rate-based methods, it may be that the percentage variance not accounted for by the heart rate-based methods represents the numerous extraneous factors (other than heart rate) that contribute towards a person's personal perception of the "difficulty" of the session. Conversely, since none of the heart rate-based equations have been validated, it cannot be ruled out that there may be inherent flaws in these

equations that may affect their relationship with the Session RPE method. It follows that if there are inherent flaws in the equations, further research may identify modifications to the equations required to more accurately calculate training load in each individual.

Accurate prescription of training toward the attainment of a desired or predicted performance requires not only the accurate quantification of training load, but knowledge of the physiological mechanisms involved in the exercise response and the ability to quantify and monitor training induced adaptations. Chapter 4 proposes the use of heart rate recovery as a means with which to monitor the training response. The relationship between heart rate recovery and training load, and whether heart rate recovery responds to acute changes in training load is investigated.

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CHAPTER 4

<p>THE RELATIONSHIP BETWEEN TRAINING LOAD AND HEART RATE RECOVERY</p>
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4.1 INTRODUCTION

An increasing demand for improvements in elite sports performances and the desire for more rapid achievement of personal fitness or sports goals require a more precise, evidence-based method of prescribing personalized training programmes. An optimal programme would prevent under training, overtraining and injury³, and produce favourable physiological adaptations towards desired outcomes at specific times. In order to achieve this, workloads need to be manipulated over time to correctly balance the stress stimulus of exercise with periods of recovery, thereby inducing positive haemodynamic adaptations¹⁴⁶ that contribute to improvements in performance. The ability to measure and monitor these positive and negative training effects would thus make a valuable contribution to the design of effective training programmes.

Endurance training induces numerous physiological adaptations, many of which have been proposed as markers of overtraining^{97;101;102;208}. However no single measure has been identified that quantitatively assesses how an athlete is responding to training. Ideally this marker should be sensitive to change, predictive, easy to quantify and require methods of measurement that are non-invasive, easy to administer and inexpensive. The nervous system, particularly the sympathetic nervous system and adrenal glands, is pivotal to the body's response to acute training stimuli and adaptation¹²⁴ and has a direct effect on heart rate. Carter et al (2003) reviewed studies investigating changes in autonomic control of heart rate with endurance training and concluded that parasympathetic activity to the heart is increased and sympathetic activity is reduced at rest and during submaximal exercise following endurance training³⁴. Changes in sleeping heart rate²⁰⁹, resting heart rate^{127-129;132}, submaximal^{127;129;142;145} and maximal heart rate¹⁵⁵ have been investigated for their potential as markers of training status, however conflicting results regarding the effects of training on these indices exist, making the usefulness of these measures to monitor training status inconclusive. Heart rate variability has been recognised as a means with which to evaluate the autonomic control of heart rate¹²⁵. However, as mentioned in Chapter 1, the interpretation of HRV indices remains inconclusive due to inconsistent methodologies used in their measurement^{27;125;160;162}. In addition, age^{130;164;170}, respiration^{160;162} and temperature¹⁶⁴ may influence measurements of HRV. Consensus about the effects

of training on HRV has also not been reached, as the disparity in training protocols used in various studies makes the comparison of results difficult^{34;128;136;160-163}(Appendix 7). The use of HRV in the assessment of training status may thus be impractical at this stage, until standardized methods are adopted.

Few studies have investigated the effects of training on the autonomic control of heart rate after the cessation of exercise^{143;194;196;197;210}. Heart rate recovery in the first minute or two after moderate to heavy exercise is a consequence of parasympathetic re-activation and sympathetic withdrawal^{30;172;173;176}. Cross-sectional studies have shown that trained athletes have a faster heart rate recovery after exercise at similar absolute intensities than untrained subjects^{143;194}. Bunc et al (1988) studied highly trained rowers¹⁹⁴ and Short and Sedlock (1997) compared untrained subjects (that had participated in two hours of walking or low-impact aerobics per week for four to six months) with trained runners, cyclists and swimmers that had been participating in more than five hours of training per week for four to six months before the study began¹⁴³. Short and Sedlock (1997) found that the heart rate recovery kinetics after exercise that elicited the same relative intensity (70%VO_{2peak}) were similar for trained and untrained participants. However, after the same absolute workload (VO₂ 1.5 L.min⁻¹) heart rate recovery was faster for the trained group¹⁴³. In contrast Hagberg et al (1980) found that the time course of heart rate recovery after submaximal exercise was faster after either an absolute or relative workload in participants that had trained for nine weeks¹⁹⁵.

Otsuki et al (2007) showed that both strength- and endurance-trained athletes have improved heart rate recovery after eight minutes of steady-state exercise at 40% of maximal oxygen uptake compared to untrained controls¹⁹⁸. In this study all athletes had been competitive for at least two years. The endurance-trained athletes had been participating in on average 5.7 sessions per week, each lasting an average of 2.6 hours at an RPE of 15-17. The strength-trained athletes had participated in 5.2 sessions per week, each lasting approximately 3.1 hours at an average RPE of 15. Strength training comprised weight training, throwing, sprint, plyometric and skills training¹⁹⁸. Longitudinal studies have found that heart rate recovery improves significantly after moderate intensity training¹⁹⁷ and that this enhanced post-exercise vagal re-activation is reversed after subsequent detraining^{196;210}. Yamamoto et al (2001) suggested that changes in cardiac autonomic regulation induced by

endurance training partly contributes to the decrease in heart rate during post-exercise recovery and that adaptations to cardiac autonomic control occur sooner in immediate post-exercise periods than at rest¹⁹⁷. These results suggest that there may be potential in using heart rate recovery to distinguish trained from untrained individuals and to establish an athlete's state of training by assessing the deceleration in heart rate after exercise¹⁹⁴.

Thus previous studies have shown that there are long-term training effects on heart rate recovery. What has not been as thoroughly investigated is whether heart rate recovery is sensitive to small acute changes in training workload. This is important within the framework of optimizing training programmes as the sensitivity of the measure would be a prerequisite for it to be used frequently to monitor the physiological response to exercise. Since heart rate recovery is governed by the autonomic nervous system^{30;173;176}, it is likely that the changes observed in heart rate recovery may provide some information about the current condition of the autonomic nervous system. The autonomic nervous system interacts with all other physiological systems, therefore monitoring changes in autonomic nervous system function in response to an exercise stimulus may reflect the body's current capacity to respond to such a stress. The use of heart rate recovery to monitor training status is appealing as it is a non-invasive measure that can be quantified easily and frequently with little inconvenience to the athlete.

Being relatively "uncharted" territory, a number of questions arise about what heart rate recovery represents and how it should be interpreted: Are the changes that have been observed in heart rate recovery with endurance training an indication of an athlete's chronic, cumulative state of fitness and/or fatigue (adaptation)? If so, could changes in heart rate recovery detect or predict overtraining or is it merely another symptom of the syndrome? Further, does heart rate recovery also change acutely in response to changes in the training stimulus (load)? If so could changes in heart rate recovery be a marker of overreaching, a more acute physiological response to increased training load, or even indicate if an athlete is under-training? To begin to answer these complex questions one first needs to establish what the relationship is between training load and heart rate recovery. Further, does heart rate recovery remain stable if training load is maintained at a level to which the body is accustomed, and does it change acutely with decreases or increases in training load.

The aim of this study was therefore to assess the relationship between training load and heart rate recovery and to investigate whether acute changes in training load were reflected in heart rate recovery after a standardized submaximal exercise test.

4.2 METHODS

4.2.1 Study Design and Subjects

Thirty-four physically active men and women were recruited for this two-week trial. The prospective observational cohort study was approved by the University of Cape Town Ethics and Research Committee and the participants gave their written informed consent after the testing protocol was explained to them. A Physical Activity Readiness Questionnaire (PAR-Q) (Appendix 8) was administered to all participants prior to their enrolment in the study, to ensure that they were not at any health risk while performing exercise during the study. Before the start of the trial body composition, maximal oxygen consumption and maximal heart rate of the participants were measured. Participants were asked to maintain the same type and quantity of *ad libitum* training during the trial as they had been doing for the three weeks prior to the trial. The subjects' *ad libitum* training during the 2-week trial was quantified using the TRIMP equation. Participants were excluded from the trial if they were injured or ill or had other reasons that prevented them from training to their normal capacity during the study (n = 6). Thus the data from 28 participants (12 men, 16 women; mean age 30 ± 5 years) were analyzed. Since there are no gender differences in heart rate recovery it was appropriate to use both men and women in this study^{178;193}.

The 28 participants selected for this study exercised regularly but varied in the average amount of training they did each week. For example, the subject that performed the least amount of training in the study did two to three sessions per week, with each session ranging between 20 and 30 minutes at an RPE of 5 to 7, using the Borg CR 0 – 10 Scale (Appendix 3). Whereas a highly trained athlete who performed the most training in the study participated in seven to eight sessions per week that ranged in duration (30 - 190 minutes) and intensity (RPE 2 to 8). Exercise modes included running, cycling and/or interval-type cardiovascular gym

training which combined stepping, elliptical machines, super-circuit and rowing machines. The heterogeneity of the participants' training strengthened the study design as it reduced the risk of sample bias and facilitated the evaluation of the effects of relative, rather than absolute changes in training volume.

The sample size was determined using the data of Lamberts et al (2004) which showed that the day-to-day variability of submaximal heart rate and recovery heart rate after exercise was about 5-6 b.min⁻¹ and 7-8 b.min⁻¹ respectively (defined as the 95% confidence intervals of within-subject range), with a standard deviation of 3 b.min⁻¹²¹¹. In the study of Lamberts et al (2004) the subjects maintained their training load over the period of the study, whereas in the current study changes in training load were observed over the 2-week trial. Therefore, in the current study the smallest meaningful difference was taken to be 9 b.min⁻¹ and a standard deviation of 6 b.min⁻¹ was estimated to accommodate possible effects of variations in training load on heart rate recovery. The sample size required for this study, in order to achieve a statistical power of 80% and a significance level of 5% was therefore $n = 7$ for each group²¹².

4.2.2 Methodology

4.2.2.1 Body composition and physiological measurements

Body mass was recorded on a calibrated scale (Seca model 708 Germany) and recorded to the nearest 100 g. The stature of each athlete was recorded to the nearest millimetre using a stadiometer (Seca model 708 Germany). Body fat percent was assessed with a near infrared (NIR) measurement on the right bicep using the Futrex-6100A/ZL (Kett Electric Laboratory, Futrex Inc. Gaithersburg, MD USA). Resting heart rate, recorded immediately after waking in the morning with a Vantage XL heart rate monitor (Polar Electro, Kempele, Finland), was taken at least three times during the trial. An incremental exercise test to volitional exhaustion was conducted on a motor driven treadmill to determine the participant's maximal oxygen uptake (VO₂max), peak treadmill running speed (PTRS) and maximum heart rate (HRmax), as described in Chapter 2.

4.2.2.2 Protocol

An outline of the two-week protocol and the timing of the measurements taken during the 2-week period are shown in Figure 4.1.

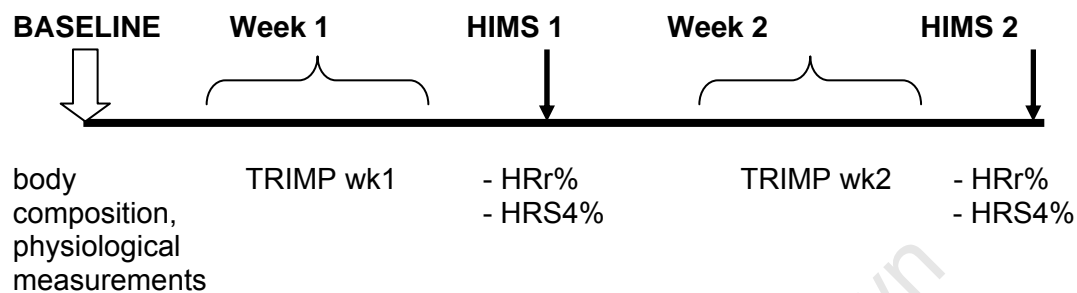


Figure 4.1: Schematic of 2-week study protocol and measurements taken during this period.

- Training load calculated using TRIMP
- HIMS = submaximal shuttle running test
- HRr% = 1-minute recovery heart rate expressed as a percent of the heart rate reached at the end of the fourth running stage of the HIMS test
- HRS4% = Heart rate at the end of the fourth running stage of the HIMS test expressed as a percent of maximal heart rate

4.2.2.3 Quantification of training load

During the two weeks of the trial each participant wore a heart rate monitor for each exercise training session to record heart rate ($\text{b}\cdot\text{min}^{-1}$) and exercise duration (min). Average heart rate during each training session (HR_{ex}), resting heart rate (HR_{rest}) and maximal heart rate (HR_{max}) were used in the Training Impulse (TRIMP) equation, as described in Equation 2⁴⁴ in Chapter 3, to quantify training load for each exercise session performed during the trial. Session training loads were added for each subject to calculate total training load for each week. TRIMP was chosen to quantify training load as it is an objective method of quantifying training that is being used frequently in research. In Chapter 3 a strong correlation was found between TRIMP and the Summated Heart Rate Zone methods suggesting that both are appropriate for the quantification of training load during continuous as well as interval training sessions. The participants in the current study engaged in very similar exercise training to the participants in Chapter 3. Therefore either of these equations

could have been used, and TRIMP was chosen as it appears to be a more robust and comprehensive assessment of physiological disturbance during exercise.

Participants were then divided into three groups based on whether they increased (Group I, n = 9), decreased (Group D, n = 8) or kept their training load the same (Group S, n = 11) in week 2 compared to week 1 of the trial. This was done by calculating the percent difference in weekly TRIMP (Equation 5). A variation of 20% was selected as a reasonable range with which to account for normal intra-individual variation. As such, those subjects that kept their training load within a 20% range were regarded as having kept their training constant over the two weeks.

$$\% \text{ difference in TRIMP} = \frac{(\text{TRIMP wk2} - \text{TRIMP wk1})}{\text{TRIMP wk2}} \times 100 \dots \dots \dots (5)$$

4.2.2.4 Heart Rate Interval Monitoring System Test

The submaximal heart rate shuttle test (Heart rate Interval Monitoring System: HIMS) was developed in this laboratory and consists of four running stages of increasing intensity, interspersed with recovery periods²¹¹ as depicted in Figure 4.2. The subjects run on a rubberised indoor floor between two lines drawn 20 meters apart. The pace of each of the running stages (8.4 km.h⁻¹, 9.6 km.h⁻¹, 10.8 km.h⁻¹ and 12.0 km.h⁻¹ respectively) is set by a pre-recorded auditory signal on compact disk. Each running stage lasts two minutes and is separated by one-minute rest periods in which the subjects stand upright and motionless with their hands by their sides. The subjects rest for two minutes after the fourth stage. Therefore, the total duration of the test is 13 minutes and the intensity of the test is controlled and constant for each test. The HIMS test was designed to be submaximal and non-invasive for athletes so that it can be administered frequently during different phases of training. Furthermore, the test is quick, easy to administer to a number of athletes simultaneously and may form part of a warm-up before a training session. In a previous study the intra-class correlation coefficient of the heart rate on a day-to-day basis during the four stages and recovery periods ranged between r = 0.94 and 0.99 in a group of subjects who maintained a constant training load²¹¹. In the current study the HIMS tests were repeated on the same day each week at approximately

the same time, in order to control for diurnal variation. Subjects were asked not to consume any caffeine, nor to exercise for at least three hours before performing the HIMS tests.

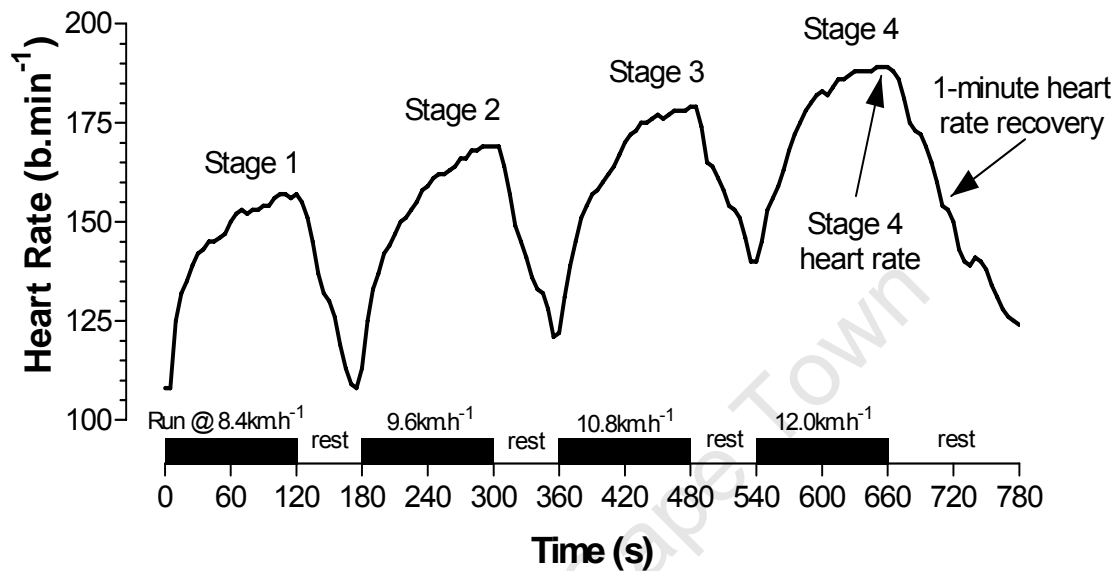


Figure 4.2: Heart rate Interval Monitoring System (HIMS) Test protocol, showing running speeds and durations, interspersed with rest periods. A typical heart rate response to this submaximal test is also shown (b.min⁻¹), with Stage 4 heart rate and 1-minute heart rate recovery values identified.

4.2.2.5 Quantification of percent heart rate recovery

Heart rate recovery was assessed during the first minute after the standardized submaximal shuttle test at the end of each week of the trial. Heart rate was recorded at 5-second intervals during the HIMS test and heart rate recovery was assessed by expressing the heart rate one minute after the fourth running stage as a percentage of the heart rate attained at the end of the fourth running stage of the test (HRR%, Equation 6).

$$\text{HRR}\% = \frac{\text{HR at 1 minute of recovery}}{\text{HR at end of Stage 4 of HIMS}} \times 100 \dots \dots \dots (6)$$

In addition, the heart rate reached at the end of the fourth running stage of the HIMS test was expressed as a percent of maximal heart rate (HRS4%, Equation 7) determined in the treadmill test to exhaustion.

$$\text{HRS4\%} = (\text{HR at end of Stage 4 of HIMS} / \text{HRmax}) \times 100 \dots \dots \dots (7)$$

4.2.3 Statistical Analyses

A Pearson's Product Moment correlation assessed the relationship between the first and second week's training load (TRIMP), HRr% and HRS4%. The 95% confidence intervals around the correlation coefficient were calculated using a spreadsheet for this purpose downloaded from www.newstats.org (accessed Dec 2006)²⁰³. An analysis of variance with repeated measures was used to determine whether there were differences between heart rate at the end of the submaximal test and recovery at week 1 compared to week 2 within groups. A Kruskal-Wallis test was used to compare changes in TRIMP, HRr% and HRS4% from week 1 to week 2 between the three groups (Group I, D and S). Similar to the data analysis in Chapter 3, the use of non-parametric statistics was required for these comparisons, as the data had unequal variance and were not always normally distributed (Kolmogorov-Smirnov one-sample test for normality). Statistical analysis was performed using STATISTICA 7.0 data analysis software system (StatSoft, Inc. Tulsa, OK, USA). All data are expressed as the mean \pm standard deviation and statistical significance was accepted at $p < 0.05$.

4.3 RESULTS

4.3.1 Subject characteristics

The 28 participants (mean age 30 ± 5 years) selected for this study trained regularly but varied in the amount of training they habitually did each week (range 82 – 747 min.wk⁻¹). Baseline measurements and selected physiological measurements calculated during the trial are shown in Table 4.1.

Table 4.1: Subject characteristics (n = 28). Data are expressed as mean \pm SD.

Subject Characteristics	Group S (5 men, 6 women)	Group D (4 men, 4 women)	Group I (3 men, 6 women)
Age (years)	29 \pm 5	30 \pm 6	30 \pm 5
Mass (kg)	68 \pm 17	66 \pm 11	64 \pm 6
Stature (cm)	167 \pm 7	172 \pm 10	169 \pm 8
Body fat (%)	22 \pm 7	18 \pm 7	20 \pm 8
VO ₂ max (ml.min ⁻¹ .kg ⁻¹)	55 \pm 7	58 \pm 9	57 \pm 8
Maximum HR (b.min ⁻¹)	189 \pm 7	192 \pm 8	191 \pm 11
PTRS (km.h ⁻¹)	16 \pm 2	17 \pm 2	17 \pm 2
Resting HR (b.min ⁻¹)	54 \pm 7	57 \pm 7	57 \pm 7
TRIMP week 1 (AU)	671 \pm 380	584 \pm 283	429 \pm 215
TRIMP week 2 (AU)	620 \pm 346	359 \pm 213	664 \pm 353

AU = arbitrary units

4.3.2 Relationship between TRIMP week 1 and TRIMP week 2

Figure 4.3A depicts the relationship between training load (TRIMP) in week 1 and TRIMP in week 2 for all subjects. Figure 4.3B illustrates how each of the Groups differed with respect to the percent change in weekly TRIMP from week 1 to week 2. Group D significantly decreased their TRIMP compared to Group S ($p = 0.04$) and Group I significantly increased their TRIMP compared to Group S ($p = 0.02$), who maintained their training load within a 20% range (Figure 4.3B). The mean percentage change in TRIMP was also different between Groups D and I ($p < 0.05$) (Figure 4.3B).

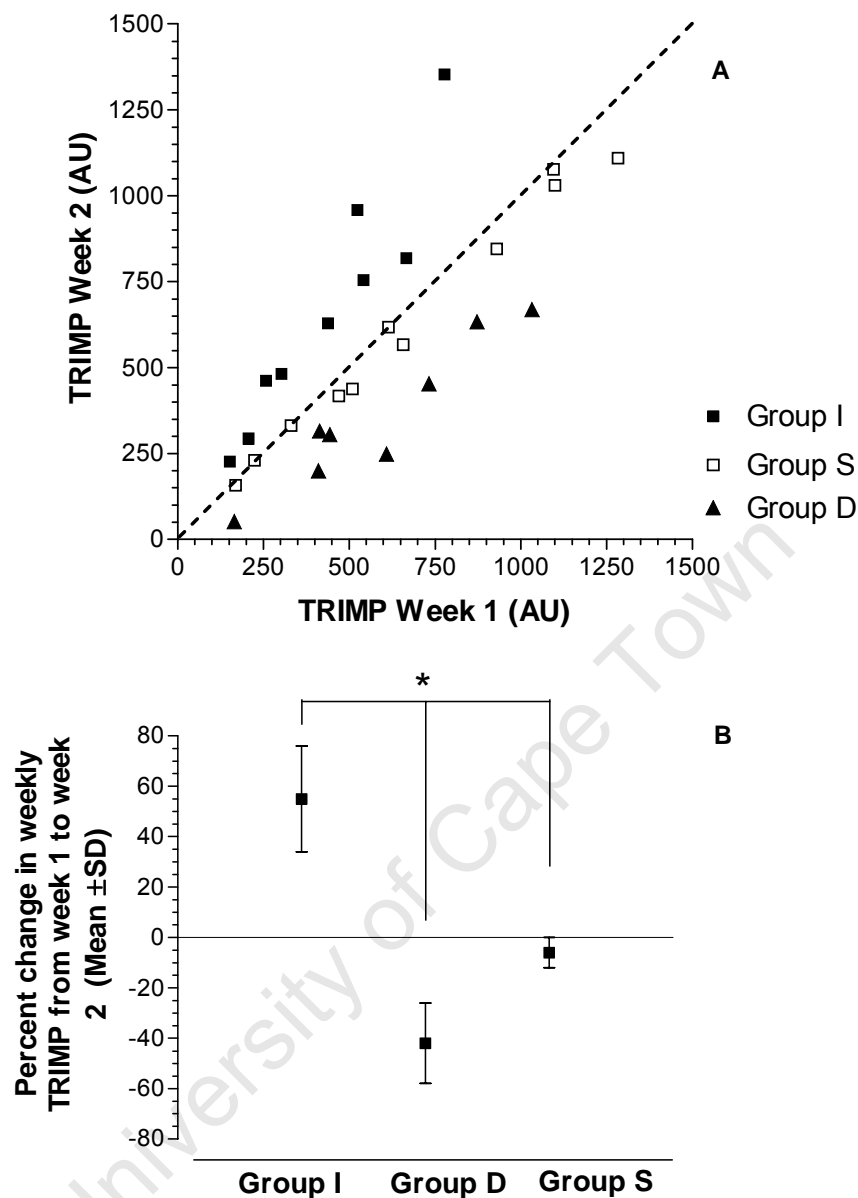


Figure 4.3: (A) Relationship between TRIMP calculated for week 1 and week 2 and (B) percent change in weekly TRIMP (mean \pm SD) from week 1 to week 2 for Group I (n = 9), Group S (n = 11) and Group D (n = 8). * Group S significantly different from Group I and Group D, and Group I significantly different from Group D ($p < 0.05$)

4.3.3 Relationship between Training load and Heart rate recovery

Data for Group S (n = 11) were used in the assessment of the relationship between training load (TRIMP) and percent heart rate recovery after the HIMS test. These participants kept their training load within a 20% range from week 1 to week 2, which was regarded as having kept their training constant over the two weeks. In this way the possible confounding effect that fluctuations in training load may have on the

relationship was controlled for. The correlation between TRIMP and heart rate recovery was significant ($p < 0.05$) for week 2 ($r = -0.68$, Figure 4.4B) and for the average of the 2 weeks ($r = -0.61$, Figure 4.4C). However the correlation was not significant for week 1 (Figure 4.4A).

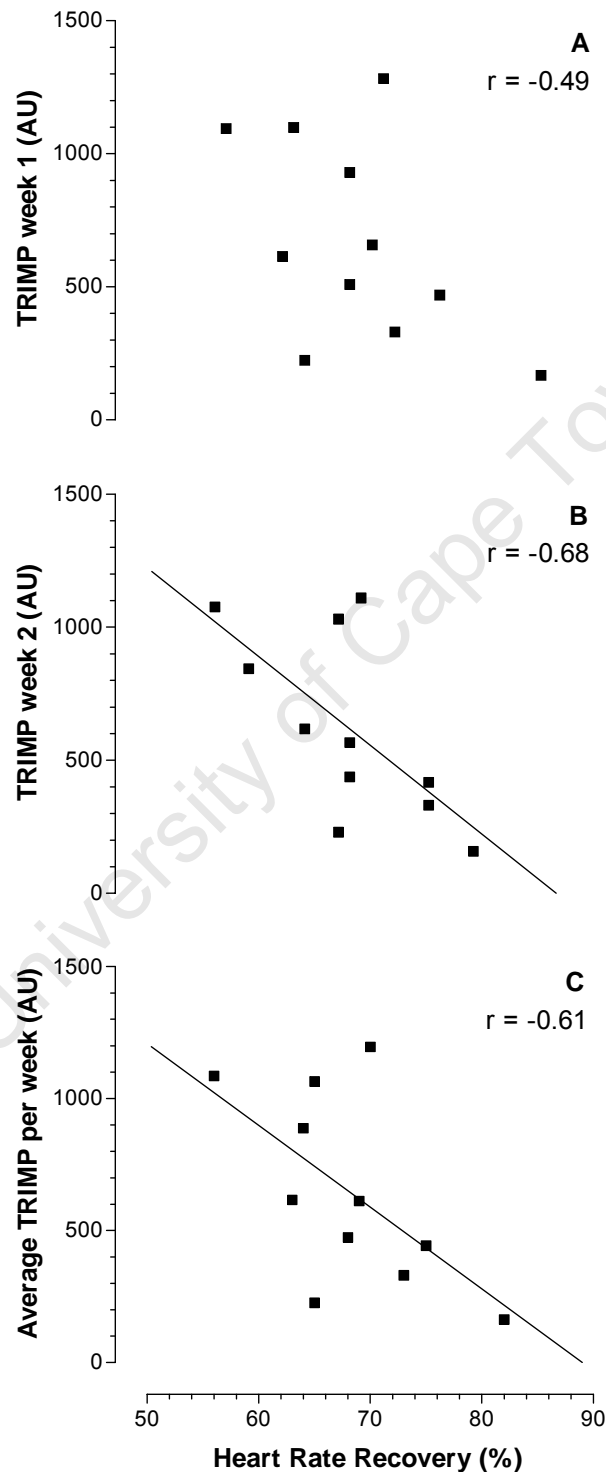


Figure 4.4: Relationship between TRIMP (AU) and Heart rate recovery (%) for (A) week 1, (B) week 2, and (C) the average for the 2 weeks, in Group S ($n = 11$).

4.3.4 Relationship between VO₂max and Heart rate recovery

Data for Group S (n = 11) were also used in the assessment of the relationship between VO₂max and heart rate recovery. A correlation of $r = -0.19$ was found between VO₂max and the average heart rate recovery after the two HIMS tests in these participants, that kept their training load constant over the two weeks.

4.3.5 Changes in heart rate recovery from HIMS 1 to HIMS 2

Table 4.2 includes the absolute heart rate at the end of the submaximal test and after 1 minute of recovery (week 1 and week 2), and the mean percent heart rate recovery (HRr%) after HIMS 1 and HIMS 2 for all groups. There were no differences between groups for heart rate measured at either the end of the submaximal test (week 1 vs. week 2) or after recovery (week 1 vs. week 2). The recovery heart rate in Group I was significantly higher in week 2 vs. week 1 (121 ± 23 vs. 112 ± 34 b.min⁻¹; $p = 0.017$). Although HRr% was not significantly different from week 1 to week 2 within each group there was a tendency for the mean percent heart rate recovery in Group I (the group that increased their training load) to be slower after HIMS 2 than after HIMS 1 (Table 4.2). Conversely, there was a tendency for heart rate recovery in Group D (who decreased their training load during week 2) to be slightly faster after HIMS 2 than after HIMS 1 (Table 4.2). The mean change in HRr% in Group I (which showed an increase) was significantly different from the decrease in HRr% in Group D ($p = 0.03$, Figure 4.5A). Mean heart rate recovery in Group S remained the same after each HIMS test (Figure 4.5A).

Table 4.2: Changes in absolute heart rate, heart rate recovery (HRr) and percent heart rate recovery (HRr%) during the standardized submaximal running (HIMS) test at week 1 and 2 for Group I, Group S and Group D. Data are expressed as mean \pm SD

Subject Characteristics N	Group S 11	Group D 8	Group I 9
<u>HIMS 1</u>			
Stage 4 heart rate (b.min ⁻¹)	176 \pm 12	175 \pm 12	173 \pm 17
HRr (b.min ⁻¹)	120 \pm 19	122 \pm 19	112 \pm 34
Absolute decrease in HR (b.min ⁻¹)	55 \pm 13	53 \pm 11	61 \pm 20
HRr% after HIMS 1	68 \pm 8	69 \pm 7	64 \pm 15
<u>HIMS 2</u>			
Stage 4 HR (b.min ⁻¹)	174 \pm 11	174 \pm 13	174 \pm 17
HRr (b.min ⁻¹)	119 \pm 16	117 \pm 24	121 \pm 23 #
Absolute decrease in HR (b.min ⁻¹)	56 \pm 12	57 \pm 13	53 \pm 13
HRr% after HIMS 2	68 \pm 7	67 \pm 9	69 \pm 9

- HRr = heart rate after 1 minute of recovery
 - HRr% = 1-minute recovery heart rate expressed as a percent of the heart rate reached at the end of the fourth running stage of the HIMS test.
- # Group I, HIMS 1 vs. HIMS 2 (p = 0.017)

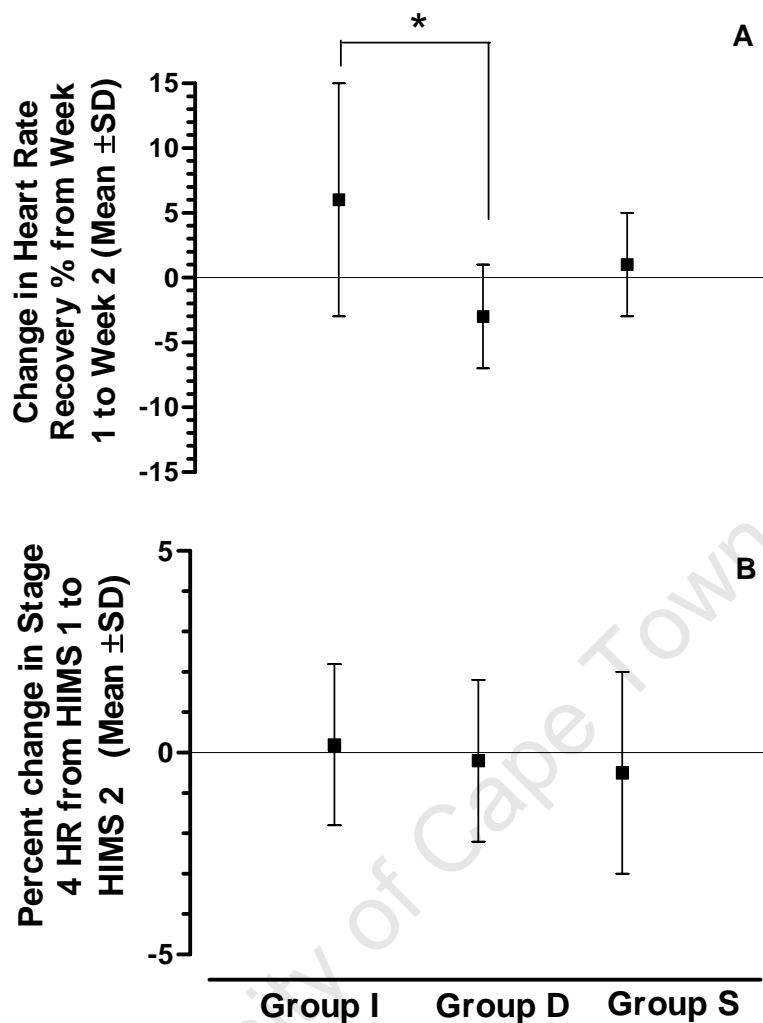


Figure 4.5: Percent change in (A) heart rate recovery (HRr%) and (B) stage 4 heart rate (HRS4%) (mean \pm SD) from HIMS 1 (end of week 1) to HIMS 2 (end of week 2) in Group S, Group I and Group D.

- HRr% = 1-minute recovery heart rate expressed as a percent of the heart rate reached at the end of the fourth running stage of the HIMS test.
- HRS4% = Heart rate at the end of the fourth running stage of the HIMS test expressed as a percent of maximal heart rate.

* Group D significantly different from Group I ($p < 0.05$)

4.3.6 Repeatability of HIMS Stage 4 heart rate

There were no differences between groups when comparing changes from HIMS 1 to HIMS 2 in the heart rate attained at the end of Stage 4 (Figure 4.5B). All groups showed strong correlations between heart rate attained at the end of Stage 4 of HIMS 1 and HIMS 2: Group S $r = 0.88$ (95% CI = 0.62 to 0.97), Group I $r = 0.95$ (95% CI = 0.74 to 0.99) and Group D $r = 0.96$ (95% CI = 0.79 to 0.99).

4.4 DISCUSSION

4.4.1 Relationship between Training load and Heart rate recovery

The first finding in this study was the existence of a significant relationship between training load calculated using TRIMP during week 2 and the subsequent heart rate recovery at the end of week 2 ($r = -0.68$, $p < 0.05$, Figure 4.4B) in participants that kept their training load the same from week 1 to week 2. When weekly TRIMP for the two weeks of the trial were averaged and HRr% after the two HIMS tests were averaged, a significant relationship was also evident ($r = -0.61$, $p < 0.05$, Figure 4.4C). The non-significant relationship between these two outcomes in week 1 may be the consequence of differences in training load in the week preceding the first week of the study (Figure 4.4A). The participants were asked to maintain the same type and quantity of training during the trial as they had been doing for the three weeks prior to the trial, however whether or not they did can not be proven quantitatively. Since estimates of training prior to the trial were self-reported it is acknowledged that a margin of error may exist, as suggested in Chapter 2. However, during the current 2-week trial heart rate was used to collect objective physiological data as was proposed in Chapter 2. This therefore negated the concern raised in Chapter 2 of potential inaccuracy in data interpretation.

The relationship between TRIMP and heart rate recovery has been investigated previously by Buchheit and Gindre (2006)⁸. They found a significant correlation ($r = -0.55$) between training load (calculated using the Baecke sport score) and post-exercise heart rate recovery time constant. They also investigated the relationship between $VO_2\text{max}$ and heart rate recovery, but found no correlation ($r = 0.01$)⁸. This has also been supported in the current study, in which a correlation of $r = -0.19$ was found between $VO_2\text{max}$ and the average of heart rate recovery after the two HIMS tests in participants that kept their training load constant over the two weeks. It is often assumed that $VO_2\text{max}$ is an indicator of “aerobic fitness” because it improves with training¹¹. Heart rate recovery has also been found to improve with training^{143;196;197}, thus one would instinctively assume this is also a measure of “fitness”. Surprisingly however, there appears to be no relationship between $VO_2\text{max}$ and heart rate recovery, which calls into question whether heart rate recovery is in fact a measure cardiorespiratory “fitness”, or whether it is measuring something

different than what is interpreted with $VO_2\text{max}$ values. Since heart rate recovery relates fairly well with training load, the measure of heart rate recovery may provide insight into the effect of habitual training loads on the autonomic nervous system function rather than being a measure of chronic cardiorespiratory adaptation. The fact that the body adapts to and can withstand higher training loads with progressive training may explain why this measure also changes over time with training. Equating heart rate recovery to “aerobic fitness”, in the same way as $VO_2\text{max}$ is, may therefore be erroneous at this time. Further research is required to completely understand what heart rate recovery represents.

4.4.2 Changes in heart rate recovery from HIMS 1 to HIMS 2

Heart rate recovery was slightly slower after the second week of the trial in subjects who increased their training load. There was no change in heart rate recovery for those subjects who maintained their training load or the subjects who decreased their training load through the trial. A decrease in heart rate recovery, as observed in Group I in this study, may represent a negative training response to an increase in training load or exercise stimulus. The basis for training is the manipulation of workloads over time to induce positive haemodynamic adaptations¹⁴⁶ that contribute to improvements in performance. The aim of this manipulation is to produce the correct balance between stress (exercise) stimuli and rest (recovery) periods, since an imbalance in these factors could lead to an overtrained state⁸³. Heart rate recovery may contribute to a better understanding of these positive and negative training effects.

Other studies have investigated the effect of training, detraining and taper on heart rate recovery specifically. Sugawara et al (2001) reported that eight weeks of training in previously untrained men improved 30-second heart rate recovery¹⁹⁶. Two weeks of subsequent detraining maintained the improved heart rate recovery, however by the fourth week of detraining heart rate recovery had returned to baseline levels¹⁹⁶. The heart rate recovery of ten track and field high school girls, who had been training daily for three to four months, was recorded before and after three weeks of detraining²¹⁰. In this case heart rate recovery was assessed as a “score” consisting of the sum of the heart rates at 1-, 2- and 3-minutes of recovery. Using

this method Michael et al (1972) reported a slower heart rate recovery after three weeks of detraining²¹⁰. Results from these studies suggest that extended periods of detraining reverse training-induced improvements in heart rate recovery. Since the current study did not investigate the long-term effects of training, nor the effects of complete cessation of exercise (detraining) on heart rate recovery, results from the current study cannot be compared to those reported in the above studies.

In a study by Brynteson and Sinning (1973) 21 men were trained for five weeks (five days per week, 30-minute exercise sessions per day) which resulted in an improvement in 5-minute heart rate recovery²¹³. Subsequently training was decreased to either one day.wk⁻¹, two days.wk⁻¹, three days.wk⁻¹ or four days.wk⁻¹ for a further five weeks. This study found that the larger decrease in training from five days.wk⁻¹ to one or two days per week reversed the training-induced improvement in heart rate recovery, whereas the smaller reduction in training to three or four days per week (possibly comparable to tapering) improved heart rate recovery slightly more²¹³. This slight improvement in heart rate recovery after a relatively small decrease in training load is similar to results in the current study in which there was a tendency for improved heart rate recovery in Group D, who had decreased their training load by 42%.

In contrast, Houmard et al (1989) found 1- and 2-minute heart rate recovery was slower after submaximal treadmill running in five highly trained runners after a 10-day taper that followed three months of intensive training²¹⁴. Mujika et al (2004) have suggested that the content of pre-taper training may have an effect on heart rate indices during the taper period²¹⁵. For example two weeks of high intensity training before a 2-week taper produced increases in submaximal and maximal heart rate and decreases in resting heart rate post-taper compared to pre-taper¹³⁷. However, post-taper values were comparable to baseline measurements before the start of the high intensity training period¹³⁷. These changes in heart rate indices during taper contradict other studies involving tapering but are credited by Mujika et al (2004) to a reversal of the physiological effects of the high intensity training immediately prior to the taper²¹⁵. This may be the case as well in the study by Houmard et al (1989) where subjects had participated in three months of high intensity training before the taper²¹⁴. In the current study, and in the study of Brynteson and Sinning (1973)²¹³ the subjects were more likely in an adapted state before the decrease in training and

subsequent tendency for improvement in heart rate recovery. However, this suggestion remains inconclusive due to limitations such as insufficient data being available or the use of many different methods of training, tapering and measuring heart rate recovery in the various studies. Participants that maintained their training load (Group S) were probably already adapted to the amount of training they performed each week, which may have been represented by the stability of their heart rate recovery during the trial. No studies were found that specifically examined the effects of small acute increases in training load (as occurred in Group I) on heart rate recovery, thus results from this study can not be compared to previous research.

4.4.3 Repeatability of HIMS Stage 4 heart rate

The HIMS test designed in this laboratory is a submaximal shuttle test that is easily administered and provides a controlled work load which precedes the measurement of heart rate recovery²¹¹. In this study there were no differences between or within groups when comparing changes in the heart rate attained at the end of Stage 4 from HIMS 1 to HIMS 2. The intra-individual heart rate responses to the test have been shown to be repeatable over five days in subjects that kept their training constant, with a variation of about six beats occurring in the submaximal heart rate attained at the end of the fourth stage of the test²¹¹. The repeatability of the heart rate measurement at this submaximal stage has been supported by results in the current study, even with acute changes in training load. A comprehensive review by Mujika et al (2004), of the physiological changes occurring during pre-event taper, includes studies that vary in taper duration from 6 to 35 days²¹⁵. The authors concluded that resting, maximal and submaximal heart rates do not change during taper, whereas increases in blood and red cell volume, haemoglobin and haematocrit indicate a positive red cell balance during this phase²¹⁵. Similarly, the stability of the heart rate reached at the end of the HIMS test in the current study suggests that submaximal heart rate represents something different from that being represented by heart rate recovery, which did change with increases and decreases in training load. Whereas heart rate during exercise may be a measure of cardiac load, heart rate recovery may represent the adaptive state or the capacity of the autonomic nervous system to respond to that cardiac load. If the HIMS test is administered frequently, a profile of changes in heart rate recovery after the test can be established for each individual,

thereby monitoring the athlete's physiological capacity to respond positively to subsequent training. While the heart rate recovery test on its own may not be diagnostic, if the data are interpreted in conjunction with other data it may provide useful information with which to design and prescribe optimal and personalized training programmes.

In conclusion the current study shows that a significant relationship exists between heart rate recovery and training load but not between heart rate recovery and $\text{VO}_{2\text{max}}$. Heart rate recovery also responds to acute changes in training load. Heart rate recovery slowed slightly after increases in training load whereas heart rate recovery tended to improve in subjects that decreased their training load. There was no change in heart rate recovery for those subjects who maintained their training load through the 2-week trial. Since heart rate recovery is governed by the autonomic nervous system^{30;173;176}, it is likely that the changes observed in heart rate recovery may represent negative and positive responses of the autonomic nervous system to training/exercise stress. Further, since the autonomic nervous system interacts with all other physiological systems, the condition of this system may give a global indication of the physiological state of the whole body. So, while changes in submaximal heart rate may be a measure of physiological stress during exercise, heart rate recovery recorded under standardised conditions may be interpreted as a practical, reliable and quantifiable measure of the body's current capacity to respond to stress.

The next level of research is to formulate longitudinal studies that confirm results from the previous cross-sectional studies. Accordingly the research described in Chapter 5 analyses the training response to a periodised endurance running training programme. Following the results of Chapter 4, a few more complex questions developed, including whether or not the relationship between training load and heart rate recovery remains when training load increases and decreases within a prolonged training programme? Whether heart rate recovery responds to acute changes in training load in the same way irrespective of what phase of training a participant is in? And lastly, whether everyone responds acutely and chronically in a similar way to endurance training? These questions are addressed in the next chapter.

CHAPTER 5

THE EFFECT OF ENDURANCE TRAINING ON HEART RATE RECOVERY

University of Cape Town

5.1 INTRODUCTION

The study described in Chapter 4, which involved participants that all exercised regularly but varied in the average amount of training they did each week, revealed three main findings. A significant relationship was found between training load (TRIMP) and heart rate recovery (HRR%) in a group of people that kept their training load constant over a 2-week period. Changes in heart rate recovery appeared to be associated with acute changes in weekly training load. Conversely, submaximal heart rate did not change with acute changes in training load, suggesting that heart rate recovery and submaximal heart rate represent different physiological entities. Previously published cross-sectional studies have shown that trained athletes have a faster heart rate recovery than untrained subjects after exercise at similar absolute intensities^{143;194}. Otsuki et al (2007) showed that both strength-trained and endurance-trained athletes have improved heart rate recovery compared to untrained controls after 8 minutes of steady-state exercise at a relative intensity¹⁹⁸. However, a potential limitation of cross-sectional studies is the inability to conclude with certainty that the results represent a true phenomenon, without considering or acknowledging that they may be influenced by an inherent variation in the participants studied. Therefore to address this potential problem a longitudinal study was required to confirm the results obtained in Chapter 4. Few longitudinal studies have investigated the overall effects of endurance training on heart rate recovery. Those studies that have monitored the effects of endurance training found that heart rate recovery improves significantly after moderate intensity training¹⁹⁷ due to an enhanced post-exercise vagal re-activation that is reversed after subsequent detraining^{196;210}. The changes that occur in cardiac autonomic regulation as a result of endurance training, and their effect on heart rate during immediate post-exercise recovery suggest that there may be potential in using heart rate recovery to assess an athlete's state of training¹⁹⁴, as was proposed in Chapter 4.

Participants of the study in Chapter 4 were asked to maintain the same type and quantity of training during the 2-week trial as they had been doing for the three weeks prior to the trial. The assumption was that these subjects were already adapted to the amount of training they performed each week and thus were in Stage 2 of the General Adaptation Syndrome⁷⁵, the Stage of Resistance, explained in section 1.4 of Chapter 1. Using the concept of the General Adaptation Syndrome, there are no

longitudinal data for subjects going from Stage 1 (Alarm Reaction) to Stage 2 (Stage of Resistance) and also possibly Stage 3 (Stage of Exhaustion). The progression in research to the formulation of a longitudinal study that attempts to confirm results from the previous cross-sectional study introduces a few more complex questions: Does the relationship between training load and heart rate recovery still remain in the situation where training load varies and increases and decreases with the progressive requirements of a training programme? Another question is does heart rate recovery respond to acute changes in training load in the same way whether the athlete/participant is in an *adapting* state (the Alarm Reaction Stage) or in an *adapted* state (Stage of Resistance) of training? Answers to these questions have important practical applications particularly with regard to using heart rate recovery as a marker.

Therefore the aims of the current study were to assess the relationship between training load (TRIMP) and heart rate recovery, and whether heart rate recovery responds to acute changes in training load in a predictable way in sedentary people that embarked on a 12-week running training programme.

5.2 10 KM RUNNING PROGRAMME

5.2.1 METHODS

5.2.1.1 Study design and participants

Ten females ranging in age from 21 to 47 years participated in a running programme offered by the Sports Science Institute of South Africa. The programme provided untrained individuals with scientifically-based training guidance towards running a 10 km race at the end of 12 weeks. These participants volunteered to be monitored for the duration of the training programme as part of a prospective observational cohort study that was approved by the University of Cape Town Ethics and Research Committee. Prospective participants completed a Modified Physical Activity Readiness Questionnaire (PAR-Q, Appendix 8). Volunteers that marked “Yes” next to 2 or more of the questions were excluded from participating in the study due to their potential health risk. All the individuals that volunteered to participate in this study (n = 10) passed the PAR-Q according to these guidelines. The eligible participants gave their informed consent after the study protocol was explained to them. Although every effort was made to collect data for the entire 12-week period, this was sometimes difficult, particularly at the beginning of the programme, therefore 10-11 weeks of data were collected and analysed. All participants successfully completed the running programme and a 10 km road race at the end of the programme.

5.2.1.2 Methodology

5.2.1.2.1 Body composition and physiological measurements

Before the start and at the end of the 12-week programme basic body composition measurements were recorded. Body mass was recorded on a calibrated scale (Seca, model 708, Germany) to the nearest 100 g. The stature of each athlete was recorded to the nearest millimetre using a stadiometer (Seca, model 708, Germany). Body fat was assessed with a near infrared (NIR) measurement on the right bicep using the Futrex-6100A/ZL (Kett Electric Laboratory, Futrex Inc. Gaithersburg, MD

USA). Resting heart rate was recorded immediately after waking in the morning with a Suunto T6 heart rate monitor (Suunto, OY, Finland) on at least 3 occasions during the programme. Maximum heart rate (HRmax) was estimated using the age-predicted equation ($220 - \text{age}$). The decision to estimate, rather than measure, maximum heart rate was taken as the participants were not accustomed to strenuous exercise and they were also unfamiliar with exercise testing protocols. Thus, requiring them to complete a maximal test at the start of the programme, when they were untrained and unaccustomed to high intensity exercise, may have deterred them from volunteering for the study. If a heart rate higher than the predicted HRmax was attained during training or during any of the HIMS tests, the higher value was taken to be HRmax in these cases.

5.2.1.2.2 10 km Running Programme

The 12-week running programme, which was based on scientific training principles, started with bouts of short duration exercise that alternated intervals of running and walking. Each of the three sessions per week began with a 5 to 15 minute walking warm-up and ended with approximately 10 minutes of stretching performed as a group and directed by the running programme instructor. As the programme progressed the duration of the running bouts increased while the walking bouts decreased. Although no standardised percent increase was implemented, the duration of the running intervals increased in small manageable increments, and the number of repetitions of running/walking intervals increased. As the programme progressed one session per week became the “longer” session, containing the highest amount of running. The other 2 sessions remained shorter. Several hilly routes were included, although not more than one per week, in order to improve the participants’ strength and fitness. The periodised nature of the programme aimed to provide a stimulus that produced appropriate musculo-tendinous and skeletal adaptation to reduce the risk of injury. The participants were advised to attend all three supervised training sessions per week and to not engage in any additional running. They were familiarised with the Session RPE Scale (Appendix 3) and advised that their perception of effort during these training sessions should equate to a “6” on the scale of “0 – 10”. The goal of the programme was to complete the training session distance irrespective of the speed with which the session was completed. However, participants were given the opportunity to run a 2.5 km time

trial as part of their training at about 6 and 11 weeks into the programme so that they could get an indication of their improvement. The subjects were allowed to participate in other modes of exercise however it was recommended that these sessions be limited to a moderate intensity (Session RPE 6).

5.2.1.2.3 Daily records

The participants were asked to fill in a diary each day which detailed each training session in terms of the type (mode) of exercise they undertook, the duration thereof and a rating of perceived exertion (on the Session RPE Scale, Appendix 3) for each exercise session. The participant's Session RPE was reported 30 minutes after every training session. They were asked to give an overall rating of how difficult the whole workout was perceived to be, irrespective of whether the session had consisted of continuous or interval training. The participants wore heart rate monitors during all of their training sessions to record heart rate and training duration (Suunto T6 heart rate monitor, Suunto, OY, Finland). A subjective assessment of muscle soreness was reported daily, at approximately the same time of day, using an analogue scale (Appendix 6). Each of three lower limb muscles (the calf, hamstring and quadriceps) was assessed at rest, during activities of daily living and during a controlled muscle stretch. The Microsoft Access interface with which these data were captured each day is shown in Appendix 6. The participant was asked to click on each of the pointers on the left and drag it across each line to a point between the cues "No pain" (on the extreme left) and "Unbearable pain" (on the extreme right) that they thought represented their muscle sensation at that time. The actual scale of the lines is 0 to 100 and the score from each was automatically saved to a spreadsheet, which was accessed and analysed by the researcher. The participants also assessed their recovery each day using the Total Quality of Recovery Scale (Appendix 4). Every morning they rated how well they felt they had recovered over the previous 24 hours (including the previous night's sleep) on a scale of 6 – 20. An Injury/Illness Report was provided for the participants to complete in the event of them sustaining an injury or becoming ill (Appendix 9). Although every effort was made to collect a complete set of data from each participant for the entire 12-week period, various logistical reasons prevented this.

5.2.1.2.4 HIMS Tests

A HIMS test, described in Chapter 4, was performed every 2 weeks at approximately the same time of day, in order to eliminate the possible influence of diurnal changes on heart rate. The ambient air temperature was maintained between 17°C and 21°C, and the pace of each of the 4 stages of the HIMS test was 7.2 km.h⁻¹, 8.4 km.h⁻¹, 9.6 km.h⁻¹ and 10.8 km.h⁻¹ respectively.

5.2.1.2.5 Outcomes

○ *TRIMP*

Each participant wore a heart rate monitor for each exercise training session to record heart rate (b.min⁻¹) and exercise duration (min). These data, along with resting heart rate and maximal heart rate were used in the TRIMP equation, described in Chapter 4, to quantify training load for each exercise session during the study. Session training loads were then summated for each subject to calculate total training load for each week (Weekly TRIMP), and for each 2-week period between each HIMS test (2-week TRIMP).

○ *Heart rate recovery*

Heart rate recovery was assessed by expressing the heart rate 1 minute after the fourth running stage as a percentage of the heart rate attained at the end of the fourth running stage of the test (HRr%), as described in Chapter 4. In addition heart rate recovery was expressed in terms of the absolute decrease in heart rate in the first minute after Stage 4 (Equation 8)

$$\text{Absolute HRr} = \text{HR at end of Stage 4 of HIMS} - \text{HR at 1 minute of recovery} \dots \dots \dots (8)$$

○ *Submaximal heart rate*

The heart rate at the end of the fourth running stage of the HIMS test was expressed as a percent of maximal heart rate (HRS4%), as described in Chapter 4.

o *Performance*

At the beginning and at the end of the training programme the participants were asked to run and/or walk 2.4 km as fast as they could without experiencing any undue pain or discomfort. Time and heart rate were recorded during the assessment. A third performance measure, defined as the total number of heart beats during the 2.4 km assessment (2.4 km heart beats, Equation 9), was also used to assess their progress.

$$2.4 \text{ km heart beats} = 2.4 \text{ km average HR (b.min}^{-1}\text{)} \times 2.4 \text{ km time (min)} \dots\dots\dots(9)$$

o *Monotony*

Foster et al (1998) developed an index of training variability called Monotony, which they thought might contribute to the development of overtraining syndrome⁵². Monotony is the daily mean training load divided by the standard deviation of training load calculated for each week (Equation 10). They suggest that high training loads and high training Monotony may both be related to negative adaptations to training⁵². Thus, Monotony was also calculated for the participants in the current study to contribute to a more comprehensive understanding of the participants' response to their training programme.

The training load (Session Load) for each exercise session was calculated using Session RPE (Appendix 3) multiplied by exercise bout duration (min) as described in Chapter 3. Thereafter the mean daily training load and the standard deviation of daily training load were calculated for each week. Weekly Monotony was calculated by dividing the daily mean by the standard deviation for each week, as described by Foster et al (1998)⁵² (Equation 10). Monotony was also calculated for each 2-week period between successive HIMS tests by dividing the daily mean by the standard deviation for each 2-week period between each HIMS test.

$$\text{Monotony} = \frac{\text{mean daily training load}}{\text{standard deviation}} \dots\dots\dots(10)$$

5.2.1.3 Data analyses

A paired t-test for dependent variables was used to assess the effect of the training programme on 2.4 km time (min), 2.4 km average HR ($\text{b}\cdot\text{min}^{-1}$), 2.4 km heart beats, 1-minute heart rate recovery and submaximal HR ($\text{b}\cdot\text{min}^{-1}$) for the group of 10 ladies. A Pearson's Product Moment correlation assessed the relationship between 2-weekly training load (TRIMP) and HRr% as well as between the percent change in TRIMP and percent change in heart rate recovery over adjacent 2-week periods. After separating the data into groups based on whether TRIMP increased, decreased or kept constant (i.e. within a 20% range) between successive HIMS tests (i.e. 2-week periods), the percent change in heart rate recovery was analysed using an analysis of variance with repeated measures. Statistical analysis was performed using STATISTICA 7.0 data analysis software system (StatSoft, Inc. Tulsa, OK, USA) and statistical significance was accepted at $p < 0.05$. Curve fitting was applied to the individual results for submaximal heart rate (%), heart rate recovery (%), absolute heart rate recovery ($\text{b}\cdot\text{min}^{-1}$), 1- and 2-week Monotony, as well as 1- and 2-week TRIMP (AU) (Figures 5.4 – 5.24). This was accomplished with the use of the Graphpad Prism Lowess curve which follows the trend of the data (GraphPad Software, San Diego California USA).

5.2.2 RESULTS

5.2.2.1 Group Results

5.2.2.1.1 Physiology and performance

The 10 women that participated in this study had a mean (\pm SD) age of 34 ± 10 years, mass of 67 ± 14 kg, height 165 ± 5 cm, body fat $33 \pm 9\%$, estimated maximum heart rate of 193 ± 8 b.min⁻¹ and resting heart rate of 67 ± 6 b.min⁻¹. No significant changes occurred in mass (kg), BMI (kg.m⁻²), body fat percent or resting heart rate (b.min⁻¹) as a result of participation in the running programme. However, a paired t-test for dependent variables showed that significant changes occurred in selected performance and physiological variables in this group. These included 2.4 km time ($p = 0.025$), 2.4 km heart beats ($p = 0.022$), 1-minute heart rate recovery ($p = 0.004$) and submaximal heart rate ($p = 0.00004$). Table 5.1 shows the average change in performance and physiological measures for the group after 11 weeks of a running training programme.

Table 5.1: Average changes in performance and physiological measures after 11 weeks of running training. Values are expressed as mean (\pm SD).

	Week 0	Week 11	Absolute change	% change
2.4 km time (min)	18.5 (\pm 1.8)	16.5 (\pm 2.3) *	-2	-12
2.4 km average HR (b.min ⁻¹)	159 (\pm 21)	157 (\pm 18)	-2	-1
2.4 km heart beats	2944 (\pm 488)	2583 (\pm 402)*	-361	-14
HR recovery (beats)	34 (\pm 8)	48 (\pm 9) *	14	29
Submaximal HR (b.min ⁻¹)	184 (\pm 13)	176 (\pm 11) *	-8	-5

* Week 11 significantly different from Week 0 ($p < 0.05$).

5.2.2.1.2 Relationship between TRIMP and heart rate recovery

Data from all 10 participants were pooled to assess the relationship between 2-weekly TRIMP and heart rate recovery, irrespective of changes in training load between 2-week periods. This is different from what was done in Chapter 4, where only the weekly data from participants that kept their training load the same in both

weeks of the study was used in the analysis. In Chapter 4 a correlation of $r = -0.61$ was found between average weekly TRIMP and heart rate recovery. In the current study a non-significant correlation of $r = -0.15$ was calculated to occur between these 2 outcomes. The relationship between percent change in 2-weekly TRIMP and percent change in heart rate recovery was also investigated (Figure 5.1). Again, a variation of 20% was selected as a reasonable range with which to account for normal intra-individual variation. As such, those subjects that kept their training load within a 20% range were regarded as having kept their training constant over the 2 weeks. Likewise, only a change in heart rate recovery greater than 5% was regarded as being significant, as depicted in Figure 5.1. The data points lying outside these ranges do not appear to show any specific relationship with regard to changes in TRIMP and subsequent changes in heart rate recovery (Figure 5.1). When data were divided into groups based on whether TRIMP increased, decreased or remained constant between successive 2-week periods, the subsequent mean percent change in heart rate recovery (HRr%) was found to be not different between groups (Figure 5.2).

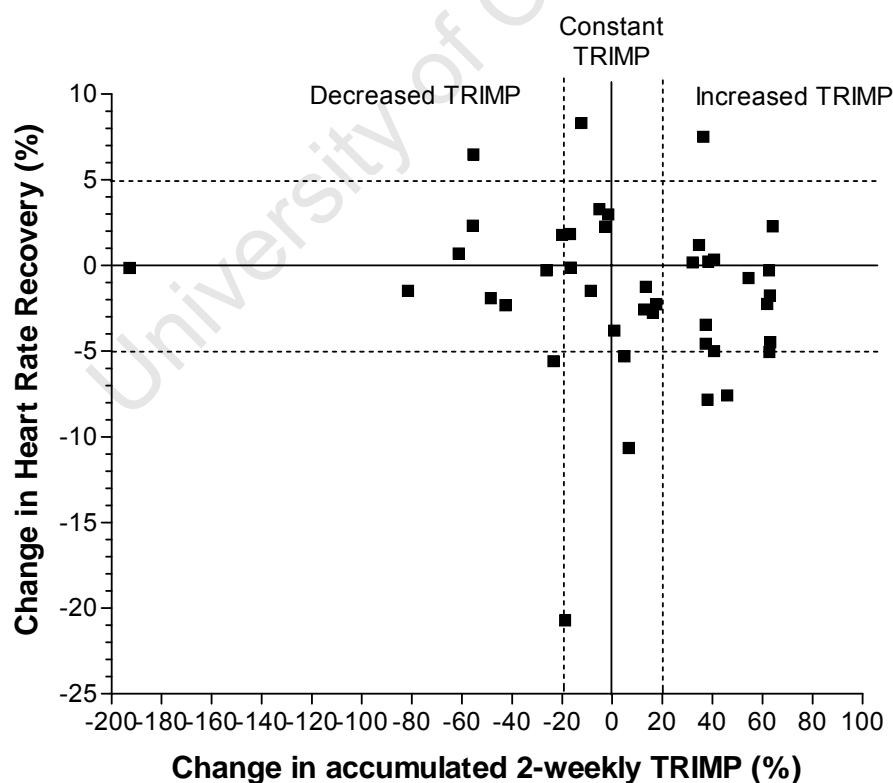


Figure 5.1: Relationship between percent change in heart rate recovery (HRr%) after HIMS tests performed at 2-week intervals and accumulated TRIMP for the 2-week periods between HIMS tests. HRr% = 1-minute recovery heart rate expressed as a percent of the heart rate reached at the end of the fourth running stage of the HIMS test.

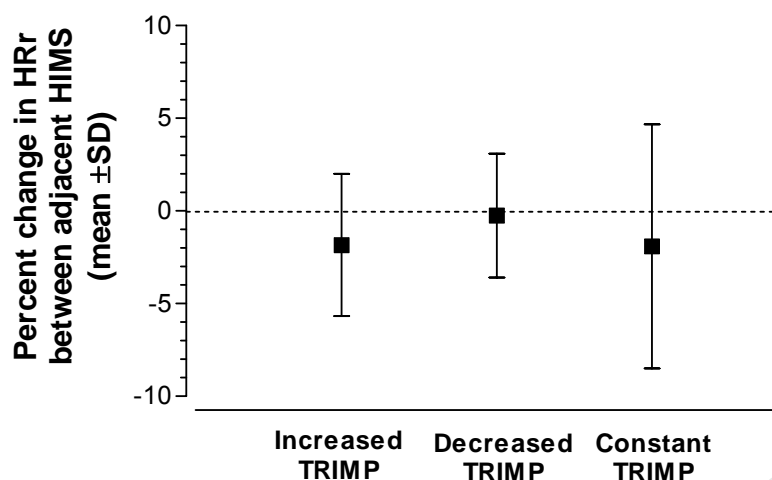


Figure 5.2: Percent change in heart rate recovery (HRr%, mean \pm SD) between adjacent HIMS tests performed every 2 weeks after an increase, decrease or no change in TRIMP accumulated over the 2-week period between HIMS tests.

Although the overall effects of the 12-week running programme are evident by the improvements in average performance and physiological measures observed in the whole group (Table 5.1), the trend observed in the study in Chapter 4, of acute changes in training loads having a predictable affect on heart rate recovery, were not evident in this group. Therefore, a more detailed investigation was prompted into the individual responses that occurred in reaction to the changes in training load experienced throughout the programme. Other factors that may have confounded or influenced this relationship were also investigated to develop a more comprehensive understanding of the response of heart rate recovery to training. Therefore the individual characteristics and physiological changes that occurred during the study are presented below as separate case studies.

5.2.2.2 Individual Results

5.2.2.2.1 CASE STUDY 1

○ Results

Participant 1 was a 42-year old female who reported that she had not been participating in any regular exercise during the month prior to beginning the 10 km running programme. She did not report any injuries or illnesses during the programme. Subject characteristics and physiological changes that occurred during the study are presented in Table 5.2.

Table 5.2: Subject characteristics and physiological changes for Participant 1 after 11 weeks of running training

Participant 1	Week 0	Week 11	Absolute change
Age (yr)	42	-	-
Mass (kg)	68.1	69.2	1.1
Height (cm)	161	-	-
BMI (kg.m ⁻²)	26.3	26.7	0.4
Body fat %	40.2	38.7	-1.5
Heart Rate (b.min ⁻¹)			
- Resting	68	64	-4
- Average (2.4 km)	166	159	-7
- Maximal	180	-	-
2.4 km heart beats	3046	2896	-150
2.4 km time (min)	18.4	18.2	-0.1
10 km time (h:min:s)	-	1:11:22	-
HIMS sub-max HR (b.min ⁻¹)	179	168	-11
HIMS HRr (beats)	32	35	3

The training of Participant 1 showed a well periodised structure as depicted in the graph of weekly TRIMP (Fig 5.4I). Total accumulated TRIMP over the entire study period was 2260 AU, the highest weekly TRIMP was 356 AU (Fig 5.4I) and average weekly TRIMP was 205 AU. Submaximal heart rate decreased by 11 b.min⁻¹ by the end of the programme (Table 5.2) but absolute 1-minute heart rate recovery only

improved by 3 beats (Table 5.2, Fig 5.4G). Performance also did not improve much, with the number of heart beats during the 2.4 km assessment decreasing by only 150 beats (Table 5.2). Total Quality of Recovery improved over the course of the running programme (Fig 5.3B), and was never reported to be worse than “*Reasonable recovery*” on the 0-20 TQR Scale (Appendix 4), suggesting that few signs of discomfort were experienced. Data from muscle soreness assessments (Fig 5.3C) may have supported this suggestion; however these data were not received from this participant.

- *Conclusion*

These results suggest that this participant may either be a “slow responder” to the training stimulus or she was training below her potential, resulting in insignificant improvements in performance and physiological adaptations. A threshold of accumulated TRIMP is likely to exist, above which training needs to be maintained in order for significant physiological adaptations to occur. Further research would be required to confirm this suggestion and to quantify this threshold for each individual. The improvement in submaximal heart rate and slight improvement in heart rate recovery also suggests that there are distinct health benefits to participating in the running programme. The pattern of changes in weekly Monotony (Fig 5.4H) is similar to the pattern of changes in weekly TRIMP (Fig 5.4I), that is when weekly TRIMP increased so too did weekly Monotony, and when weekly TRIMP decreased weekly Monotony decreased as well. The implications and causes of this association are unclear.

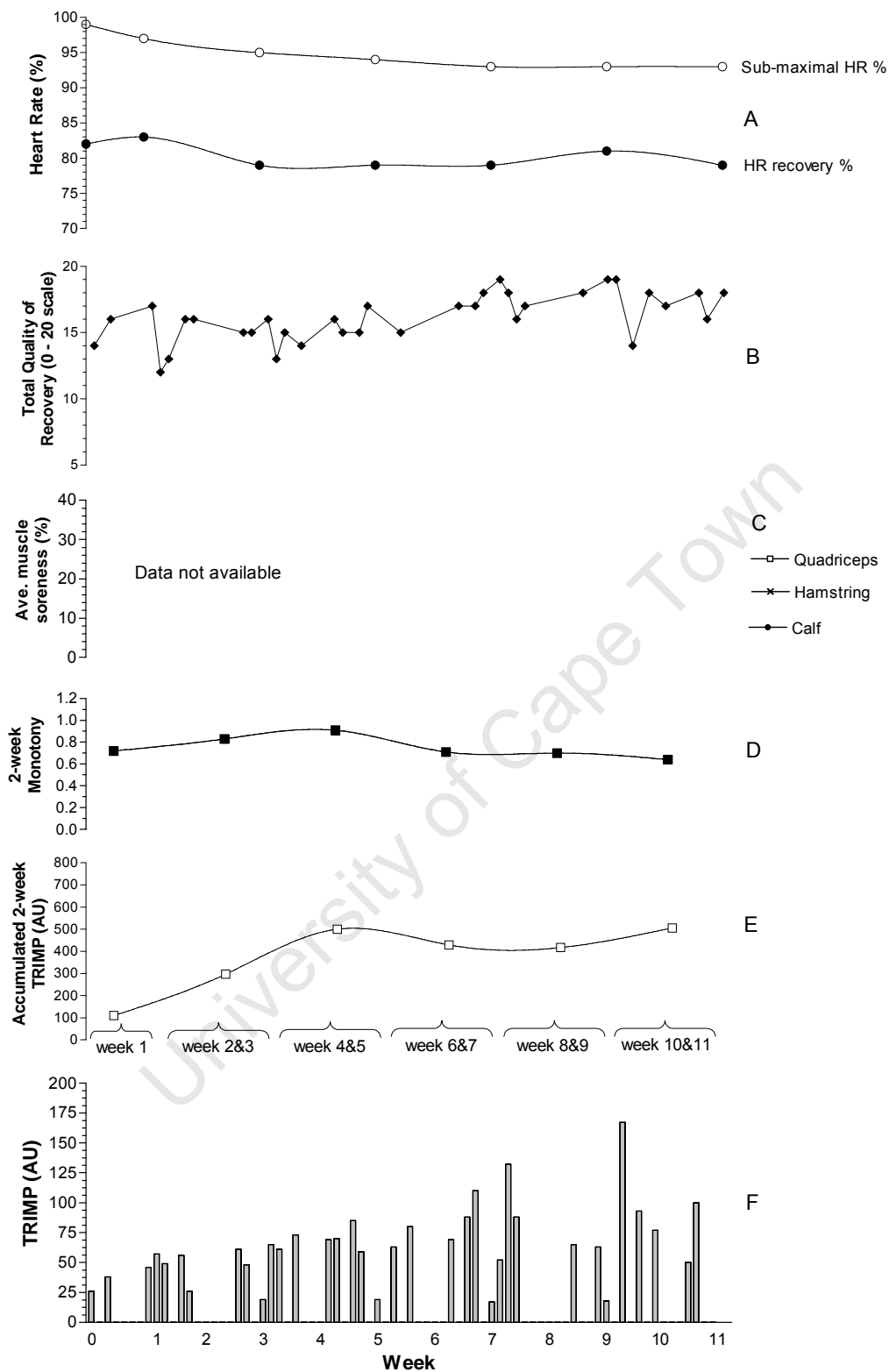


Figure 5.3: Changes in physiological and outcome measures for Participant 1 after 11 weeks of running training. A - Submaximal heart rate percent (○) and heart rate recovery percent (●). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (□), Hamstring (x) and Calf (●). D - Monotony accumulated over 2-week periods between HIMS tests. E - TRIMP (AU) accumulated over 2-week periods between HIMS tests. F - TRIMP (AU) for each training session.

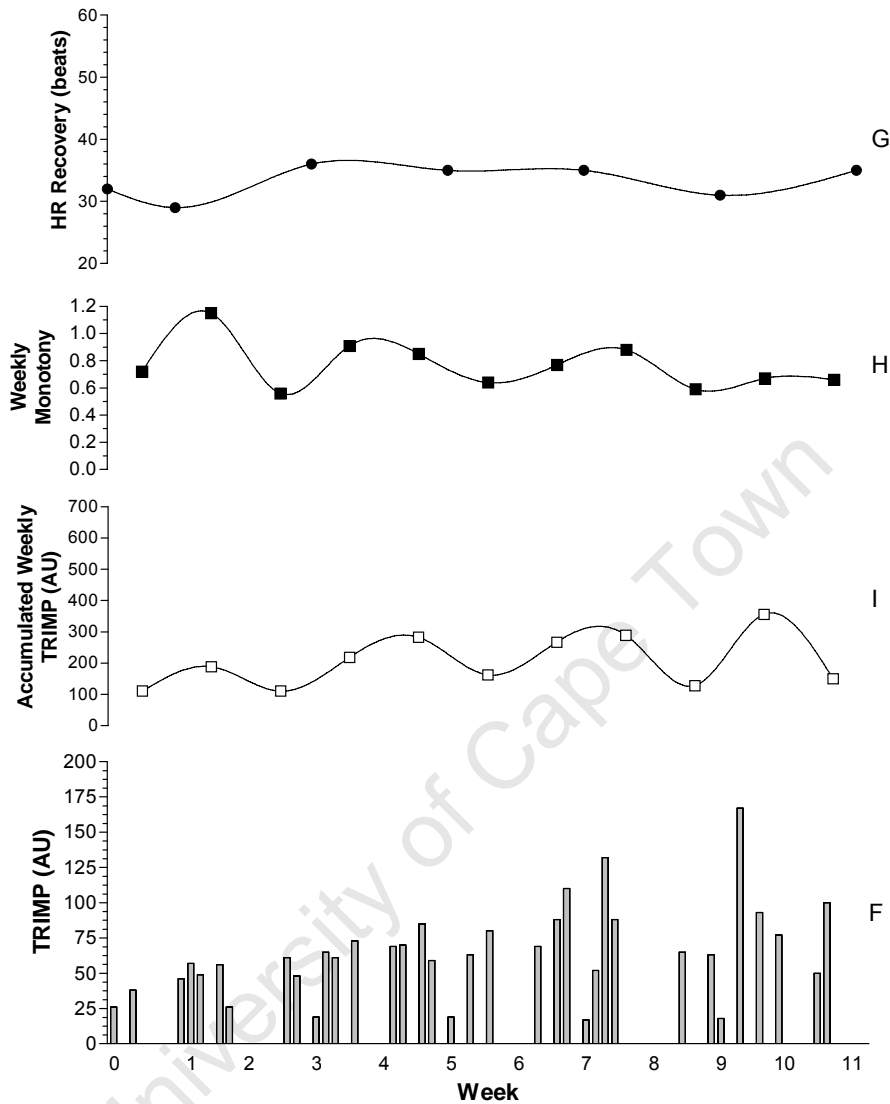


Figure 5.4: Changes in physiological and outcome measures for Participant 1 after 11 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F – TRIMP (AU) for each training session.

5.2.2.2.2 CASE STUDY 2

○ Results

Participant 2 was a 35-year old female who had not engaged in any regular exercise during the month prior to starting the 10 km running programme. She did not report having any injuries or illnesses during the 12 weeks of the programme. Four months after completing this 10 km running programme, participant 2 went on to run a 56km ultra-marathon in a time of 6:24:33 (h:min:s). Subject characteristics and physiological changes that occurred during the study are presented in Table 5.3.

Table 5.3: Subject characteristics and physiological changes for Participant 2 after a running training programme

Participant 2	Week 0	Week 11	Absolute change
Age (yr)	35	-	-
Mass (kg)	47	-	-
Height (cm)	165.5	-	-
BMI (kg.m ⁻²)	17.2	-	-
Body fat %	17.2	-	-
Heart Rate (b.min ⁻¹)			
- Resting	58	65	7
- Average (2.4 km)	129	143	14
- Maximal	198	-	-
2.4 km heart beats	2124	1716	-408
2.4 km time (min)	16.5	12.0	-4.5
10 km time (h:min:s)	-	0:56:02	-
HIMS sub-max HR (b.min ⁻¹)	197	185	-12
HIMS HRr (beats)	32	51	19

Total accumulated TRIMP over the study period was 2642 AU, the highest weekly TRIMP was 424 AU (Fig 5.6I) and average weekly TRIMP was 240 AU. Submaximal heart rate decreased by 12 b.min⁻¹ by the end of the programme (Table 5.3) and absolute 1-minute heart rate recovery improved by 19 beats (Table 5.3, Fig 5.6G). Performance also improved significantly, with the number of heart beats during the 2.4 km assessment decreasing by 408 beats and her time improving by 4.5 minutes

(Table 5.3). Subjective assessments of muscle soreness remained below 10 on an analogue scale of 0-100 (Appendix 6) except on two occasions, during week 3 and 4 (Fig 5.5C). These dramatic increases in muscle soreness do not appear to be related to any specific intervention in the training programme. Although not reported, the possibility of strenuous recreational activity causing these increases in muscle soreness can not be ruled out. Data from assessments of Total Quality of Recovery (Fig 5.5B) may have contributed to a more comprehensive understanding of this participant's daily recovery from training bouts; however these data were not received from this participant.

- *Conclusion*

These results suggest that this participant may be a “fast responder” to the training stimulus as she appears to have adapted well, evidenced by significant improvements in performance and physiological adaptations with little muscle discomfort. The pattern of changes in weekly Monotony (Fig 5.6H) is very similar to the pattern of changes in weekly TRIMP (Fig 5.6I), such that when weekly TRIMP increased, so too did weekly Monotony, and vice versa. The implications and causes of this finding are unclear.

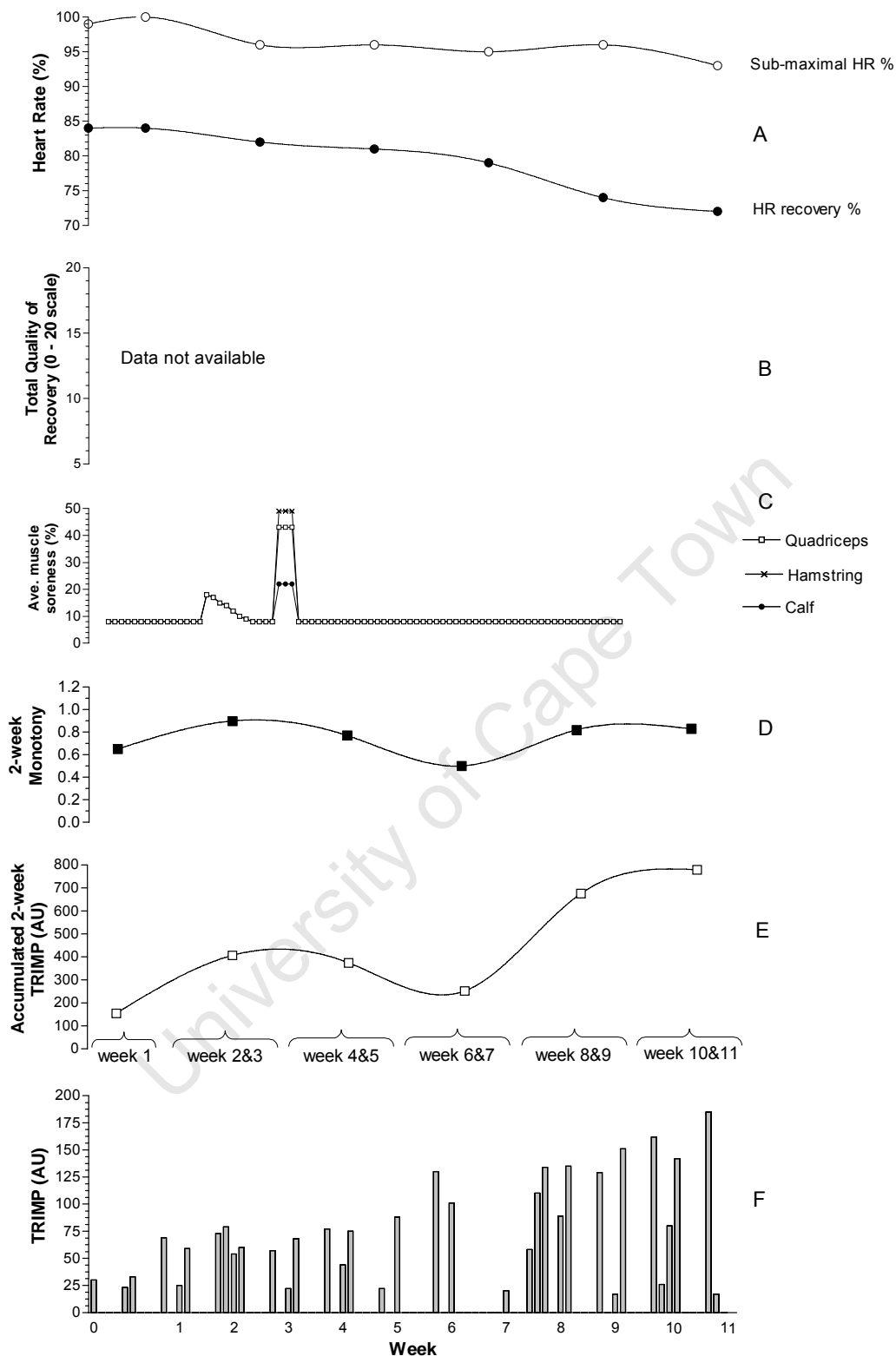


Figure 5.5: Changes in physiological and outcome measures for Participant 2 after 11 weeks of running training. A - Submaximal heart rate percent (○) and heart rate recovery percent (●). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (□), Hamstring (x) and Calf (●). D - Monotony accumulated over 2-week periods between HIMS tests. E - TRIMP (AU) accumulated over 2-week periods between HIMS tests. F - TRIMP (AU) for each training session.

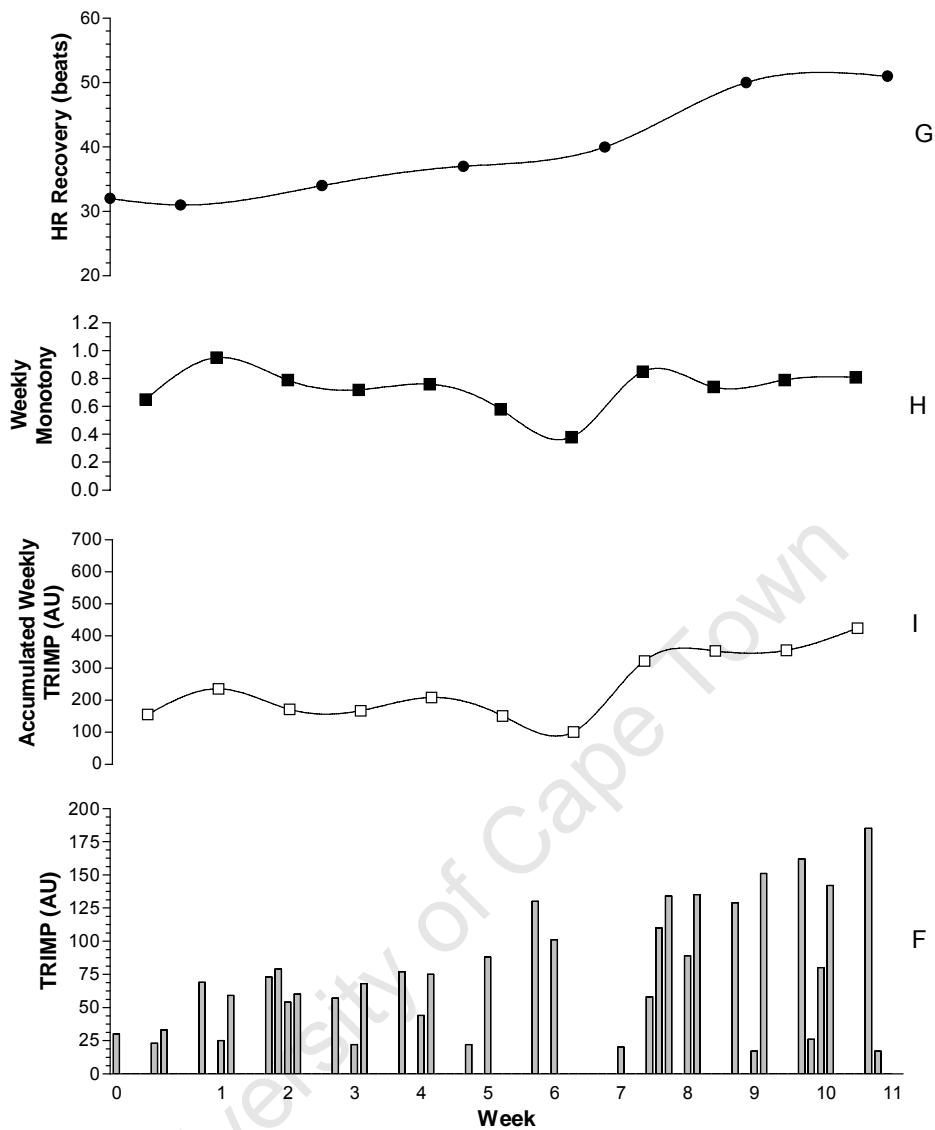


Figure 5.6: Changes in physiological and outcome measures for Participant 2 after 11 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F - TRIMP (AU) for each training session.

5.2.2.2.3 CASE STUDY 3

○ Results

Participant 3 was a 34-year old female who reported she had not been doing any regular exercise for at least a month prior to embarking on the 10 km running programme. She reported having a cold during weeks 2, 5, 9 and 10 of the 12-week programme. This affected her ability to train optimally during weeks 2 and 5, and prevented her from training during weeks 9 and 10. Subject characteristics and physiological changes that occurred during the study are presented in Table 5.4.

Table 5.4: Subject characteristics and physiological changes for Participant 3 after a running training programme

Participant 3	Week 0	Week 10	Absolute change
Age (yr)	34	-	-
Mass (kg)	68.7	72	3.3
Height (cm)	176	-	-
BMI (kg.m ⁻²)	22.2	23.2	1.1
Body fat %	32.3	32.7	0.4
Heart Rate (b.min ⁻¹)			
- Resting	66	60	-6
- Average (2.4 km)	179	163	-16
- Maximal	193	-	-
2.4 km heart beats	3493	2752	-742
2.4 km time (min)	19.5	16.9	-2.6
10 km time (h:min:s)	-	1:12:42	-
HIMS sub-max HR (b.min ⁻¹)	192	185	-7 #
HIMS HRr (beats)	32	37	5 #

Data from the HIMS test performed after week 8

Total TRIMP accumulated over the entire study period was 2203 AU, the highest weekly TRIMP was 293 AU (Fig 5.8I) and average weekly TRIMP was 220 AU. Submaximal heart rate decreased by 7 b.min⁻¹ by week 8 of the programme (Table 5.4) and absolute 1-minute heart rate recovery improved by 5 beats (Table 5.4, Fig 5.8G). Data from the HIMS test performed after week 8 have been reported in Table

4 and used to assess the effect of training on submaximal heart rate and heart rate recovery, as it was suspected that the cold and fever reported during weeks 9 and 10 may have had an effect on these indices during the last HIMS test (after week 10). This can be seen in Fig 5.7A and Fig 5.8G in which data from the last HIMS has been included. Whether it was the illness itself that caused the decrease in heart rate recovery or the fact that training ceased during that period cannot be discerned with the available data. Despite the illnesses reported, participant 3 performed significantly better in the 2.4 km assessment at the end of the running programme (total number of heart beats decreased by 742 beats, Table 5.4). Ratings of Total Quality of Recovery below 10 on the 0 – 20 TQR Scale (Appendix 4) in weeks 2, 7, 9 and 10 (Fig 5.7B) appear to relate somewhat to the illnesses reported by the participant in weeks 2, 5, 9 and 10 (Fig 5.7F).

- o *Conclusion*

The participant's Total Quality of Recovery in this instance may have reflected the effect that the illness had on recovery rather than being an indication of how the participant was recovering from the training stimulus. This suggestion may be supported by that fact that only slight increases in muscle soreness were reported in weeks 1, 8 and 9 (Fig 5.7C). A "spike" in weekly training Monotony is evident in week 1 and week 4 (Fig 5.8H) and precedes reported illnesses in weeks 2 and 5 (Fig 5.8F). As suggested by Foster et al⁵² there may exist a "threshold" of training Monotony, above which the risk of becoming ill is increased. For this participant the threshold may be around 1.0. However, no preceding spike was evident before the illness that was reported in weeks 9 and 10 (Fig 5.8F). Significant improvements in performance in this case suggest that this participant may be a "fast responder", even to the small training loads which she experienced.

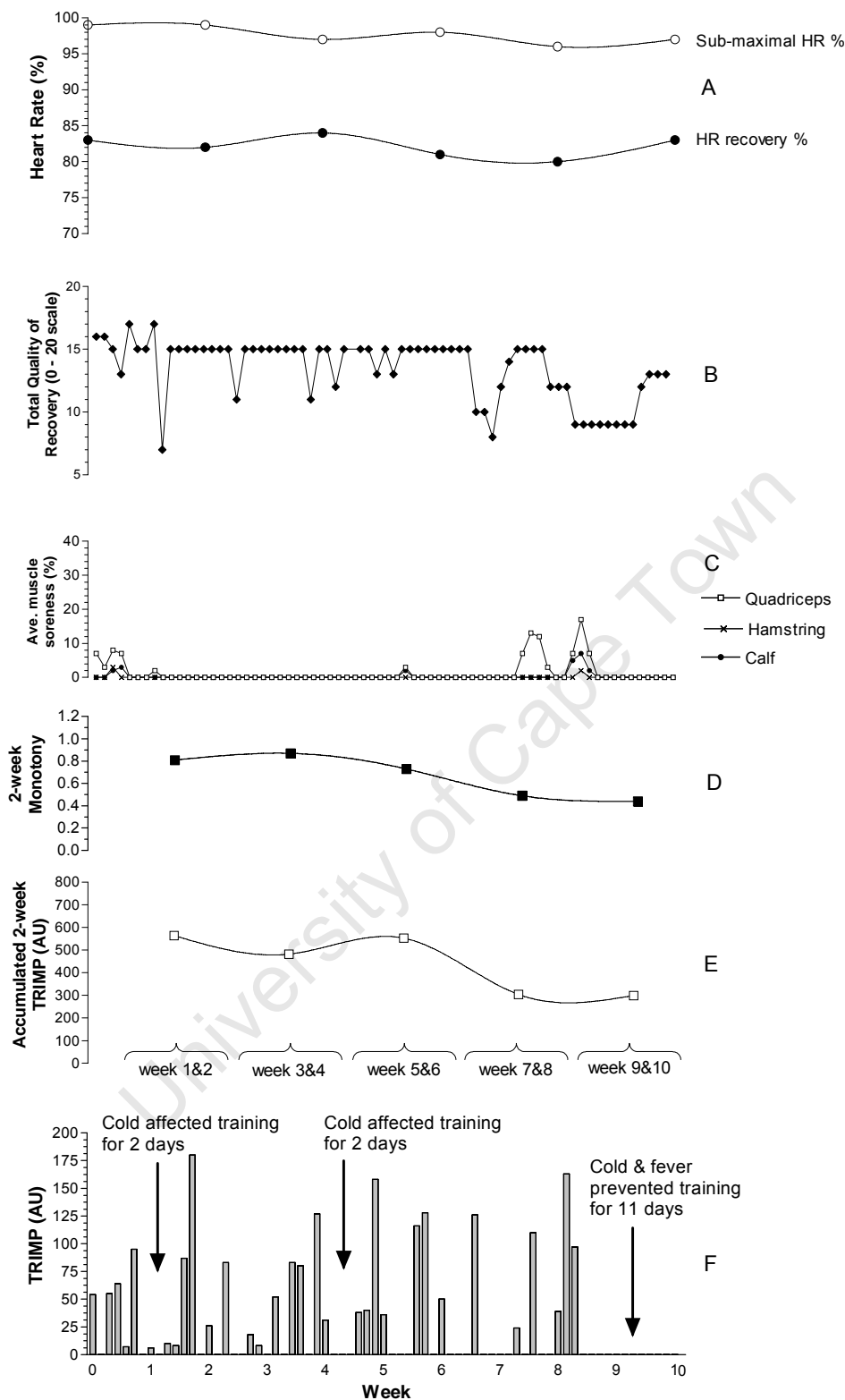


Figure 5.7: Changes in physiological and outcome measures for Participant 3 after 10 weeks of running training. A - Submaximal heart rate percent (\circ) and heart rate recovery percent (\bullet). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (\square), Hamstring (\times) and Calf (\bullet). D - Monotony accumulated over 2-week periods between HIMS tests. E - TRIMP (AU) accumulated over 2-week periods between HIMS tests. F - TRIMP (AU) for each training session.

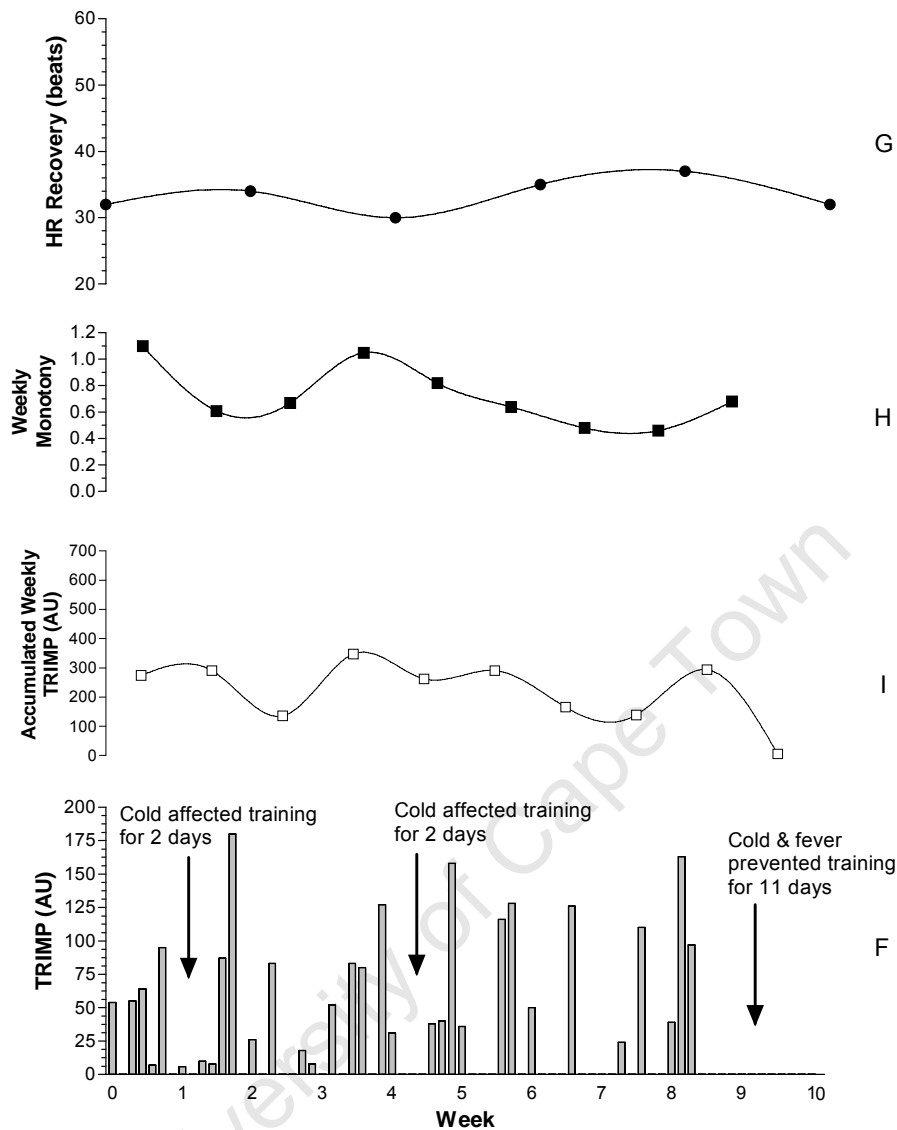


Figure 5.8: Changes in physiological and outcome measures for Participant 3 after 10 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F - TRIMP (AU) for each training session.

5.2.2.2.4 CASE STUDY 4

○ Results

Participant 4 was a 45-year old female who reported that she had been participating in one 60-minute step class and 20 minutes of walking per week during the month before she began the 10 km running programme. During week 7 of the 12 week programme she reported experiencing nausea and headaches that prevented her from training for 2 days. During week 9 menstrual pain prevented her from training for 2 days, and during week 10 pain in her left calf affected her ability to train optimally for 1 day. Subject characteristics and physiological changes that occurred during the study are presented in Table 5.5.

Table 5.5: Subject characteristics and physiological changes for Participant 4 after a running training programme

Participant 4	Week 0	Week 11	Absolute change
Age (yr)	45	-	-
Mass (kg)	60.3	63.1	2.8
Height (cm)	158	-	-
BMI (kg.m ⁻²)	24.2	25.3	1.1
Body fat %	38.4	35.6	-2.8
Heart Rate (b.min ⁻¹)			
- Resting	61	51	-10
- Average (2.4 km)	152	153	1
- Maximal	181	-	-
2.4 km heart beats	2736	2889	153
2.4 km time (min)	18.0	18.9	0.9
10 km time (h:min:s)	-	1:13:54	-
HIMS sub-max HR (b.min ⁻¹)	163	156	-7
HIMS HRr (beats)	41	54	13

Total accumulated TRIMP over the entire study period was 2570 AU, the highest weekly TRIMP was 334 AU (Fig 5.10I) and average weekly TRIMP was 234 AU. Submaximal heart rate decreased by 7 b.min⁻¹ by the end of the programme (Table 5.5) and absolute 1-minute heart rate recovery improved by 13 beats (Table 5.5 and

Fig 5.10G). Performance decreased, with the number of heart beats during the 2.4 km assessment increasing by 153 beats and time increasing by 0.9 minutes (Table 5.5). Total Quality of Recovery remained within a range of between 15 and 20 on the 0-20 TQR Scale (Appendix 4) throughout the programme (Fig 5.9B) suggesting that the participant recovered well each day. The highest spike in weekly training Monotony occurred in week 6 (Fig 5.10H) and was the only recording of weekly Monotony above 1.0. Late in week 7 the participant reported nausea and headache that prevented her from training for 2 days (Fig 5.10F).

- *Conclusion*

These results suggest that this participant may either be a “slow responder” to the training stimulus provided during the training programme or she was training below her potential. While physiological changes are evident, the decrease in performance suggests that the participant may still be in an “adaptation phase” of training, where changes in physiology have not yet translated into improvements in performance. The participant also reported muscle soreness that remained between 10 and 20 on an analogue scale of 0-100 (Appendix 6) throughout the programme, with a few distinct peaks above 25 (Fig 5.9C). This may support the suggestion that she is a slow responder rather than she was training below her potential. The improvement in submaximal heart rate and heart rate recovery despite decreases in performance also suggests that there are possible health benefits as a result of participating in the running programme.

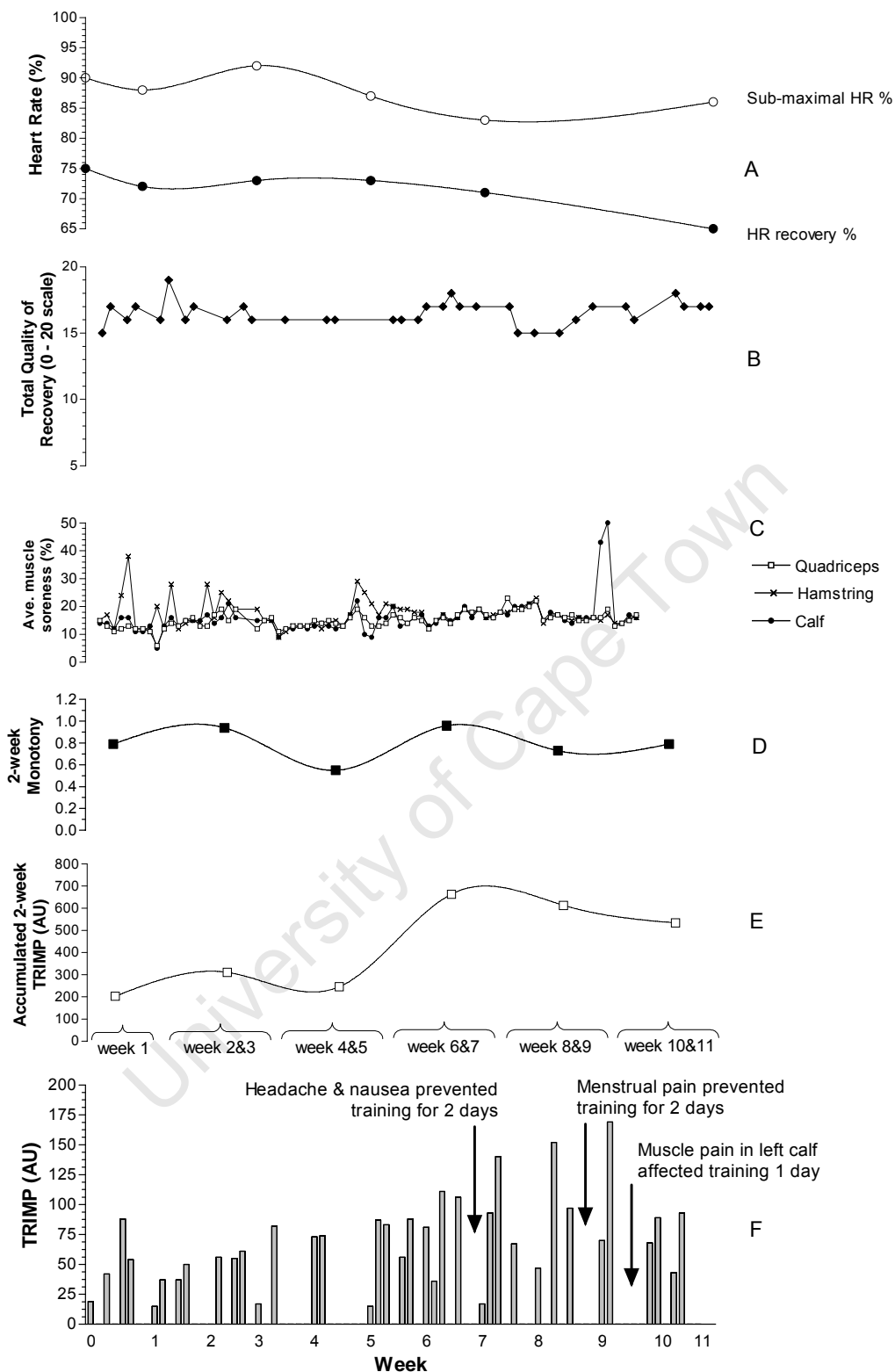


Figure 5.9: Changes in physiological and outcome measures for Participant 4 after 11 weeks of running training. A - Submaximal heart rate percent (\circ) and heart rate recovery percent (\bullet). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (\square), Hamstring (\times) and Calf (\bullet). D - Monotony accumulated over 2-week periods between HIMS tests. E - TRIMP (AU) accumulated over 2-week periods between HIMS tests. F - TRIMP (AU) for each training session.

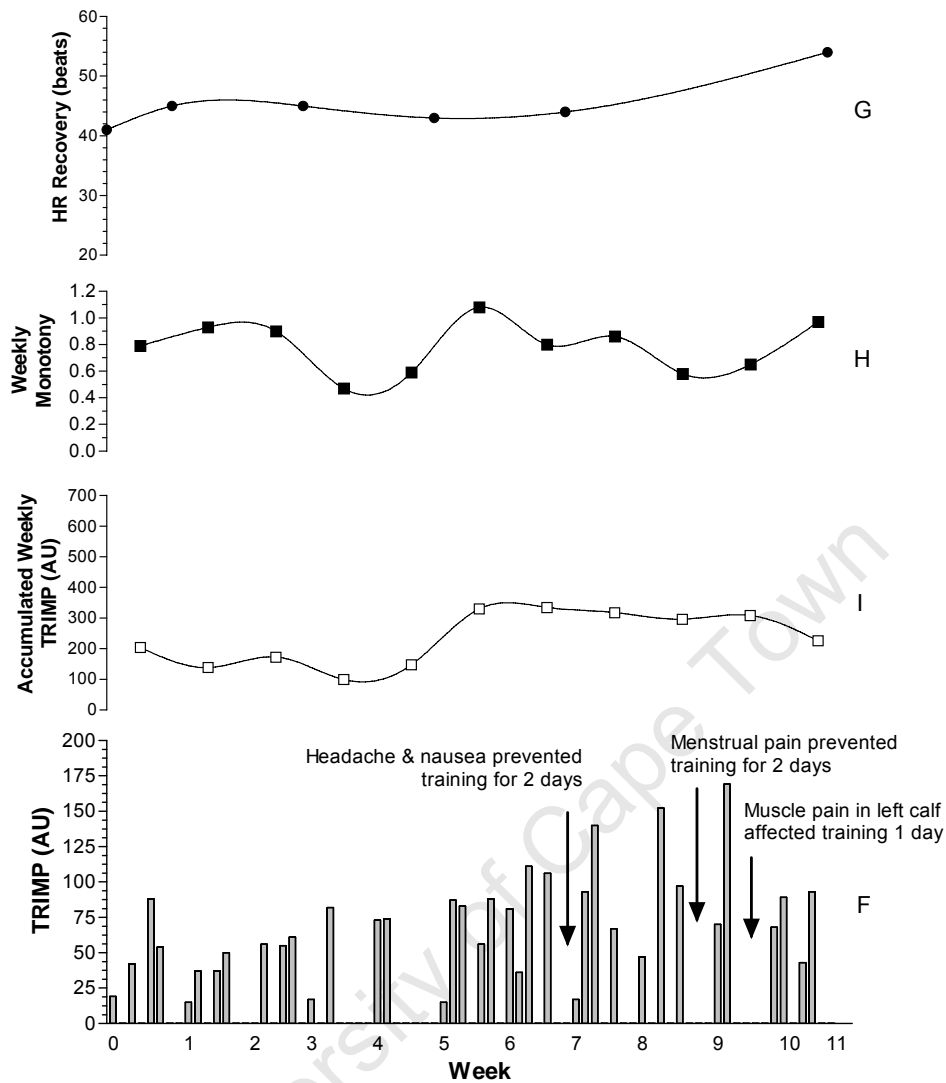


Figure 5.10: Changes in physiological and outcome measures for Participant 4 after 11 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F - TRIMP (AU) for each training session.

5.2.2.2.5 CASE STUDY 5

○ Results

Participant 5 was a 47-year old female who was participating in one to two 60-minute walks per week during the month before the start of the 10 km running programme. During weeks 6 and 7 of the programme she had bronchitis which prevented her from training for 13 days, and affected her ability to train optimally for a further 3 days. Subject characteristics and physiological changes that occurred during the study are presented in Table 5.6.

Table 5.6: Subject characteristics and physiological changes for Participant 5 after a running training programme

Participant 5	Week 0	Week 10	Absolute change
Age (yr)	47	-	-
Mass (kg)	99.7	98.6	-1.1
Height (cm)	168	-	-
BMI (kg.m ⁻²)	35.3	34.9	-0.4
Body fat %	45.5	45.7	0.2
Heart Rate (b.min ⁻¹)			
- Resting	65	65	0
- Average (2.4 km)	144	158	14
- Maximal	183	-	-
2.4 km heart beats	3180	2912	-268
2.4 km time (min)	22.1	18.4	-3.7
10 km time (h:min:s)	-	1:30:16	-
HIMS sub-max HR (b.min ⁻¹)	163	161	-2
HIMS HRr (beats)	43	50	7

Total accumulated TRIMP over the entire study period was 1739 AU, the highest weekly TRIMP was 248 AU (Fig 5.12I) and average weekly TRIMP was 174 AU. Submaximal heart rate decreased by 2 b.min⁻¹ by the end of the programme (Table 5.6) and absolute 1-minute heart rate recovery improved by 7 beats (Table 5.6, Fig 5.12G). Performance also improved, with the number of heart beats during the 2.4 km assessment decreasing by 268 beats and time improving by 3.7 minutes (Table

5.6). Total Quality of Recovery remained around 15 on the 0-20 TQR Scale (Appendix 4), indicating “*Good Recovery*” throughout the running programme (Fig 5.11B). The training programme is designed to have an increase in training load at about six weeks. However, Participant 5 missed this valuable part of the programme due to illness (Fig 5.11F), yet still showed improvements in performance overall. Interestingly, the trend towards a decrease in submaximal heart rate and the improvements in heart rate recovery seemed to be interrupted after week 6, the time of the reported illness (Fig 5.11A and 5.12G), suggesting an influence of either bacterial or viral infection on heart rate. The participant reported fluctuations in muscle soreness between 0 and 20 on an analogue scale of 0-100 (Appendix 6) throughout the rest of the training period (Fig 5.11C). The Monotony of Participant 5’s training increased to around 0.9 in week 5 (Fig 5.12H) and was followed by a report of bronchitis in week 7 that prevented the participant training for 13 days (Fig 5.12F). This supports Foster’s proposal of a relationship between increases in training Monotony and banal illness⁵². However a similar increase in Monotony in weeks 1 and 2 (Fig 5.12H) was not followed by a subsequent report of illness.

- *Conclusion*

These results suggest that this participant may be a “fast responder” to even small training loads (stimuli), since significant improvements in performance and evidence of physiological adaptations were observed despite the relatively small weekly and total TRIMP to which this participant’s body was exposed. This finding shows that if a threshold of accumulated TRIMP exists, as discussed above, then the threshold value may be unique to each individual. Participant 5 for example appears to have a low threshold value to which she adapted well, whereas Participant 1 did not adapt as well to weekly and accumulated training loads of higher value.

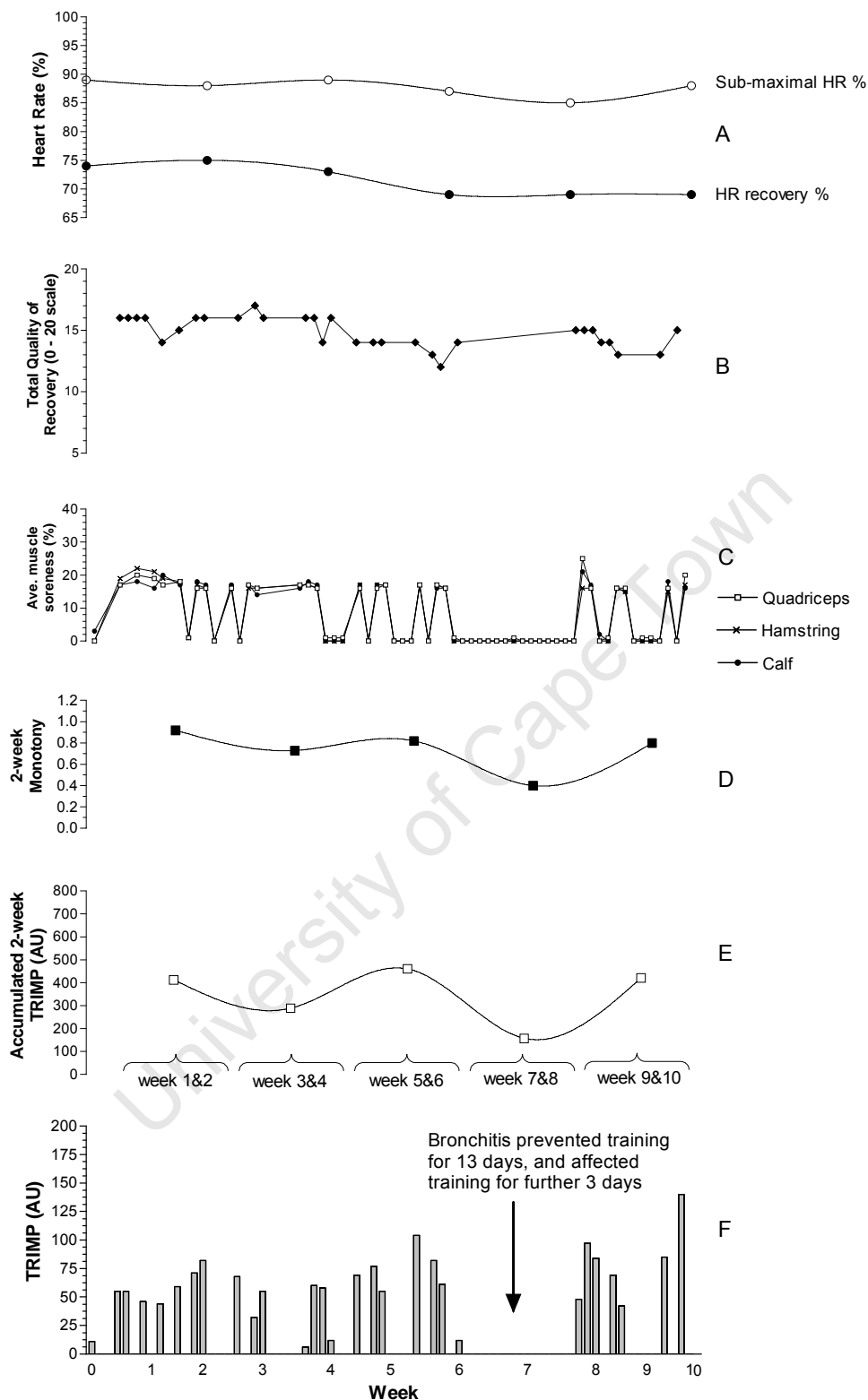


Figure 5.11: Changes in physiological and outcome measures for Participant 5 after 10 weeks of running training. A - Submaximal heart rate percent (○) and heart rate recovery percent (●). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (□), Hamstring (x) and Calf (●). D - Monotony accumulated over 2-week periods between HIMS tests. E - TRIMP (AU) accumulated over 2-week periods between HIMS tests. F - TRIMP (AU) for each training session.

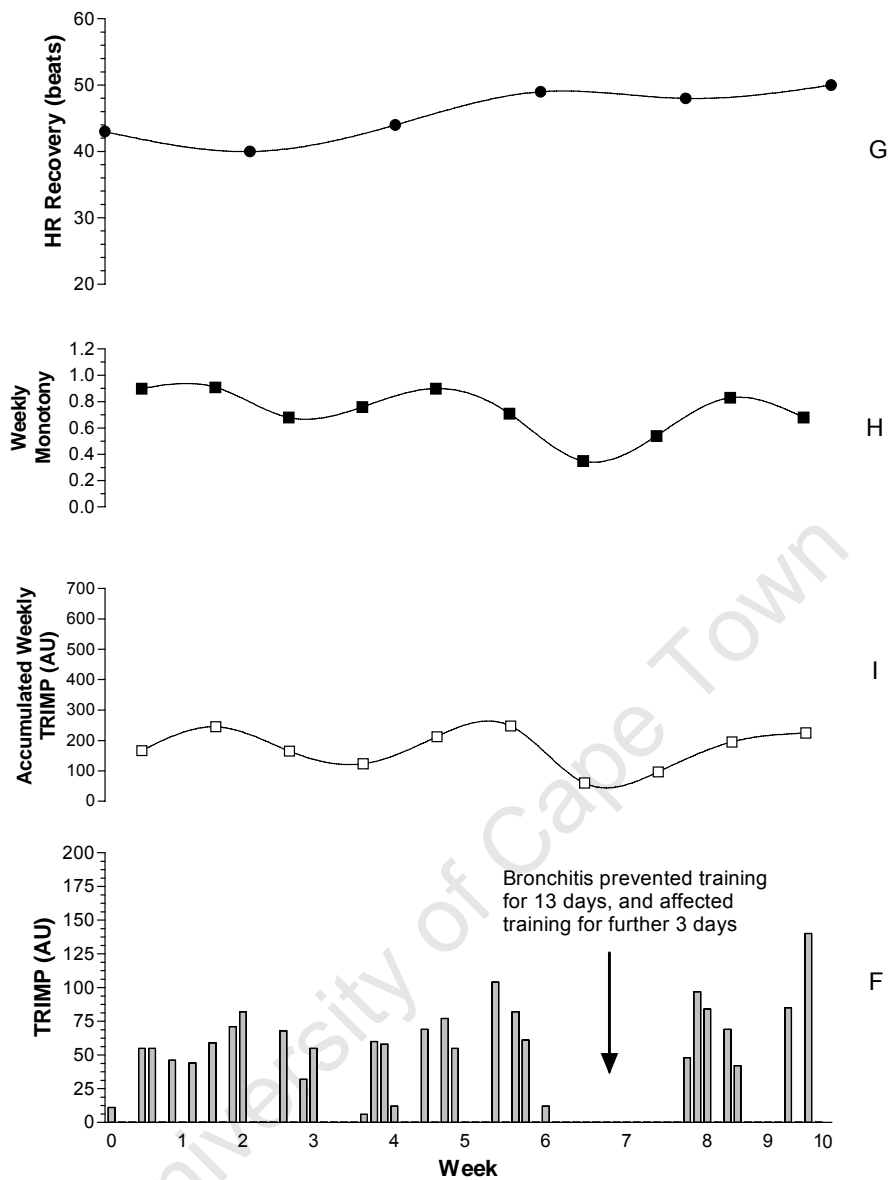


Figure 5.12: Changes in physiological and outcome measures for Participant 5 after 10 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F - TRIMP (AU) for each training session.

5.2.2.2.6 CASE STUDY 6

○ Results

Participant 6 was a 25-year old female who reported she had been participating in 20 minutes of running and 10 minutes of stepping twice a week as well as 30 minutes of super-circuit training each week for the month prior to the start of the 10 km running programme. She did not report having any injuries or illnesses during the 12 weeks of the programme. The time in which she ran her 10 km at the end of the running programme was not reported, however four months after completing this programme, participant 6 went on to run a 21km (half marathon) race in a time of 2:21:39 (h:min:s). Subject characteristics and physiological changes that occurred during the study are presented in Table 5.7.

Table 5.7: Subject characteristics and physiological changes for Participant 6 after a running training programme

Participant 6	Week 0	Week 11	Absolute change
Age (yr)	25	-	-
Mass (kg)	57	-	-
Height (cm)	165	-	-
BMI (kg.m ⁻²)	20.9	-	-
Body fat %	22	-	-
Heart Rate (b.min ⁻¹)			
- Resting	73	82	9
- Average (2.4 km)	140	119	-21
- Maximal	195	-	-
2.4 km heart beats	2567	2142	-425
2.4 km time (min)	18.3	18.0	-0.3
10 km time (h:min:s)	-	-	-
HIMS sub-max HR (b.min ⁻¹)	185	180	-5
HIMS HRr (beats)	35	56	21

The training of Participant 6 showed a well periodised structure as depicted in the graph of weekly TRIMP (Fig 5.14I). Total accumulated TRIMP over the entire study period was 2237 AU, the highest weekly TRIMP was 297 AU (Fig 5.14I) and average

weekly TRIMP was 203 AU. Submaximal heart rate decreased by $5 \text{ b}\cdot\text{min}^{-1}$ by the end of the programme (Table 5.7) and absolute 1-minute heart rate recovery improved by 21 beats (Table 5.7, Fig 5.14G). Although her 2.4 km time did not improve much, the number of heart beats during the assessment decreased by 425 beats, due to a considerable decrease in average heart rate during the assessment (Table 5.7). Assessments of muscle soreness did not exceed 15 on the analogue scale of 0-100 (Appendix 6) throughout the running programme (Fig 5.13C), suggesting that few signs of discomfort were experienced. Data from assessments of Total Quality of Recovery (Fig 5.13B) would have been useful to confirm this deduction however these data were not received from this participant.

- *Conclusion*

These results suggest that Participant 6 may be a “fast responder” to even small training stimuli, since significant improvements in performance and physiological adaptations were observed despite the relatively small weekly and total TRIMP to which her body was exposed. The pattern of changes in weekly Monotony (Fig 5.14H) is very similar to the pattern of changes in weekly TRIMP (Fig 5.14I); that is when weekly TRIMP increased so too did weekly Monotony, and vice versa. The implications and causes of this are unclear.

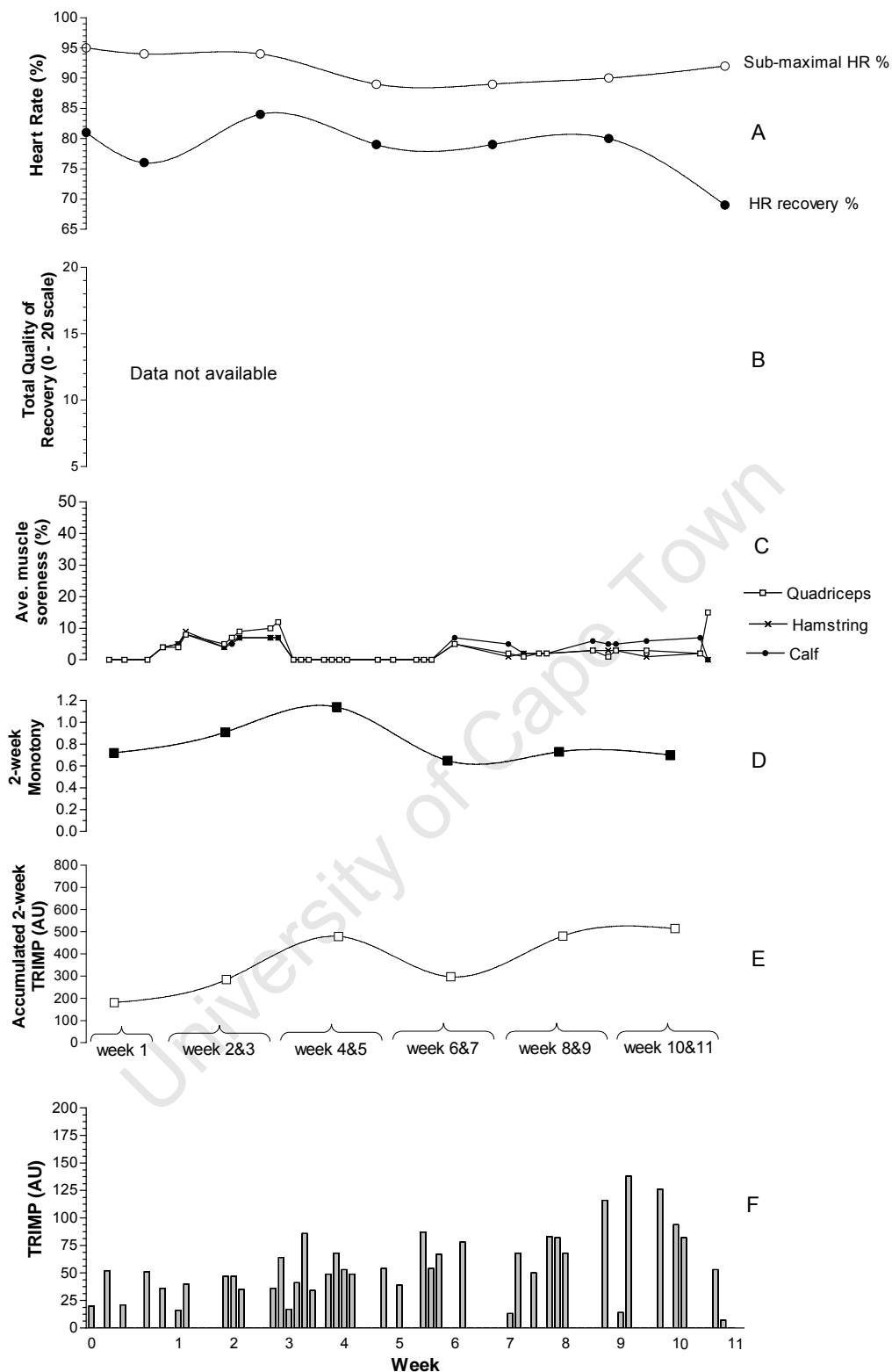


Figure 5.13: Changes in physiological and outcome measures for Participant 6 after 11 weeks of running training. A - Submaximal heart rate percent (○) and heart rate recovery percent (●). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (□), Hamstring (x) and Calf (●). D - Monotony accumulated over 2-week periods between HIMS tests. E - TRIMP (AU) accumulated over 2-week periods between HIMS tests. F - TRIMP (AU) for each training session.

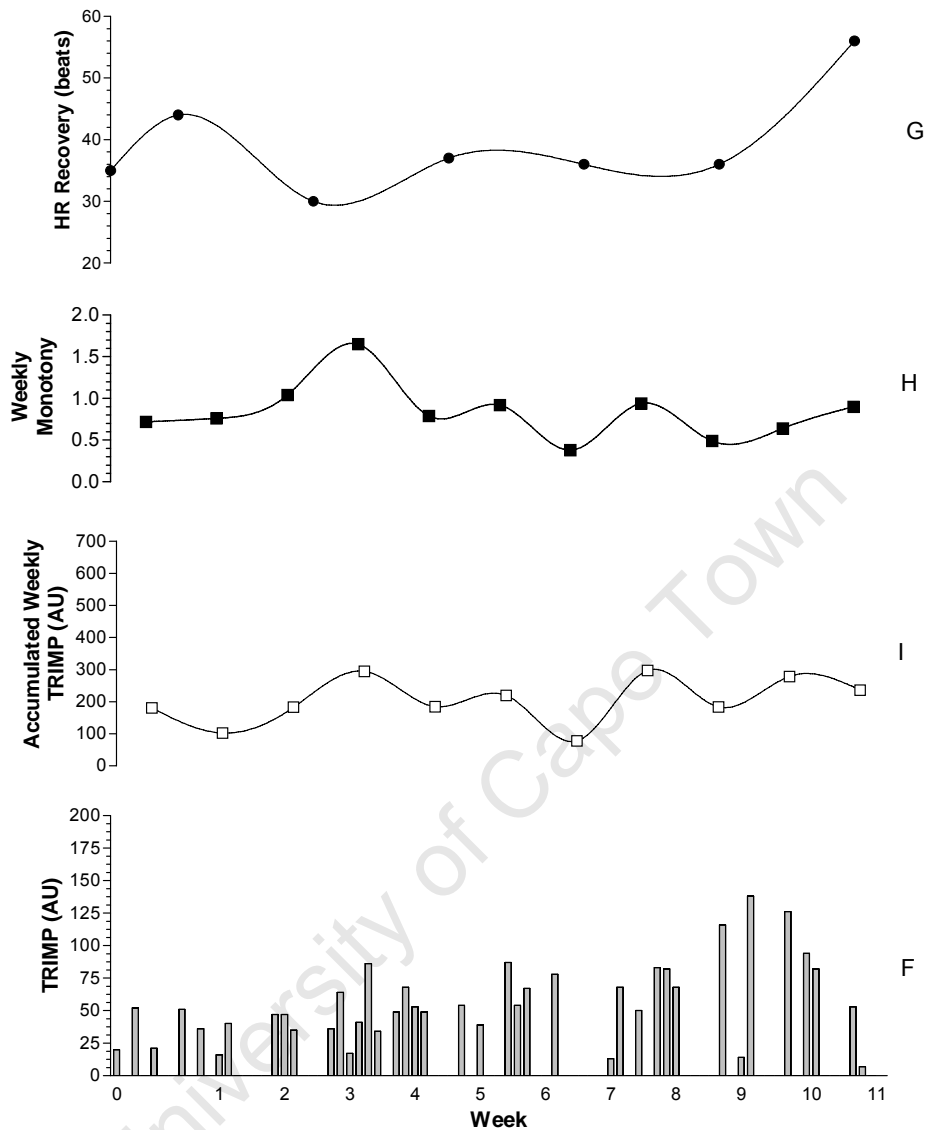


Figure 5.14: Changes in physiological and outcome measures for Participant 6 after 11 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F – TRIMP (AU) for each training session.

5.2.2.2.7 CASE STUDY 7

○ Results

Participant 7 was a 21-year old female who had completed a 30-minute run, a 30-minute swim and a 15-minute cycle each week in the 4 weeks preceding the start of the 10 km running programme. She did not report having any injuries or illnesses during the 12 weeks of the programme. One exercise session at the beginning of week 7 accumulated many more TRIMP (459 AU) than any of the other training sessions during the study period. This was a 109km cycling race that participant 7 completed in 4h39min. Subject characteristics and physiological changes that occurred during the study are presented in Table 5.8.

Table 5.8: Subject characteristics and physiological changes for Participant 7 after a running training programme

Participant 7	Week 0	Week 11	Absolute change
Age (yr)	21	-	-
Mass (kg)	61.5	61	-0.5
Height (cm)	166	-	-
BMI (kg.m ⁻²)	22.3	22.1	-0.2
Body fat %	32.1	29.1	-3.0
Heart Rate (b.min ⁻¹)			
- Resting	65	69	4
- Average (2.4 km)	-	-	-
- Maximal	199	-	-
2.4 km heart beats	-	-	-
2.4 km time (min)	-	-	-
10 km time (h:min:s)	-	-	-
HIMS sub-max HR (b.min ⁻¹)	185	173	-12
HIMS HRr (beats)	39	52	13

Total accumulated TRIMP over the study period was 3852 AU, the highest weekly TRIMP was 754 AU (Fig 5.16I) and average weekly TRIMP was 350 AU. Submaximal heart rate decreased by 12 b.min⁻¹ by the end of the programme (Table 5.8) and absolute 1-minute heart rate recovery improved by 13 beats (Table 5.8, Fig

5.16G). No performance data were reported for this participant however it is evident that significant physiological adaptations occurred over the period of the running programme. Total Quality of Recovery (Fig 5.15B) remained above 17 (“*Very good recovery*”) on the 0-20 TQR Scale (Appendix 4) and very little muscle soreness (Fig 5.15C) was reported throughout the study, indicating that very few signs of discomfort were experienced. Three slight declines in Total Quality of Recovery to scores of 17 and 18 in weeks 2, 7 and 10 appeared to coincide with increases in TRIMP immediately before (Fig 5.15F and 5.16I).

- *Conclusion*

These results suggest that this participant may be a “fast responder” as she appears to have adapted well physiologically to the training stimulus. In addition, no negative physiological effects were reported, suggesting that this participant was training above her individual training load threshold required to induce physiological adaptations, but below an upper limit that might induce muscle soreness and sub-optimal recovery. The pattern of changes in weekly Monotony (Fig 5.16H) appears to be inversely related to the pattern of changes in weekly TRIMP (5.16I). As such, when weekly TRIMP increased weekly Monotony decreased (i.e. training during that week became more varied), and when weekly training load decreased weekly Monotony increased. This is in contrast to most of the other participants, whose changes in Monotony appeared very similar to changes in TRIMP. The implications of this difference between participants, and what makes the Monotony-TRIMP relationship unique in participant 7 is unclear at this stage.

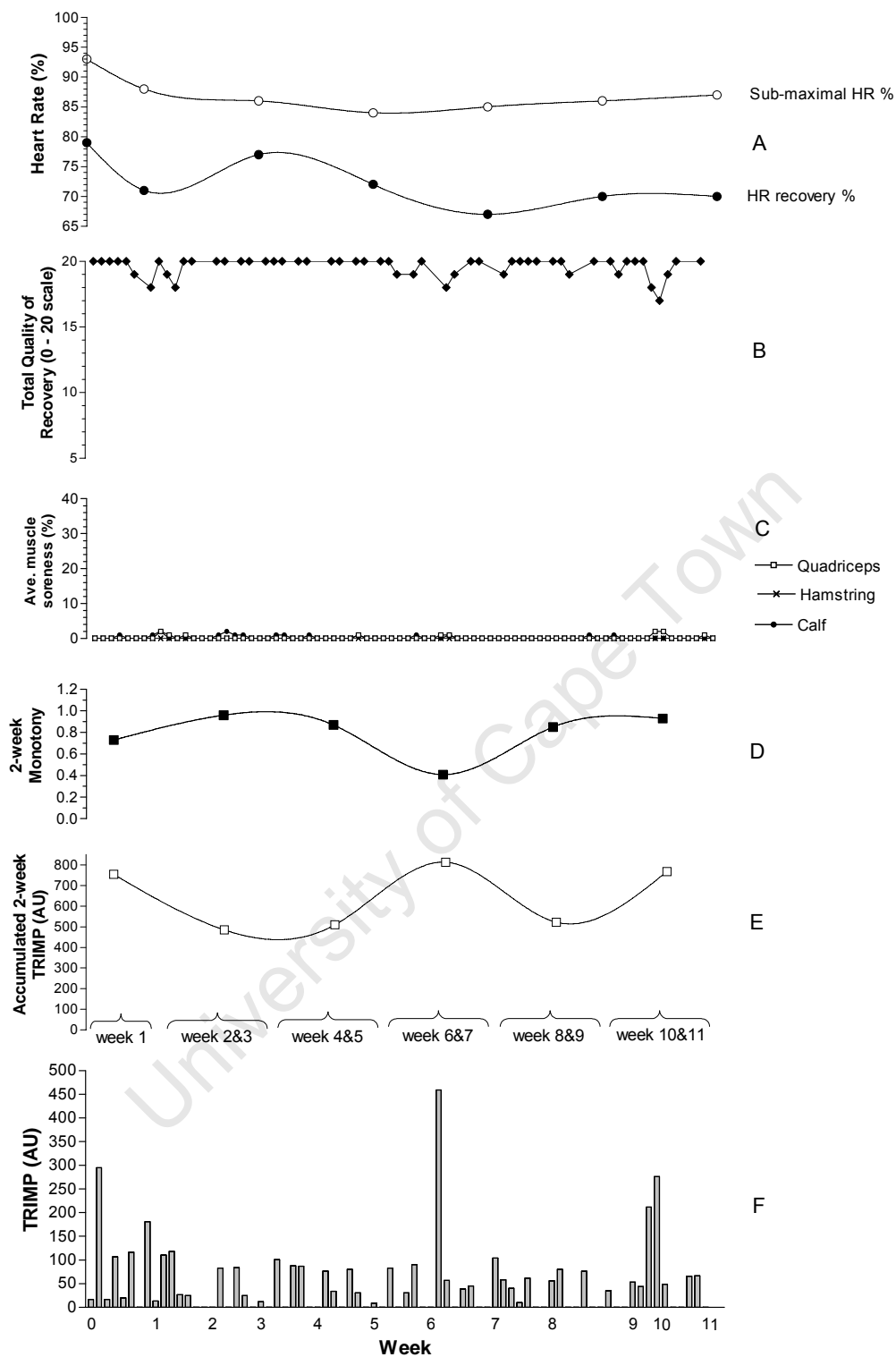


Figure 5.15: Changes in physiological and outcome measures for Participant 7 after 11 weeks of running training. A - Submaximal heart rate percent (\circ) and heart rate recovery percent (\bullet). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (\square), Hamstring (\times) and Calf (\bullet). D - Monotony accumulated over 2-week periods between HIMS tests. E - TRIMP (AU) accumulated over 2-week periods between HIMS tests. F - TRIMP (AU) for each training session.

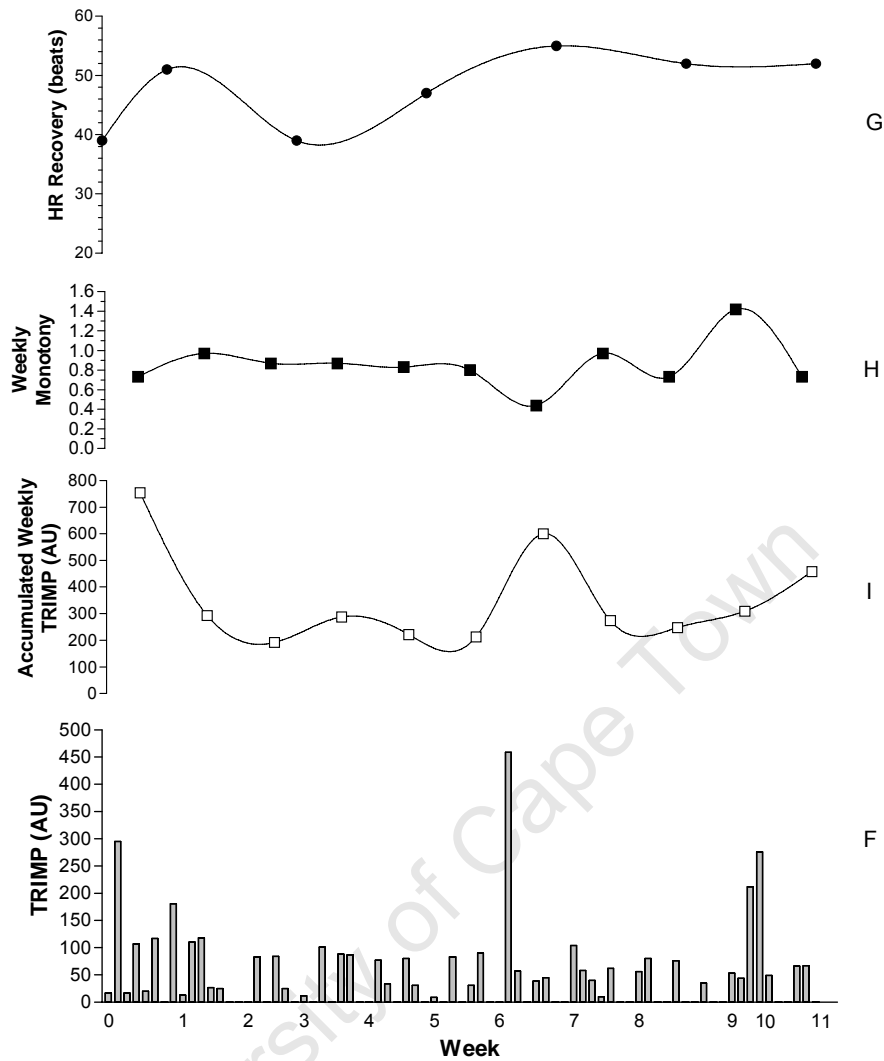


Figure 5.16: Changes in physiological and outcome measures for Participant 7 after 11 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F – TRIMP (AU) for each training session.

5.2.2.2.8 CASE STUDY 8

○ Results

Participant 8 was a 26-year old female who reported that she had not been participating in any regular exercise during the month prior to beginning the 10 km running programme. She did not report having any injuries or illnesses that affected her training during the 12 weeks of the programme. Subject characteristics and physiological changes that occurred during the study are presented in Table 5.9.

Table 5.9: Subject characteristics and physiological changes for Participant 8 after a running training programme

Participant 8	Week 0	Week 10	Absolute change
Age (yr)	26	-	-
Mass (kg)	68.1	70.2	2.1
Height (cm)	162	-	-
BMI (kg.m ⁻²)	25.9	26.7	0.8
Body fat %	35.1	35.7	0.6
Heart Rate (b.min ⁻¹)			
- Resting	65	71	6
- Average (2.4 km)	185	184	-1
- Maximal	202	-	-
2.4 km heart beats	3682	2647	-1035
2.4 km time (min)	19.9	14.4	-5.5
10 km time (h:min:s)	-	1:04:28	-
HIMS sub-max HR (b.min ⁻¹)	202	191	-11 #
HIMS HRr (beats)	37	52	15 #

Data from the HIMS test performed after week 8

The training of Participant 8 showed a well periodised structure as depicted in the graph of weekly TRIMP (Fig 5.18I). Total accumulated TRIMP over the entire study period was 2931 AU, the highest weekly TRIMP was 469 AU (Fig 5.18I) and average weekly TRIMP was 293 AU. Submaximal heart rate decreased by 11 b.min⁻¹ by the end of the programme (Table 5.9) and absolute 1-minute heart rate recovery improved by 15 beats (Table 5.9, Fig 5.18G). Data from the HIMS test performed at

the end of week 8 were used to calculate the effect of the running training programme on the indices of submaximal heart rate and heart rate recovery (Table 5.9), as the participant engaged in strenuous exercise before the final HIMS test. The participants were asked not to exercise at least 3 hours before performing the HIMS tests, as it was believed this has an effect on heart rate and heart rate recovery, as can be seen in Fig 5.17A and 5.18G, in which the final HIMS data are included. Performance improved significantly, with time decreasing by 5.5 minutes and the number of heart beats decreasing by 1035 beats during the 2.4km assessment (Table 5.9). Total Quality of Recovery tended to improve slightly over the course of the running programme (Fig 5.17B), and was never reported to be worse than “*Reasonable recovery*” on the 0-20 TQR Scale (Appendix 4). In the first 2 weeks moderate to high muscle soreness was reported, possibly indicating a slow initial response to the training stimulus (Fig 5.17C). After the second week muscle soreness generally remained below 10 on the analogue scale of 0-100 (Appendix 6). Two distinct peaks in muscle soreness were reported in weeks 4 and 7, which do not appear to be related to any specific intervention in the training programme. Although not reported, the possibility of strenuous recreational activity causing these increases in muscle soreness cannot be ruled out.

- o *Conclusion*

These results suggest that this participant may be a “fast responder” as she appears to have adapted optimally to the training stimulus. The pattern of changes in weekly Monotony (Fig 5.18H) appears to be inversely related to the pattern of changes in weekly TRIMP (Fig 5.18I). As such, when weekly TRIMP, increased weekly Monotony decreased and vice versa. The implications of this inverse relationship cannot be established with the available data.

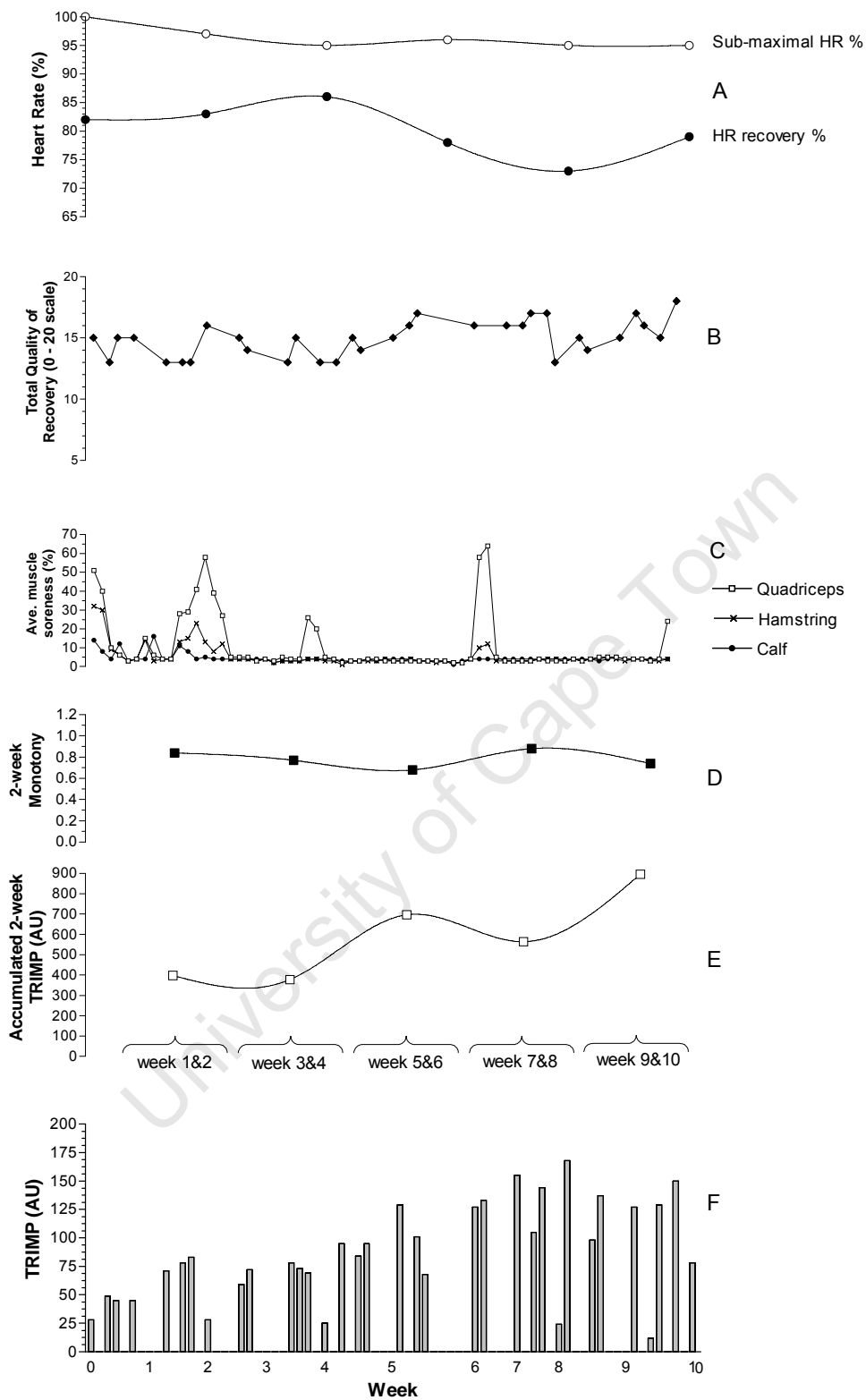


Figure 5.17: Changes in physiological and outcome measures for Participant 8 after 10 weeks of running training. A - Submaximal heart rate percent (○) and heart rate recovery percent (●). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (□), Hamstring (x) and Calf (●). D - Monotony accumulated over 2-week periods between HIMS tests. E - TRIMP (AU) accumulated over 2-week periods between HIMS tests. F - TRIMP (AU) for each training session.

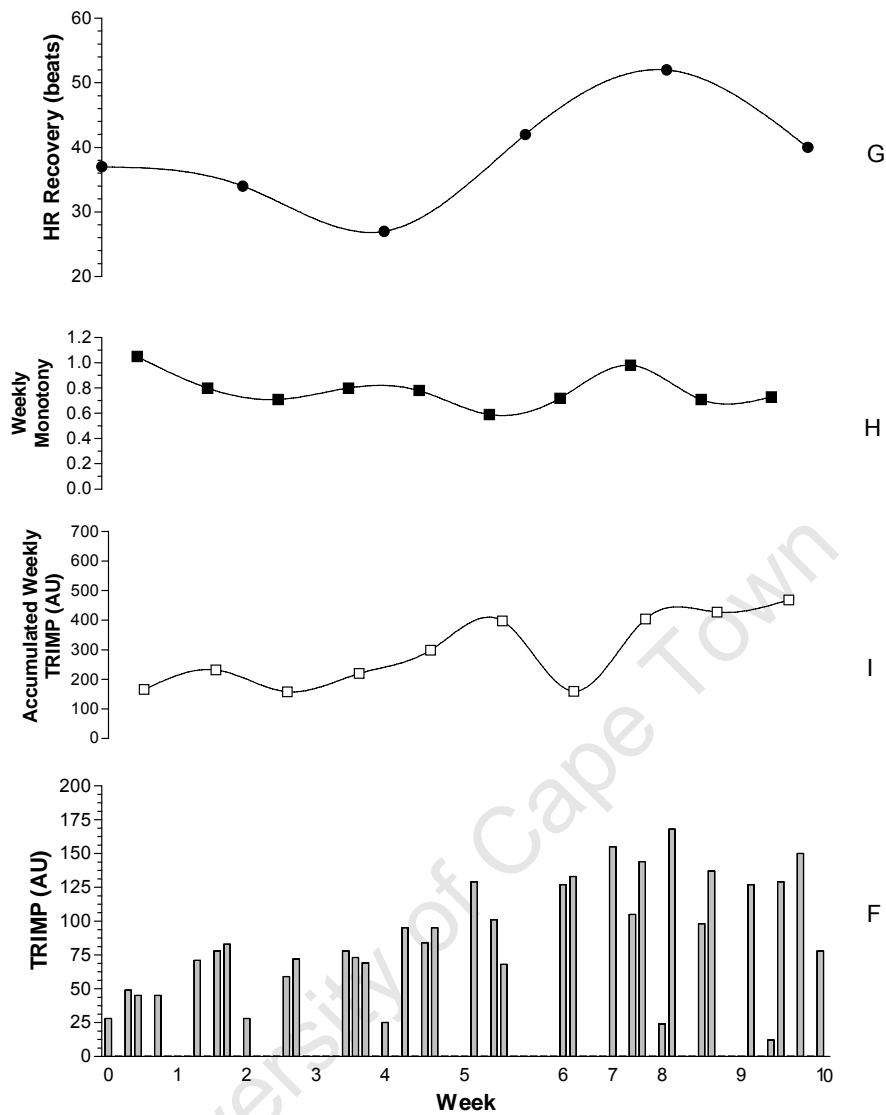


Figure 5.18: Changes in physiological and outcome measures for Participant 8 after 10 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F - TRIMP (AU) for each training session.

5.2.2.2.9 CASE STUDY 9

o Results

Participant 9 was a 21-year old female student who reported that although she usually attends the gym regularly, she had been ill during the month prior to the start of the 10 km running programme, and thus had not been able to do any exercise during that time. She did not report having any injuries or illnesses during the 12 weeks of the programme. Four months after completing this 10 km running programme, participant 9 ran a 21km (half marathon) race in a time of 2:38:58 (h:min:s). Subject characteristics and physiological changes that occurred during the study are presented in Table 5.10.

Table 5.10: Subject characteristics and physiological changes for Participant 9 after a running training programme

Participant 9	Week 0	Week 11	Absolute change
Age (yr)	21	-	-
Mass (kg)	67.6	-	-
Height (cm)	165	-	-
BMI (kg.m ⁻²)	24.8	-	-
Body fat %	29	-	-
Heart Rate (b.min ⁻¹)			
- Resting	76	76	0
- Average (2.4 km)	147	158	11
- Maximal	199	-	-
2.4 km heart beats	2597	2712	115
2.4 km time (min)	17.7	17.2	-0.5
10 km time (h:min:s)	-	1:06:15	-
HIMS sub-max HR (b.min ⁻¹)	191	183	-8
HIMS HRr (beats)	15	58	43

Total accumulated TRIMP over the study period was 3342 AU, the highest weekly TRIMP was 432 AU (Fig 5.20I) and average weekly TRIMP was 304 AU. Submaximal heart rate decreased by 8 b.min⁻¹ by the end of the programme (Table 5.10) and absolute 1-minute heart rate recovery improved by 43 beats (Table 5.10,

Fig 5.20G). Performance however did not improve, with the number of heart beats during the 2.4 km assessment increasing by 115 beats (Table 5.10). This can be explained by an increase in average heart rate and a slight decrease in time (Table 5.10). The initial increase in training at the beginning of the programme was followed closely by an improvement in heart rate recovery of 14 beats (Fig 5.20G). Over the next 2 weeks weekly training load decreased slightly and then stayed the same for the following 6 weeks (Fig 5.20I). Over the same period heart rate recovery reverted back to baseline levels. In week 9 training load was increased again (Fig 5.20I), and heart rate recovery appeared to respond favourably again (Fig 5.20G). The pattern of changes in weekly Monotony (Fig 5.20H) is very similar to the pattern of changes in weekly TRIMP (Fig 5.20I), that is when weekly TRIMP increased so too did weekly Monotony, and vice versa. Muscle soreness generally remained below 10 on the analogue scale of 0-100 (Appendix 6), suggesting that few signs of muscular discomfort were experienced during the programme (Fig 5.19C). On one occasion, in week 3, muscle soreness increased slightly above 10, but this score does not appear to be related to any specific intervention in the training programme. Data from assessments of Total Quality of Recovery (Fig 5.19B) may have contributed to a more comprehensive understanding of this participant's daily recovery from training bouts however these data were not received from this participant.

- *Conclusion*

These results suggest that from a physiological perspective participant 9 responds quickly and significantly to changes in training load. She may also have a high threshold of training load above which her body is stimulated to adapt, as she engaged in relatively high weekly training loads compared to the other participants, with few adverse effects. By the end of the study heart rate recovery had improved dramatically, but performance was not improved. The improvement in submaximal heart rate and heart rate recovery suggests that there are health benefits to participating in the running programme. A possible explanation for the decrease in performance despite positive physiological changes is that the participant may still have been in an "adaptation phase" of training, where changes in physiology had not yet been translated into improvements in performance. Alternatively, external factors such as work stress or personal issues, not reported in this study, may have influenced her performance in the 2.4 km assessment after the training programme.

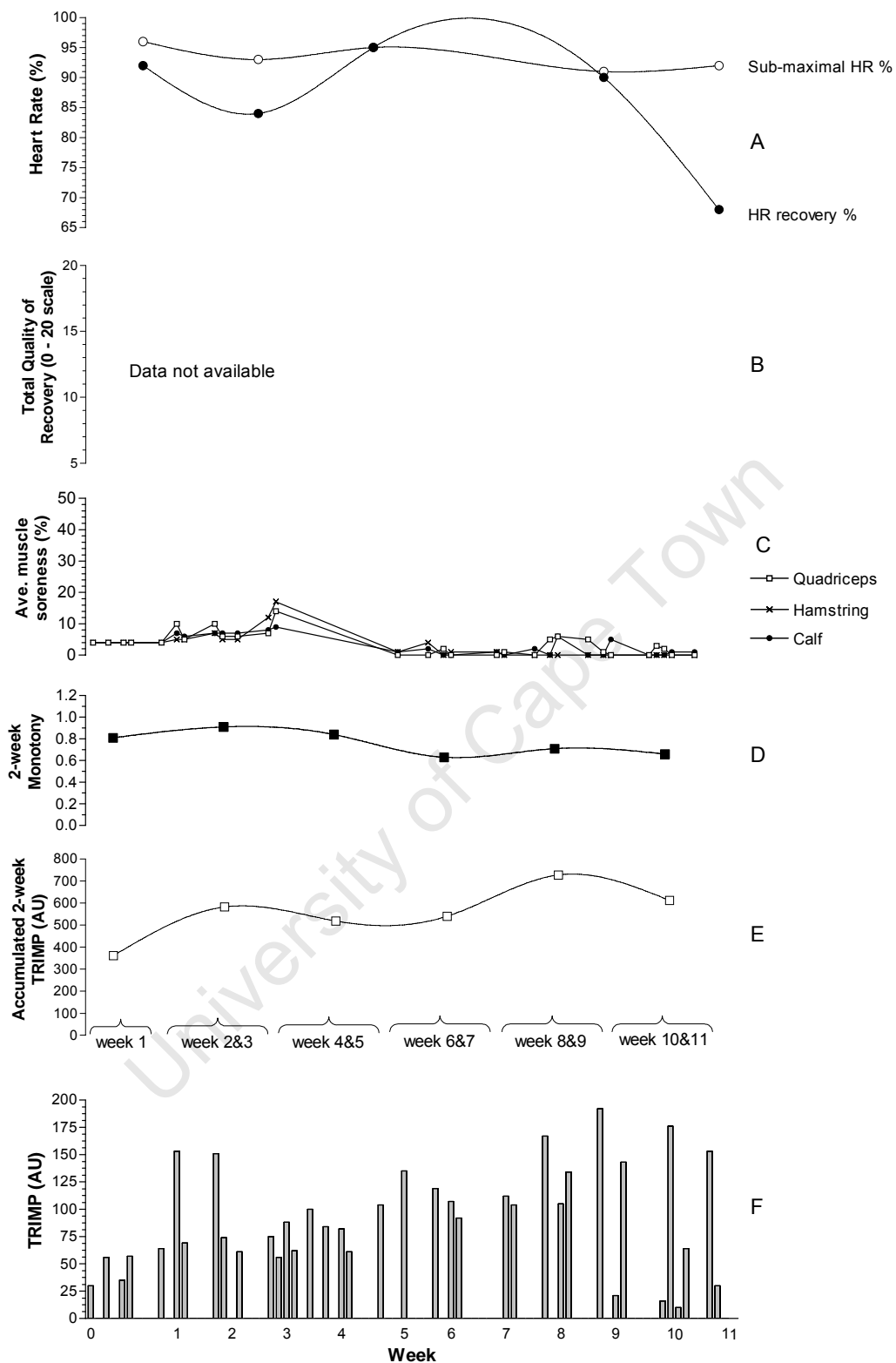


Figure 5.19: Changes in physiological and outcome measures for Participant 9 after 11 weeks of running training. A - Submaximal heart rate percent (○) and heart rate recovery percent (●). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (□), Hamstring (x) and Calf (●). D - Monotony accumulated over 2-week periods between HIMS tests. E - TRIMP (AU) accumulated over 2-week periods between HIMS tests. F - TRIMP (AU) for each training session.

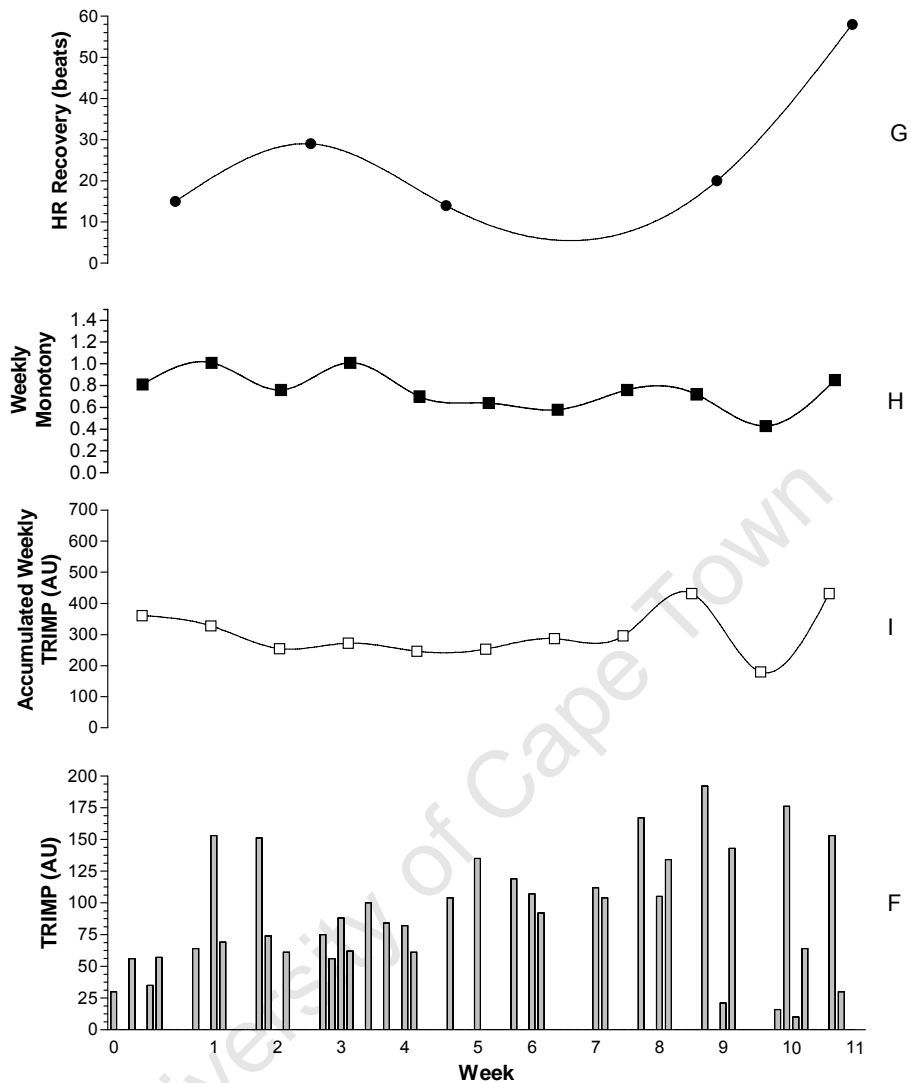


Figure 5.20: Changes in physiological and outcome measures for Participant 9 after 11 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony, I - Weekly TRIMP (AU). F - TRIMP (AU) for each training session.

5.2.2.2.10 CASE STUDY 10

○ Results

Participant 10 was a 45-year old female who indicated that she had been doing three 10 to 20-minute bouts of cycling, three 40-minute walks and two 10 to 20-minute runs per week during the month before beginning the 10 km running programme. During week 1 of the programme she was prescribed an antibiotic and painkiller to treat an infected elbow and this prevented her from training for 2 days. Subject characteristics and physiological changes that occurred during the study are presented in Table 5.11.

Table 5.11: Subject characteristics and physiological changes for Participant 10 after a running training programme

Participant 10	Week 0	Week 11	Absolute change
Age (yr)	45	-	-
Mass (kg)	69.2	67.2	-2.0
Height (cm)	163	-	-
BMI (kg.m ⁻²)	26.0	25.3	-0.8
Body fat %	38.8	37.5	-1.3
Heart Rate (b.min ⁻¹)			
- Resting	72	73	1
- Average (2.4 km)	186	174	-12
- Maximal	195	-	-
2.4 km heart beats	3075	2584	-491
2.4 km time (min)	16.5	14.9	-1.7
10 km time (h:min:s)	-	1:03:39	-
HIMS sub-max HR (b.min ⁻¹)	187	182	-5
HIMS HRr (beats)	31	36	5

Total accumulated TRIMP over the entire study period was 3341 AU, the highest weekly TRIMP was 614 AU (Fig 5.22I) and average weekly TRIMP was 304 AU. Submaximal heart rate decreased by 5 b.min⁻¹ by the end of the programme (Table 5.11) and absolute 1-minute heart rate recovery only improved by 5 beats (Table

5.11, Fig 5.22G). During the first half of the programme heart rate recovery became more delayed, but then began improving again in the second half of the programme. Performance improved significantly, with the number of heart beats during the 2.4 km assessment decreasing by 491 beats (Table 5.11). Total Quality of Recovery tended to decrease over the course of the running programme (Fig 5.21B) and moderate to high muscle soreness was reported after week 5 particularly in the quadriceps and hamstrings (Fig 5.21C). The pattern of changes in weekly Monotony (Fig 5.22H) is very similar to the pattern of changes in weekly TRIMP (Fig 5.22I), that is when weekly TRIMP increased so too did weekly Monotony, and vice versa.

- *Conclusion*

This participant had progressively decreasing recovery scores and increases in muscle soreness, particularly in the second half of the programme. Her performance improved and there was a tendency for a decrease in submaximal heart rate and an improvement in heart rate recovery in the latter stages of the programme. This suggests that this participant may have responded slower to the training stimulus compared to other subjects in the study, but nevertheless adapted well to the training programme. It seems that she may also have a higher threshold of training load as positive improvements in heart rate recovery became evident in the second half of the programme when training loads increased substantially. Participant 10 accumulated one of the highest total and average weekly training loads in the group, and had the highest weekly TRIMP. The results of participant 10 highlight the necessity of a holistic view when monitoring training. An examination of the Total Quality of Recovery and muscle soreness in isolation, particularly in the second half of the programme may have led to the conclusion that the participant was not adapting well to the intensity of training. However, this may have just been a period of overreaching which stimulated adaptation, and which appeared to translate later into a positive shift in submaximal heart rate and heart rate recovery and significant improvements in performance. This shows that performance along with other physiological measures such as heart rate, muscle soreness and recovery scores for example all need to be considered within the context of optimising an athlete's adaptation to training.

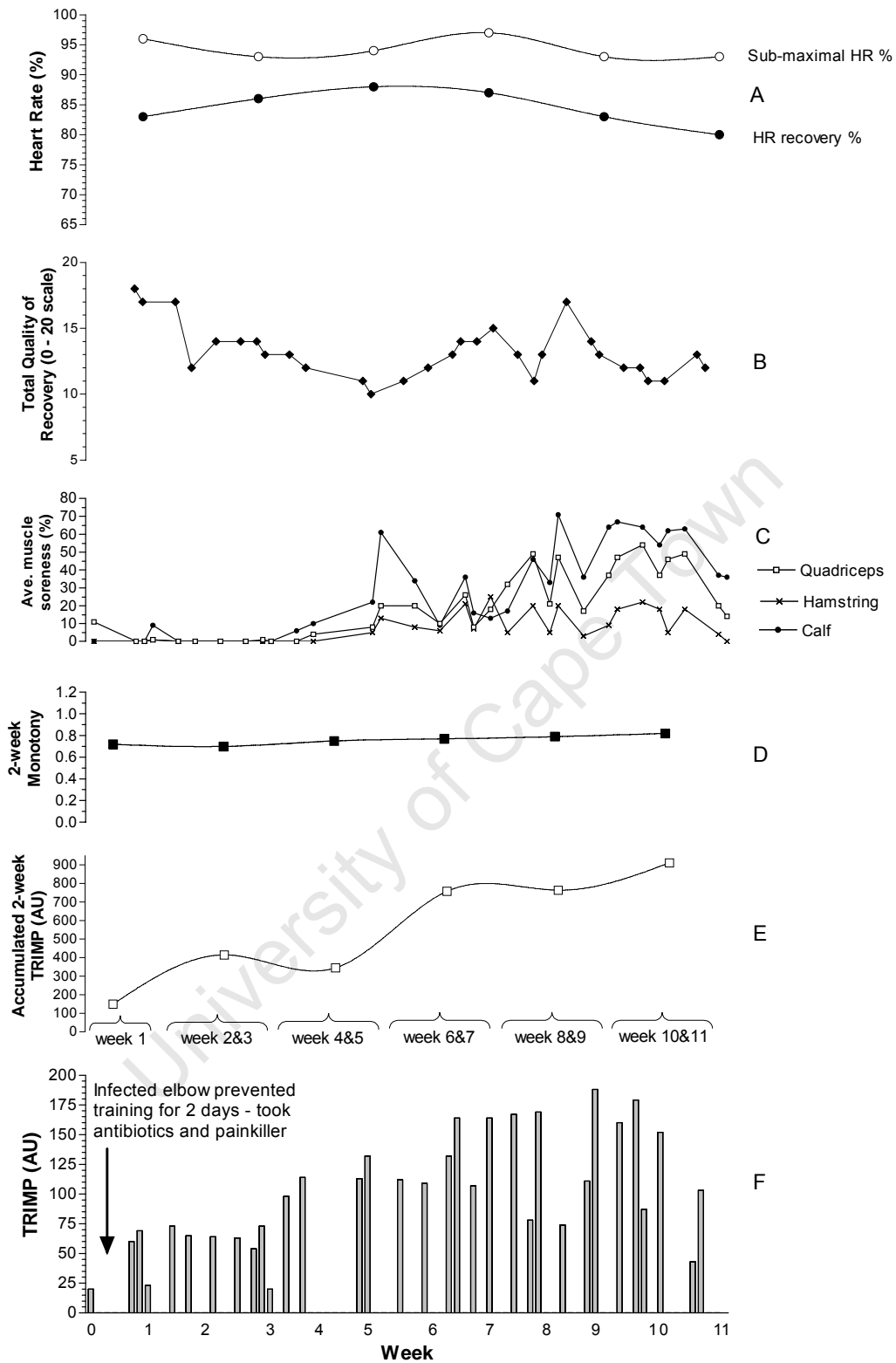


Figure 5.21: Changes in physiological and outcome measures for Participant 10 after 11 weeks of running training. A - Submaximal heart rate percent (○) and heart rate recovery percent (●). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (□), Hamstring (x) and Calf (●). D - Monotony accumulated over 2-week periods between HIMS tests. E - TRIMP (AU) accumulated over 2-week periods between HIMS tests. F - TRIMP (AU) for each training session.

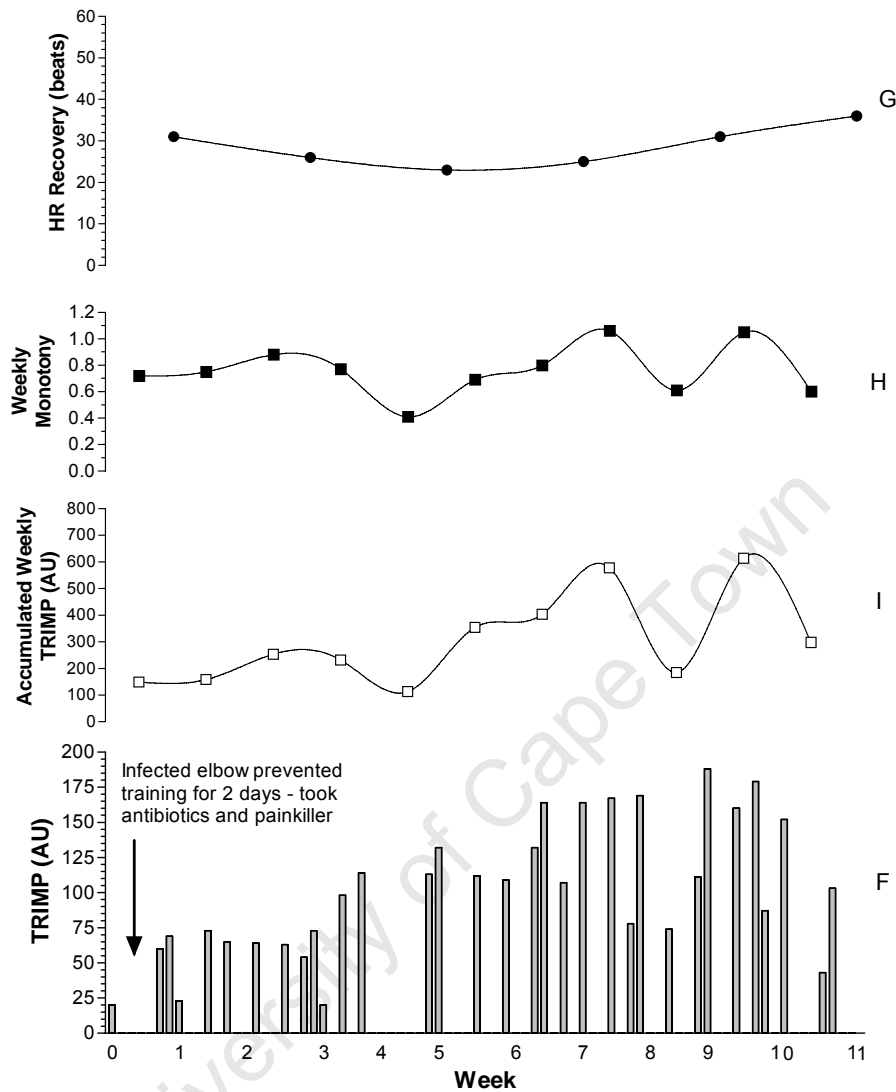


Figure 5.22: Changes in physiological and outcome measures for Participant 10 after 11 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F - TRIMP (AU) for each training session.

5.2.3 DISCUSSION

5.2.3.1 Group Results

5.2.3.1.1 Physiology and performance

Twelve weeks of endurance running training had a significant effect on selected performance and physiological variables in ten sedentary ladies, including 2.4 km time and the number of heart beats during the 2.4 km assessment. In addition, the health benefits of the programme are evident from the improvement in 1-minute heart rate recovery and reduction in submaximal heart rate. These findings are similar to the results of Sugawara et al (2001) who reported that eight weeks of training in previously untrained men improved 30-second heart rate recovery¹⁹⁶. Many other studies have found similar decreases in heart rate at the same submaximal intensity after endurance training. The explanation for this has been attributed to a decrease in sympathetic activity to the heart^{83;102;129;141-144}.

The improvements in heart rate recovery observed in the current study support previous findings, including cross-sectional studies showing that endurance trained athletes have a faster heart rate recovery after the cessation of exercise compared to sedentary subjects^{143;174;194}. Longitudinal studies have also shown accelerated heart rate recovery after 6 and 8 weeks of moderate intensity endurance training^{196;197}. An association has been found between a delayed heart rate recovery and subsequent mortality^{172;179} in patients with cardiovascular disease^{180;181}. A proposed mechanism to explain this relationship suggests that the protective effect that parasympathetic tone affords the cardiovascular system is diminished during delayed recovery¹⁷⁵. Therefore an improvement in heart rate recovery appears to have distinct health benefits.

In the current study resting heart rate did not change significantly after 12 weeks of running training. Many studies have reported slight decreases in resting heart rate after endurance training¹²⁷⁻¹³³. Melanson and Freedson (2001) however also found no difference in the resting heart rate of previously sedentary individuals after 16 weeks of training¹³⁶. In the study by Melanson and Freedson (2001) participants trained 3 times a week, 30 minutes per session and only increased the intensity of

exercise from 70%HRreserve in the first 2 weeks to 80%HRreserve from week 3¹³⁶. The difference in results may be explained by the low intensity of training in this study¹³⁶ and in the current study, compared to the studies in which slight decreases in resting heart rate were observed.

5.2.3.1.2 Relationship between TRIMP and heart rate recovery

In the current study a non-significant correlation ($r = -0.15$) was found between TRIMP and heart rate recovery. This is in contrast to the correlation of $r = -0.61$ ($p < 0.05$) that was found between TRIMP and heart rate recovery in Chapter 4. The difference in these results may be due to the influence of the systematic changes in training load that occurred in the current study. In Chapter 4 only data from participants that kept their training load the same over 2 weeks was used in the analysis, whereas the analysis in the current study also included participants with increases and decreases in training load. Changes in training load such as these cause disturbances in homeostasis which would be reflected in the autonomic nervous system and measured as changes in heart rate recovery. This would then alter the training load-HRr relationship that was evident when training load was kept constant (Chapter 4). Results from this series of case studies compared to results in Chapter 4 suggest that the relationship between training load and heart rate recovery may be affected by the phase of training, as defined by the General Adaptation Syndrome, that the athlete is currently in. For example, it may be argued that the subjects in Chapter 4 were in the Resistance Stage of the General Adaptation Syndrome, whereas the subjects in the current study were in the Alarm Reaction Stage.

When data were divided into groups based on whether TRIMP increased, decreased or remained constant between successive 2-week periods, the subsequent mean percent changes in heart rate recovery were also not different. This is in contrast to the trends found in Chapter 4 towards improvements in heart rate recovery with decreases in weekly training load and decreases in heart rate recovery with increases in training load. Differing results in this case may be the consequence of different periods of time being used in which to calculate training load. The 2-week period used in this study may be too long to be regarded as an “acute” stress, particularly since the participants were in a more dynamic physiological state (Alarm

Reaction Stage) compared to participants in Chapter 4 (Resistance Stage). The 1-week period used in Chapter 4 might better reflect the acute response of heart rate recovery to changes in training load. These suggestions require further investigation.

5.2.3.2 Individual Results

5.2.3.2.1 Physiological and performance changes with training

It was surprising to see that some participants did not improve their 2.4 km time after the completion of the running training programme. On closer inspection of the data an interesting phenomenon was observed. Of those participants that did not improve their time, most had a decrease in average heart rate during the 2.4 km assessment. This could be explained by them possibly being conservative about “pushing themselves” to give a maximal effort. Most of the participants had a sedentary background and were therefore not familiar with high intensity exercise. (This supports the decision not to ask the participants to perform a maximal HR or VO_2 test, as many would probably not have given a maximal effort.) The total number of heart beats during the 2.4 km assessment was therefore also used to assess physiological performance. Based on this outcome measure, 7 of the 10 participants improved their 2.4 km performance after the running programme. Of these seven, 5 also showed a decrease in submaximal heart rate and an improvement in heart rate recovery. Performance data were missing for one additional participant, but she also showed a decrease in submaximal heart rate and an improvement in heart rate recovery after training. The other 2 participants that improved performance either did not show a decrease in submaximal heart rate or they did not have an improvement in heart rate recovery. The 2 participants that did not improve performance both showed decreases in submaximal heart rate as well as improvements in heart rate recovery. This shows an inter-individual variability in the way individuals respond to exercise training. This needs to be considered in developing a quantifiable relationship between training, physiological adaptations and performance, and also has implications for exercise prescription.

5.2.3.2.2 Individual response to training

It may be argued from a biological perspective that the purpose of exercise training is to expose the body to a physiological stress with the ultimate goal of inducing adaptation. However, if the stress is not sufficient it is unlikely that adaptation will occur¹¹. In the current study there was a broad range of average weekly TRIMP, highest weekly TRIMP and total accumulated TRIMP among the participants, and yet despite the disparities most participants still improved their performance. Some participants appeared to respond well to relatively low training loads (Participants 3, 5 and 6 for example) where others responded well to much higher training loads (Participants 2, 7, 8 and 10). The small improvements in performance and physiological measures in Participant 1 suggest that she was training below her training load threshold. The steady-state exercise intensity that elicits a lactate concentration of approximately 4 mmol.L⁻¹ has been suggested to be the most favourable for inducing optimal physiological adaptations for endurance events¹⁹. As such, this is probably the first physiological measure that has been proposed to be used in the identification of a training intensity “threshold”. Weltman et al (1992) found that training at or above the lactate threshold resulted in similar improvements in VO₂, and velocity at the lactate threshold and at fixed blood lactate concentrations of 2.0 and 2.5 mM during the first four months of training in untrained women²¹⁶. Stegmann et al (1981) however, warn that this “optimal” lactate level may range anywhere from 2.0 to 7.5 mmol.L⁻¹ among different athletes²⁰. This highlights the wide variation in the way in which individuals respond to exercise and the need to identify each individual's training load threshold when prescribing training in order to optimise their physiological adaptations.

The variance in training-induced adaptations may be due to several factors such as age, gender, training history, psychological factors²¹⁷, initial training status; mode, duration, intensity and frequency of training¹⁰⁹, recovery potential, exercise capacity, non-training stress factors and stress tolerance^{86;218}. Jones and Carter (2000) in their review of the effects of endurance training on the parameters of “aerobic fitness” note that the magnitude of change in VO₂max may be governed by many of the above factors⁸⁶. In addition exercise economy differs significantly between individuals and may be influenced by the velocities/power outputs at which they habitually train⁸⁶. Bell et al (2000) found that concurrent strength and endurance

training resulted in training adaptations that differed from those that occurred after either strength or endurance training alone, emphasizing the importance of considering exercise mode when assessing training-induced adaptations¹¹⁰. Skinner et al (2000) found that individual changes in power output and VO_2max in response to 20-weeks of cycling training varied significantly among people who trained at heart rates associated with the same $\% \text{VO}_2\text{max}$ ¹⁷. Similarly Hellard et al (2005) found that swimmers reacted differently to the same training load⁷³. Al-Ani et al (1996) reported an increase in HRV in 9 out of 11 people after 6 weeks of endurance training. However, the HRV of the two other subjects decreased after training. The fact that HRV of these two participants had an opposite response to training than the other 9 participants highlights the individuality in the training response¹⁶².

In the current study it appeared as though there were “slow responders” and “fast responders” to the running training programme. The performance of Participant 4 did not improve despite a decrease in submaximal heart rate and an improvement in heart rate recovery. This may indicate that although physiological changes were occurring, these had not yet been translated into improvements in performance. This may be indicative of the participant still being in the adaptation phase (the Reaction Phase in the General Adaptation Syndrome). Participant 10 improved performance overall but the physiological adaptations only became positive after about 6 weeks of training. Participant 1 appeared to either be a “slow responder”, or she was not pushing herself hard enough and thus did not achieve the necessary adaptations to improve performance significantly. The physiological measures of submaximal heart rate and heart rate recovery appeared to respond relatively soon after the start of the training programme in Participants 2, 3, 5, 6, 7 and 8 with a concomitant improvement in performance. It may be assumed that by the end of the training programme these participants had entered Stage 2 of the General Adaptation Syndrome, the Resistance Stage. The physiological measures of Participant 9 also appeared to respond quickly to changes in training load however these physiological changes were not translated into an improvement in performance. Participant 9 was a medical student preparing for and writing year-end exams, and although not reported, the possibility of external stresses such as these influencing her performance in the 2.4 km assessment can not be ruled out. The influence of external stresses on an athlete therefore needs to be considered when assessing how an athlete is responding to training.

Hellard et al (2005) suggested that performance may be affected by the phase of training in which the athlete is, as they found differing short-term, intermediate- and long-term effects on performance⁷³. Similarly, Avalos et al (2003) found that they could separate swimmers into groups that reacted well to either a long-term, mid-term or short-term training period, emphasising the need to individualize the distribution of training loads throughout a season to facilitate optimal adaptation in each athlete²¹⁹. Skinner et al (2001) found that age, gender, race and initial fitness had little influence on how VO_2 max changed after a standardized endurance training programme, but that there were low, medium and high responders in both sexes, all ages and at all fitness levels²²⁰. They suggested that genetic factors may be responsible for the wide variation in individual responses²²⁰. Genetic traits may contribute substantially not only to the way in which athletes adapt to training, but also to the observed heterogeneity in performance ability between athletes²²¹. The potential for improvement in performance or optimal adaptation may also be influenced by a genetic predisposition for performance in a specific mode of exercise²²².

5.2.3.2.3 Monotony

Monotony, an index of training variability, was developed by Foster et al (1998)⁵². They suggest that high training loads and high training Monotony may both be related to negative adaptations to training⁵². Monotony was therefore calculated in the current study to provide more information about the response of each individual to the training loads. Foster et al (1998) studied the relationship between Monotony and banal illness and found that 77% of illnesses reported were associated with a preceding spike in Monotony⁵². In the case studies presented in this chapter there were some incidences when an increase in Monotony (i.e. less variation in training load) was followed by reported illness, however in other incidences when (a) illness was reported, there was no preceding spike in Monotony, and (b) a spike in Monotony was not followed by a report of illness. Foster et al (1998) suggest the existence of an individual threshold (of Monotony for example) above which the risk of becoming ill is increased⁵². Participants 3, 4 and 5 may have exceeded their individual weekly Monotony thresholds which contributed (amongst other factors) to the development of illness approximately a week later. The other participants did not

report illness during the training programme, yet spikes in their weekly training Monotony are evident. This may indicate that their individual thresholds (as suggested by Foster et al (1998)) were not exceeded.

In the current study the relationship between changes in TRIMP and changes in Monotony over the period of the programme seemed to be very similar. That is, when weekly TRIMP increased there was also an increase in weekly Monotony, and vice versa. The reason for this is unclear. Only in Participants 7 and 8 did this relationship appear to be inverse, so that when weekly TRIMP increased there was a decrease in weekly Monotony (i.e. training became more varied) and vice versa. What is unique about these 2 participants is not apparent. Monotony is an interesting and relevant factor to monitor in highly trained athletes that are frequently training at high loads and in whom small changes in training and recovery could have important consequences. In the population investigated in this study, however, Monotony may not be as applicable since the frequency of training per week was much less and the training loads much smaller. One also needs to acknowledge that there are many other factors that could cause illness besides Monotony of training, and these need to be considered and monitored.

5.3 IRONMAN

5.3.1 METHODS

5.3.1.1 Participant

In April 2006 a 28-year old male approached the MRC/UCT Research Unit for Exercise Science and Sports Medicine, Department Human Biology to enquire about having his training progress monitored as he prepared for his first Ironman Triathlon in March 2007. He was training under the guidance (via e-mail communication) of a trainer of his choice in Australia. For the following 32 weeks (April 2006 – December 2006) the participant made his training information available on a daily basis. At the beginning of December 2006, his trainer suggested he no longer train with a heart rate monitor nor analyse his training in as much detail as he had been doing over the previous 8 months. For this reason the collection of his training data was terminated. He continued with his supervised training and successfully completed his first Ironman Triathlon in Port Elizabeth, South Africa in March 2007 in a time of 15 hours 26 minutes 33 seconds.

5.3.1.2 Methodology

5.3.1.2.1 Body composition and physiological measurements

Anthropometric measurements were recorded before, and periodically during 32 weeks of his training. Body mass was recorded on a calibrated scale (Seca model 708 Germany) and recorded to the nearest 100 g. His stature was recorded to the nearest mm using a stadiometer (Seca model 708 Germany). Body fat percent was assessed with a near infrared (NIR) measurement on the right bicep using the Futrex-6100A/ZL (Kett Electric Laboratory, Futrex Inc. Gaithersburg, MD USA). Resting heart rate was recorded immediately after waking in the morning with a Suunto T6 heart rate monitor (Suunto, OY, Finland) and reported five times during the monitoring period. An incremental exercise test to volitional exhaustion was conducted on a motor driven treadmill before the monitoring period began to

determine the subject's maximal oxygen uptake ($VO_2\text{max}$), peak treadmill running speed (PTRS) and maximum heart rate (HRmax) as described in Chapter 2.

5.3.1.2.2 Ironman training programme

The monitoring of the participant's training towards the 2007 Ironman began in April 2006. A brief inspection of the weekly TRIMP graph (Fig 5.23E) revealed that the coach attempted to follow a pattern of prescribing alternating weeks of heavy training and light training.

5.3.1.2.3 Daily records

The participant completed a training diary each day which detailed each training session in terms of the type (mode) of exercise, the duration thereof and a rating of perceived exertion (on the Session RPE Scale, Appendix 3) for each exercise session. The Session RPE was reported 30 minutes after every training session by giving an overall rating of how difficult the whole workout was perceived to be, irrespective of whether continuous or interval training was performed. A subjective assessment of muscle soreness was reported daily, at approximately the same time of day, using an analogue scale created in Microsoft Access (Appendix 6) as described in Daily Records in section 5.2.1.2.3. An Injury/Illness Report was provided for the participant to complete in the event of him sustaining an injury or becoming ill (Appendix 9). A heart rate monitor was worn during all training sessions to record training heart rate and duration of the training session. Unfortunately, it was not possible to measure heart rate during the swimming sessions accurately. This may have been due to the heart rate monitor not being water proof, or due to inconsistent contact between the monitor and the body in the horizontal swimming position.

5.3.1.2.4 HIMS Tests

A HIMS test, as described in Chapter 4, was scheduled to be performed every 2 weeks at approximately the same time of day, in order to eliminate the possible influence of diurnal changes on heart rate. The participant was asked not to consume any caffeine, nor to exercise strenuously within 3 hours before doing the

test. Heart rate was recorded at 2-second intervals during the HIMS tests and for 2 minutes after the cessation of running the fourth stage of the test.

5.3.1.2.5 Outcomes

Training load for each exercise session was quantified using TRIMP (described in Chapter 3). Session training loads were summated to calculate total training load for each week. In order to calculate TRIMP for each of the swimming sessions, heart rate was estimated using the RPE reported during the swim session and the heart rate elicited at a similar RPE during other training sessions. Although this was an estimate we felt it would provide a truer reflection of TRIMP than would be calculated if the swimming data was omitted completely. Heart rate recovery (HRr%) was assessed by expressing the heart rate 1 minute after the fourth running stage of the HIMS test as a percentage of the heart rate attained at the end of the fourth running stage of the test, as described in Chapter 4. Submaximal heart rate (the heart rate reached at the end of the fourth running stage of the HIMS test) was expressed as a percentage of maximal heart rate (HRS4%), as described in Chapter 4.

5.3.2 RESULTS

The participant was a 28-year old male who had aspirations to compete in his first Ironman Triathlon in March 2007. In April 2006, when monitoring began, he reported that he had been participating in approximately one 30-minute run, a 60 to 90-minute run, two 45 to 60-minute runs (or one of these runs being substituted with a cycle of the same duration), a 150 to 240-minute cycle, a 45 to 60-minute swim and a 75-minute swim each week. For the following 32 weeks (April 2006 – December 2006) the participant completed a training diary and assessment of muscle soreness, and recorded his training heart rate daily. During weeks 21 and 22 the participant reported having bronchitis which prevented him from training for 2 weeks. During week 26 he again reported having bronchitis, this time he was unable to train for 9 days. At this time he started using inhalers - Beclate daily, and Ventolin once or twice during training when his chest felt tight. Subject characteristics and physiological changes for the Ironman Participant after 32 weeks of prescribed training are presented in Table 5.12.

Table 5.12: Subject characteristics and physiological changes for the IronMan Participant after 32 weeks of prescribed training

Ironman Participant	Week 0	Week 32	Absolute change
Age (yr)	28	-	-
Mass (kg)	93.5	87.8	-5.7
Height (cm)	194	194	-
BMI (kg.m ⁻²)	24.8	23.3	-1.5
Body fat %	18.8	14.6	- 4.2
VO ₂ max (ml.min ⁻¹ .kg ⁻¹)	48.9	-	-
Peak treadmill running speed (km.h ⁻¹)	14.5	-	-
Heart Rate (b.min ⁻¹)			
- Resting	62	56	-6
- Maximal	190	-	-
HIMS sub-max HR (b.min ⁻¹)	185	180	-5
HIMS HRr (beats)	37	64	27
Ironman total time (h:min:s)	-	15:26:33	-
- swim time	-	01:34:36	-
- cycle time	-	07:20:28	-
- run time	-	06:04:54	-
- Overall position=1224/1308, category pos (male 25-29yrs)=130/132			

Average weekly TRIMP was 473 AU and the highest weekly TRIMP recorded was 957 AU (Fig 5.23E). Submaximal heart rate decreased by 5 b.min⁻¹ by the end of the 32 weeks (Table 5.12) and absolute 1-minute heart rate recovery improved by 27 beats (Table 5.12 and Fig 5.23B). No performance data are available as periodic performance tests were not planned in his training programme. However it is evident that significant physiological adaptations occurred over the training period. Muscle soreness (Fig 5.23C) remained on average below 20 on the analogue scale of 0-100 (Appendix 6), suggesting that relatively few signs of discomfort were experienced despite the high training loads. Two peaks in muscle soreness above 20 occurred during week 1 and week 12. This could have been the result of an increase in training load in a preceding training session, however many other increases in individual sessions were not followed by increases in muscle soreness. So the exact cause of these two peaks in muscle soreness is unclear and the possibility of

strenuous recreational activity causing these increases in muscle soreness can not be ruled out.

Submaximal heart rate and heart rate recovery did not respond immediately to changes in training load, and in fact did not change much in the first 23 weeks of training (Fig 5.23A). This may suggest that the athlete may be a slow responder to training stimuli, and may still have been in an adaptation phase of training. Alternatively, since the athlete was not accustomed to such high intensity training, he may have been going through an overreaching phase in which it appeared that training was having a negative effect (or at least not having a positive effect) on physiological systems. The low muscle soreness reports however, suggest that the participant was not training above an individual training load upper limit that might induce soreness and sub-optimal recovery. In addition the training programme appears well periodised with adequate recovery periods to balance the intense training sessions. Weekly Monotony is depicted in Figure 5.23D and shows that training during most weeks was varied, as represented by lower values of the Monotony ratio. The pattern of changes in weekly Monotony appears to be similar to the pattern of changes in weekly TRIMP (Fig 5.23E). As such, when weekly TRIMP increased so too did weekly Monotony, and vice versa. The implications and causes of this are unclear.

Between week 23 and 28 there was a significant change in heart rate recovery (Fig 5.23A and B). The exact cause of this improvement is unclear, but could be due in part to the rest period the athlete was forced to take due to having bronchitis. The athlete also started using an inhaler on a daily basis from week 26 however the exact mechanism of how the use of an inhaler could improve heart rate recovery requires further investigation.

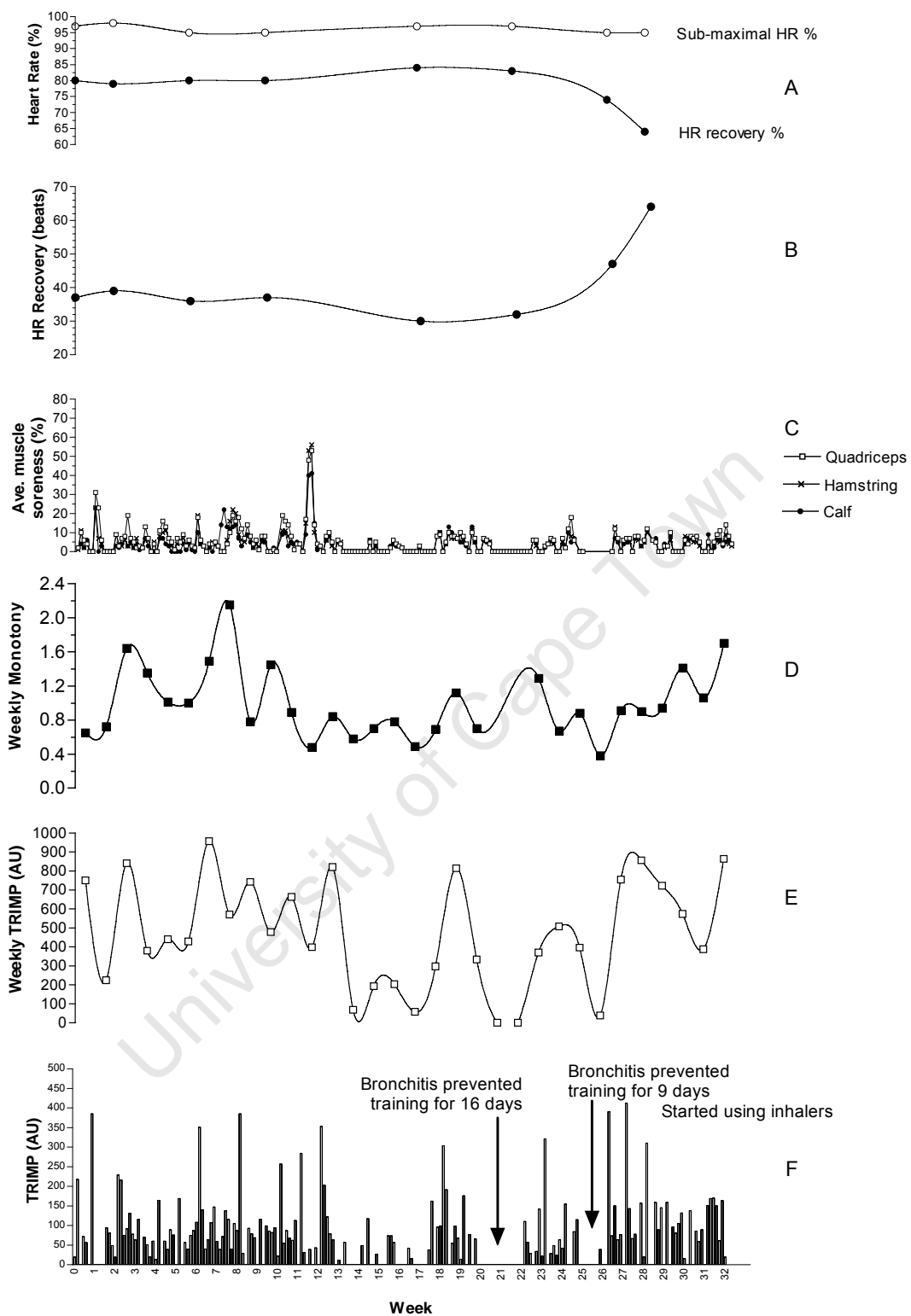


Figure 5.23: Changes in physiological and outcome measures for the Ironman Participant after 32 weeks of triathlon training. A - Submaximal heart rate percent (\circ) and heart rate recovery percent (\bullet). B - Absolute heart rate recovery (beats). C - Average muscle soreness (%) in Quadriceps (\square), Hamstring (\times) and Calf (\bullet). D - Weekly Monotony. E - Weekly TRIMP (AU). F - TRIMP (AU) for each training session.

5.3.3 DISCUSSION

Over the 32 weeks in which the participant's physiological responses to training were monitored, submaximal heart rate decreased by only 5 $\text{b}\cdot\text{min}^{-1}$. This change however is within the expected day-to-day variation in submaximal heart rate²¹¹. Heart rate recovery did not respond immediately to changes in training load, and only showed improvements after 23 weeks of training. In an attempt to explain the dramatic improvement in heart rate recovery, two possibilities were considered. The concept of "supercompensation" describes an improvement in exercise performance that occurs after a recovery period that was preceded by a fatigue-inducing training stimulus of appropriate intensity (i.e. overreaching)²²³. During the recovery period adaptations occur that result in higher levels of functional capacity²²³, however the physiological mechanisms underlying this improvement are not well understood²¹⁵. This phenomenon usually occurs over a period of a few weeks and is often used by athletes before sporting events (i.e. tapering) to elicit maximal performances at the events. A "supercompensation" effect on heart rate recovery following a taper period has not been comprehensively researched. Results from Chapter 4 in this thesis showed a tendency for heart rate recovery to improve slightly after a group of athletes had decreased their training load by 42% from one week to the next. Similarly, Brynteson and Sinning (1973) found that a small reduction in training from five 30-minute exercise sessions per week to three or four sessions per week (possibly comparable to tapering) also improved heart rate recovery slightly²¹³. The possible mechanism underlying this concept requires further investigation. Interestingly, a comprehensive review by Mujika et al (2004) on the physiological changes associated with tapering found that tapering had negligible effects on submaximal heart rate²¹⁵. This is supported by the current case study, in which there were no meaningful changes in submaximal heart rate during and after the periods of reduced training.

The possibility that the bronchodilators that the participant used may have had an effect heart rate recovery was also considered. The participant began using a corticosteroid inhaler (Beclate) daily from week 26 and a β_2 agonist (Ventolin) inhaler once or twice during training when his chest felt tight. The use of medications by inhalation is being closely monitored at sporting events to minimize any performance advantage the systemic effects of the medication may provide the athlete²²⁴. Non-

selective β_2 agonists (e.g Ventolin) have sympathetic stimulatory effects on the cardiac system, such as increasing heart rate and force of myocardial contraction, which may improve performance. Inhaled β_2 agonists also have an immediate therapeutic effect on bronchoconstriction²²⁴. However, it is unlikely that the participant's use of Ventolin only once or twice during training could have been the cause of the dramatic improvement in heart rate recovery observed in this study. There are also concerns that systemic corticosteroids may provide a competitive advantage as they have a range of varied metabolic effects²²⁴. However, the maximal beneficial effects of corticosteroid inhalation therapy such as Beclate may take several weeks to 3 months to manifest²²⁴, thus reducing the possibility that the participant's daily use of this medication in this study had an appreciable effect on heart rate recovery.

5.4 SUMMARY AND CONCLUSIONS

The case studies described in this Chapter revealed the following conclusions. In general, when the data were pooled, endurance training had a significant positive effect on selected performance variables, improved 1-minute heart rate recovery and reduced submaximal heart rate. Some participants did not improve their 2.4 km time after the completion of the running training programme. However, of those participants that did not improve their time, most had a decrease in average heart rate during the 2.4 km assessment. Some participants appeared to respond well to relatively low training loads while others responded well to much higher training loads. In the current study it also appeared as though there were "slow responders" and "fast responders" to the running training programme. This highlights the wide variability in the way individuals respond to exercise. Bagger et al (2003) described the magnitude of individual variation in a number of factors often used to assess training adaptations³⁷. This is important to distinguish whether a change in the variable is due to training or due to random biological fluctuation³⁷. Training adaptations and subsequent changes in performance are highly personal phenomena requiring the identification of each individual's training load threshold and variation in response in order to personalise training prescription and optimise physiological adaptation.

In the 10 km running training programme a weak correlation was found between TRIMP and heart rate recovery. This may be due to the influence of the systematic changes in training load that occurred with the periodised nature of the training programme. Changes in training load such as these cause disturbances in homeostasis, particularly in the autonomic nervous system¹²⁴. This disturbance may be reflected in changes in heart rate recovery, which could alter the training load-heart rate recovery relationship that was evident when training load was kept constant (Chapter 4). Results from this study, along with results from Chapter 4, suggest that the relationship between training load and heart rate recovery may be affected by the phase of training in which the person is, for example in the Alarm Reaction Stage, the Resistance Stage or the Stage of Exhaustion of the General Adaptation Syndrome. An alternative suggestion is that the relationship between changes in training load and subsequent changes in heart rate recovery needs to be established on an individual basis.

At this stage it is difficult to establish a cause-effect relationship as there is no gold standard which can be used to quantify the training response. In the context of optimising training prescription, performance is not the ultimate outcome measure. It is the individual's capacity to adapt ("adaptability" or "responsiveness") which needs to be quantified; a concept that was investigated in this thesis. If it is accepted that numerous physiological adaptations occur with chronic training, one must expect that adaptations will also occur in an individual's response to training, as this is a direct consequence of the physiological systems underlying it. The correlation between training and the changes in physiological response is highly personal and depends on many factors that influence an individual's tolerance of an exercise load. The variables measured in the current studies should thus be interpreted in context as, at this stage, there is no single variable that tells the whole story. Mujika et al (1996) suggested that individual chronic training adaptation profiles could be developed by studying individual fatigue and fitness curves in order to better understand an individual's response to a training bout⁷⁴. At this stage, until further research has investigated individual heart rate recovery responses, it is recommended that a holistic view be taken in monitoring training adaptations. This may include measures of performance and physiological and psychological changes as well as extraneous factors which might influence the rate of adaptation.

CHAPTER 6

<p>THESIS SUMMARY AND CONCLUSIONS</p>
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University of Cape Town

INTRODUCTION

This thesis investigated current methods of quantifying training load, and proposed the use of heart rate recovery to monitor the physiological response to training. A number of research questions arose during this work and have been presented and discussed in each chapter of this thesis. The next section summarises the conclusions in each chapter, answers each of these questions and discusses the implications and practical applications of these conclusions.

CHAPTER 2

Question:

Do athletes actually do what they say they do with regard to exercise training duration?

Answer:

Fifty-nine percent of participants accurately self-reported the average weekly duration of their endurance training. Less than one third (24%) over-estimated their average weekly training duration and only 17% under-estimated the duration of training they were doing. The margin of error between self-reported and actual training duration may have a significant negative impact on the accuracy of training prescription if self-reported data alone is used to quantify exercise training. In addition, since exercise duration is one of the more easily quantifiable components of training, it is reasonable to expect even greater disparity between actual and self-reported data involving other components of training such as intensity. This study therefore concludes that the quantification of an athlete's training may be inaccurate when relying exclusively on self-reported data.

CHAPTER 3Question:

How are the different methods of quantifying training related?

Answer:

Training load calculated for *ad libitum* training using the Training Impulse (TRIMP) equation correlated best with the Summated Heart Rate Zone method. This is not surprising since both methods use the direct physiological measure of heart rate as a fundamental part of the calculation. The strong correlation found between these two methods suggests that both may be suitable for the quantification of continuous as well as interval training sessions. The Session RPE method correlated well with the Summated Heart Rate Zone method, explaining approximately 71% of the variance. The relationship between the Session RPE method and TRIMP accounted for about 58% of the variance. Thus, in circumstances where heart rate monitors are not available, the subjective Session RPE method remains useful in giving reasonable assessments of training load.

Question:

What characteristics may explain the variance not accounted for in the relationship between objective and subjective methods of quantifying training load?

Answer:

Results from this study suggest that, in athletes that spend a greater percentage of their training time doing high-intensity exercise, the TRIMP and the Summated Heart Rate Zone equations may over-estimate training load compared to the Session RPE method. Conversely, in athletes that spend proportionally more of their training time doing low intensity exercise, these HR-based methods may under-estimate training load when compared to the training load calculated using the Session RPE method. An alternative interpretation is that for those athletes spending more time doing low-intensity exercise, the Session-RPE method may over-estimate training load, whereas for other athletes participating in proportionally more high intensity exercise the Session-RPE method under-estimates training load compared to the objective methods.

CHAPTER 4Question:

How is training load (TRIMP) related to heart rate recovery?

Answer:

A significant inverse relationship was found between training load calculated using TRIMP and heart rate recovery in participants that kept their training load the same over a 2-week period (i.e. the higher the training load, the faster the heart rate recovery). Interestingly, no correlation was found between $VO_2\max$ and heart rate recovery, which is surprising since both of these measures have previously been equated to “aerobic fitness”. Since heart rate recovery relates fairly well to training load, it may be postulated that heart rate recovery provides a sensitive marker of the effects of habitual training loads on autonomic nervous system function, rather than being a measure of general cardiorespiratory adaptation.

Question:

Does heart rate recovery respond to acute changes in training load?

Answer:

This study showed that heart rate recovery was slightly slower after the second week of a 2-week period in subjects that increased their training load from one week to the next. This decrease in heart rate recovery may represent the negative training response to an increase in training load (exercise stimulus). There was no change in heart rate recovery for participants who maintained their training load. Furthermore, there was a tendency for heart rate recovery to increase in the people who decreased their training load over the two weeks. Since heart rate recovery is governed by the autonomic nervous system it is likely that the changes observed in heart rate recovery may represent negative and positive responses of the autonomic nervous system to exercise stimuli.

Question:

Does submaximal heart rate reflect acute changes in training load?

Answer:

This study found no changes in the submaximal heart rate in response to either increases, decreases or the maintenance of training load over a 2-week period. This suggests that submaximal heart rate represents something different from that being represented by heart rate recovery. Thus, whereas heart rate during exercise may be a measure of cardiac load, heart rate recovery after exercise may represent the adaptive state or the capacity of the autonomic nervous system to respond to that cardiac load.

CHAPTER 5

Question:

Does heart rate recovery reflect chronic adaptation to increases in training load during a periodised endurance training programme?

Answer:

Twelve weeks of endurance running training improved the average heart rate recovery in the first minute after exercise and reduced average submaximal heart rate in ten previously sedentary participants. These changes are proposed to be the result of a decrease in sympathetic activity to the heart. Chapter 4 found no change in submaximal heart rate over an acute 2-week period when training loads were either increased or decreased. However, the participants in Chapter 4 were already in an adapted state of training (Stage of Resistance), whereas the participants in Chapter 5 were in the Alarm Reaction Stage, particularly during the early part of the programme. The chronic physiological effects and the associated health benefits of the 12-week running programme are therefore evident from the changes observed in heart rate recovery and submaximal heart rate.

Question:

Does the relationship between training load and heart rate recovery persist in a periodised training programme, when training load varies?

Answer:

A weak relationship was found between TRIMP and heart rate recovery. This is in contrast to the significant correlation that was found in Chapter 4, when training load was held constant. The systematic changes in training load that form the structure of a periodised training programme may have influenced this relationship. These results suggest that the relationship between training load and heart rate recovery may be affected by the participant's current phase of training. This is in accordance with the different phases of training as defined by the General Adaptation Syndrome. In Chapter 4 participants were assumed to already be adapted to the amount of training they were doing (i.e. in the Resistance Stage of the General Adaptation Syndrome). In Chapter 5 the sedentary subjects were not accustomed to exercise training which placed them in a phase of adaptation (the Alarm Reaction Stage of the General Adaptation Syndrome). Thus it may be important to consider the phase of training or adaptation when assessing or monitoring the training response.

Question:

Does heart rate recovery respond in a predictable way to acute changes in training load during a periodised training programme?

Answer:

Mean percent heart rate recovery did not change in a predictable way in response to increases and decreases in training load between successive 2-week periods. This is in contrast to the trends found in Chapter 4, which showed decreases in heart rate recovery with increases in training load and a slight improvement in heart rate recovery with decreases in weekly training load. Differing results in this case may be due to the fact that participants were in different phases of training, as discussed previously. In addition the different time periods used in each of the studies to calculate training load may also have contributed to the disparity of results. The 2-week period used in this study may be too long to be regarded as an "acute" stress. The 1-week period used in Chapter 4 might better reflect the acute response of heart rate recovery to changes in training load.

Question:

Do individuals adapt in a similar way (acutely and chronically) after exposure to an endurance training programme?

Answer:

Although physical performance for the group, assessed by the 2.4 km time and the total number of heart beats during the 2.4 km assessment, improved after the completion of the running training programme, there were varying ways in which these changes manifested. Some participants improved their 2.4 km time significantly however others either did not improve their time or only improved it slightly. Most of these participants tended to complete the 2.4 km assessment at a lower average heart rate. Some participants appeared to respond well to relatively low training loads where others responded well to much higher training loads. In the current study it also appeared as though there were “slow responders” and “fast responders” to the running training programme. This highlights the wide variability in the way individuals respond physiologically to exercise and how this is translated into changes in performance. The relationship between training stimulus and physiological adaptation depends on many factors that influence an individual’s tolerance of an exercise load. The practical application of this finding is that there is a need to identify each individual’s training load threshold and response, to be able to personalise training prescription and optimise physiological adaptation on an individual basis.

INTERPRETATION AND PRACTICAL APPLICATIONS

Quantifying training load

The precise quantification of training load may contribute to a more accurate assessment of how an athlete is responding to the prescribed training. Although questionnaires about physical activity and training habits may provide information for monitoring general trends in population activity, attempts to quantify exercise dosage from data collected with questionnaires remain inadequate. Chapter 2 showed that the quantification of an athlete's training may be inaccurate when relying exclusively on self-reported data. Lack of accurate data may influence the effectiveness of customised training programmes. It is therefore recommended that the margin of error introduced by the use of self-reported data in research studies and training prescription should be accounted for, or where possible objective physiological measurements should be used to corroborate self-reported data.

Heart rate has previously been considered the most appropriate, practical and objective method of measuring exercise intensity. Heart rate-based equations such as TRIMP and Summated Heart Rate Zones Method have been developed in an attempt to quantify internal physiological training load during aerobic activity. There are however logistical problems with the use of heart rate-based methods to quantify training load. For example, if the heart rate monitor fails to record data for a training session for technical reasons there will be no information about the training load. In contrast, the Session RPE method does not depend on equipment or technology and therefore the risk of losing data about a session is low. The practical usefulness of the Session RPE method in quantifying training load is thus emphasised when the monitoring of training needs to be quick and easy and where heart rate monitors are not available. In these cases the Session RPE method provides reasonably accurate assessments of training load. However this recommendation needs to be considered in the context that there are many factors that may affect an athlete's personal perception of physical effort and that these factors need to be considered when using Session RPE.

Chapter 3 also investigated possible reasons for the relatively poor correlations found in some athletes between the objective (heart rate-based) and subjective (RPE-

based) quantification methods. It was suggested that since Session RPE is a more global indication of the difficulty of an exercise bout than heart rate, the variance not accounted for by the heart rate-based methods represents the numerous extraneous factors that contribute towards a person's personal perception of the difficulty of the session. Conversely, since to our knowledge neither of the heart rate-based equations assessed in Chapter 3 have been validated, it cannot be ruled out that there may be inherent flaws in these equations that may affect their relationship with the Session RPE method. For example, as proposed in Chapter 3, an explanation for the lack of precision is that these equations may disproportionately weight the heart rate data recorded during higher intensity exercise, and may under-estimate training load when proportionally more time is spent doing lower intensity exercise.

Monitoring the training response

Being able to monitor training load and assess the effectiveness of training adaptations is becoming increasingly important in the development of personalized training programmes that produce effective improvements in fitness and sports performance while at the same time reducing the risk of injury and overtraining. Ideally the technique of monitoring adaptations to exercise training should require simple, practical and non-invasive measures yet offer valuable, individualized information to the participant.

It is often assumed that $VO_2\text{max}$ is an indicator of "aerobic fitness" because it improves with training¹¹. Heart rate recovery also improves with training^{143;196;197}, thus a logical assumption is that this is also a measure of "fitness". However, in Chapter 4 no relationship was found between $VO_2\text{max}$ and heart rate recovery suggesting that these indices may measure and represent different entities. A significant relationship was found between training load and heart rate recovery in a group of trained athletes. Heart rate recovery is regulated by the autonomic nervous system. Therefore heart rate recovery may provide insight into the effect of habitual training loads on autonomic nervous system function rather than simply being a measure of general cardiorespiratory adaptation.

The basis for training is the manipulation of stress (exercise) stimuli and rest (recovery) periods over time to induce positive adaptations that contribute to improvements in performance. The decrease in heart rate recovery observed in Chapter 4 in participants that increased their training load acutely, may represent a negative training response to an increase in exercise stimulus. Conversely, after an acute decrease in training load, comparable to tapering, heart rate recovery may have a tendency to improve in athletes in an already adapted state of training. Submaximal heart rate did not respond in the same way as heart rate recovery to either increases or decreases in training load, suggesting that these two indices must also represent something different. Chapter 4 proposed that the use of heart rate recovery measures may contribute to a better understanding of positive and negative training effects and may in this way assist in optimising training prescription.

Chapter 5 revealed that heart rate recovery also responds to chronic exposure to endurance training. In a group of sedentary participants twelve weeks of endurance running training had a positive effect on selected performance variables (grouped data); improved 1-minute heart rate recovery and reduced submaximal heart rate. However, when the data were analysed on an individual basis some participants appeared to respond well to relatively low training loads whereas others responded well to much higher training loads. The data suggested that there were “slow responders” and “fast responders” to the running training programme.

Collectively, the results from Chapter 4 and 5 suggest that the relationship between training load and heart rate recovery may be affected by the participant’s phase of training and stage of adaptation. For example, it may be argued that most of the participants in Chapter 5 were in the Alarm Reaction Stage whereas the participants in Chapter 4 were in the Resistance Stage of the General Adaptation Syndrome. In this context a quantifiable relationship between changes in training load and subsequent changes in heart rate recovery needs to be established on an individual basis.

Figure 6.1 summarises the practical applications of some of the main findings from Chapter 4 and 5. At this stage it is not possible to establish a cause-effect relationship as there is no “gold standard” which can be used to quantify the training response. The relationships between training, the physiological response and

subsequent changes in performance are highly personal phenomena that depend on many factors that influence an individual's tolerance of an exercise load. In order to personalise training prescription and optimise physiological adaptation each individual's training load threshold and the variation in training response needs to be identified.

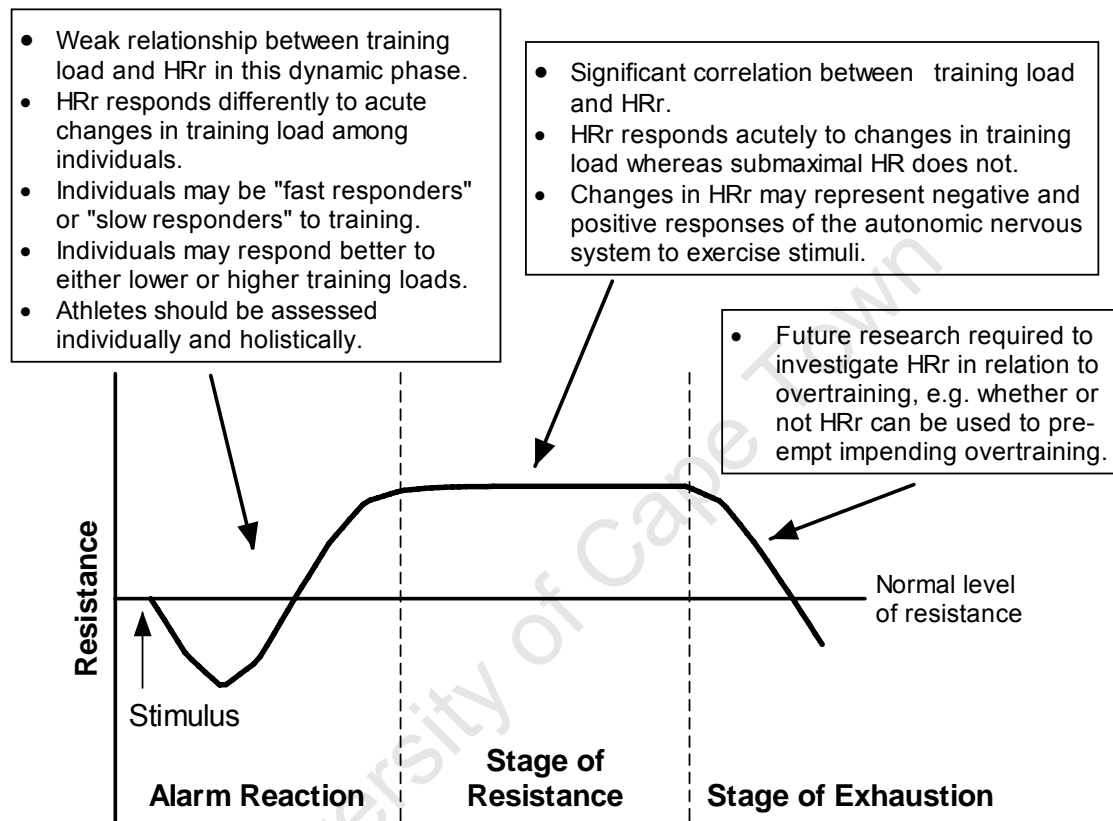


Figure 6.1: Summary of some of the main findings in Chapter 4 and 5 in the context of the General Adaptation Syndrome (Selye, 1974).

- HRr = heart rate recovery

FUTURE RESEARCH

Further research needs to confirm the findings in Chapter 3 as these findings may have important implications for using equations that attempt to quantify internal training stress. If the heart rate based equations do disproportionately weight the heart rate data recorded during higher intensity exercise and under-estimate training load when proportionally more training time is spent in lower-intensity exercise, further research may identify modifications to the equations required to more

accurately calculate training load in each individual. Alternatively if it is the Session RPE equation that over-estimates training load for athletes spending more time doing low-intensity exercise and under-estimates training load when proportionally more training time is spent in higher-intensity exercise, then the measure of Session RPE needs to be revisited and refined. Further investigation is also required to establish the exact cause of the relatively poor correlation between objective and subjective quantification methods found in some athletes. Previous studies have validated the Session RPE against heart rate based methods of quantifying training load^{51;52}. However, to our knowledge none of the heart rate-based equations have been validated either. Energy expenditure may be the “gold standard” against which all of these theoretical equations need to be validated.

The studies in Chapter 4 and 5 were the first studies to investigate the possibility of using heart rate recovery as a monitoring tool to assess training status. As such the questions asked were simple and clear in order to answer fundamental questions first, before more complex investigations are attempted in the future. Therefore care was taken not to over-interpret the results. Chapter 5 found that the heart rate recovery of the individuals participating in a 12-week running programme responded differently to changes in training load, and suggested that the phase of training may have an important influence on how heart rate recovery adapts to training. Therefore future research needs to consider and/or control the phase of training in which participants are when they are studied, and control the increases and decreases in training loads experienced by the participants. This refinement will facilitate the investigation and development of individual profiles of quantifiable training responses for each participant.

CLOSING STATEMENT

The findings of the studies in Chapters 2 – 5 of this thesis can be interpreted to suggest that there is no single variable that can be used exclusively as a marker of training status. Certainly there are data to show that monitoring heart rate recovery may contribute to a better understanding of positive and negative training effects on autonomic nervous system function. From a practical perspective if the HIMS test is administered frequently, a profile of changes in heart rate recovery after the test may

be established and monitored for each individual. While the heart rate recovery test on its own may not be diagnostic, the data from this thesis suggests that heart rate recovery can be an informative measure when interpreted in conjunction with other data such as measures of performance and physiological and psychological changes, taking into account extraneous factors that might influence the rate of adaptation. Gathering these data regularly can create a comprehensive assessment of an athlete's response to training and may provide useful information with which to design and prescribe personalized training programmes. At this stage, until further research has established a thorough understanding of what heart rate recovery represents and has investigated individual heart rate recovery responses during different phases of training, it is recommended that a holistic view be adopted in monitoring training adaptations on an individual basis.

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CHAPTER 7

REFERENCES

University of Cape Town

1. Williams JG, Eston RG: Determination of the intensity dimension in vigorous exercise programmes with particular reference to the use of the rating of perceived exertion. *Sports Med.* 8:177-189, 1989
2. Budgett R: Fatigue and underperformance in athletes: the overtraining syndrome. *Br.J.Sports Med.* 32:107-110, 1998
3. Meeusen R, Duclos M, Gleeson M, et al: Prevention, diagnosis and treatment of the overtraining syndrome. *Eur J Sport Sci* 6:1-14, 2006
4. Wenger HA, Bell GJ: The interactions of intensity, frequency and duration of exercise training in altering cardiorespiratory fitness. *Sports Med.* 3:346-356, 1986
5. Coyle EF: Physical activity as a metabolic stressor. *Am.J.Clin.Nutr.* 72:512S-520S, 2000
6. Hopkins WG: Quantification of training in competitive sports. Methods and applications. *Sports Med.* 12:161-183, 1991
7. Shephard RJ: Limits to the measurement of habitual physical activity by questionnaires. *Br.J.Sports Med.* 37:197-206, 2003
8. Buchheit M, Gindre C: Cardiac parasympathetic regulation: respective associations with cardiorespiratory fitness and training load. *Am.J.Physiol Heart Circ.Physiol* 291:H451-H458, 2006
9. Arts FJ, Kuipers H: The relation between power output, oxygen uptake and heart rate in male athletes. *Int.J.Sports Med.* 15:228-231, 1994
10. Xu F, Rhodes EC: Oxygen uptake kinetics during exercise. *Sports Med.* 27:313-327, 1999
11. Brooks GA, Fahey TD, White TP, et al: *Exercise Physiology – Human Bioenergetics and its Applications (ed3)*. USA, McGraw-Hill Companies, Inc., 2000
12. Kohrt WM, Morgan DW, Bates B, et al: Physiological responses of triathletes to maximal swimming, cycling, and running. *Med.Sci.Sports Exerc.* 19:51-55, 1987

-
13. Swain DP: Energy cost calculations for exercise prescription: an update. *Sports Med.* 30:17-22, 2000
 14. Swain DP, Leutholtz BC, King ME, et al: Relationship between % heart rate reserve and % VO₂ reserve in treadmill exercise. *Med.Sci.Sports Exerc.* 30:318-321, 1998
 15. Swain DP, Leutholtz BC: Heart rate reserve is equivalent to %VO₂ reserve, not to %VO₂max. *Med.Sci.Sports Exerc.* 29:410-414, 1997
 16. Baldwin J, Snow RJ, Febbraio MA: Effect of training status and relative exercise intensity on physiological responses in men. *Med.Sci.Sports Exerc.* 32:1648-1654, 2000
 17. Skinner JS, Wilmore KM, Krasnoff JB, et al: Adaptation to a standardized training program and changes in fitness in a large, heterogeneous population: the HERITAGE Family Study. *Med.Sci.Sports Exerc.* 32:157-161, 2000
 18. Pyne DB, Lee H, Swanwick KM: Monitoring the lactate threshold in world-ranked swimmers. *Med.Sci.Sports Exerc.* 33:291-297, 2001
 19. Jacobs I: Blood lactate. Implications for training and sports performance. *Sports Med.* 3:10-25, 1986
 20. Stegmann H, Kindermann W, Schnabel A: Lactate kinetics and individual anaerobic threshold. *Int.J.Sports Med.* 2:160-165, 1981
 21. Eniseler N: Heart rate and blood lactate concentrations as predictors of physiological load on elite soccer players during various soccer training activities. *J.Strength.Cond.Res.* 19:799-804, 2005
 22. Majumdar P, Khanna GL, Malik V, et al: Physiological analysis to quantify training load in badminton. *Br.J.Sports Med.* 31:342-345, 1997
 23. Swart J, Jennings CL: Use of blood lactate concentration as a marker of training status. *S Afr J Sports Med* 16:3-7, 2004
 24. Urhausen A, Gabriel HH, Weiler B, et al: Ergometric and psychological findings during overtraining: a long-term follow-up study in endurance athletes. *Int.J.Sports Med.* 19:114-120, 1998

-
25. Jeukendrup AE, Hesselink MK: Overtraining--what do lactate curves tell us? *Br.J.Sports Med.* 28:239-240, 1994
 26. Kirwan JP, Costill DL, Flynn MG, et al: Physiological responses to successive days of intense training in competitive swimmers. *Med.Sci.Sports Exerc.* 20:255-259, 1988
 27. Achten J, Jeukendrup AE: Heart rate monitoring: applications and limitations. *Sports Med.* 33:517-538, 2003
 28. Robinson DM, Robinson SM, Hume PA, et al: Training intensity of elite male distance runners. *Med.Sci.Sports Exerc.* 23:1078-1082, 1991
 29. Polanczyk CA, Rohde LE, Moraes RS, et al: Sympathetic nervous system representation in time and frequency domain indices of heart rate variability. *Eur.J.Appl.Physiol Occup.Physiol* 79:69-73, 1998
 30. Kannankeril PJ, Le FK, Kadish AH, et al: Parasympathetic effects on heart rate recovery after exercise. *J.Investig.Med.* 52:394-401, 2004
 31. Yamamoto Y, Hughson RL, Peterson JC: Autonomic control of heart rate during exercise studied by heart rate variability spectral analysis. *J.Appl.Physiol* 71:1136-1142, 1991
 32. Nakamura Y, Yamamoto Y, Muraoka I: Autonomic control of heart rate during physical exercise and fractal dimension of heart rate variability. *J.Appl.Physiol* 74:875-881, 1993
 33. Robinson BF, Epstein SE, Beiser GD, et al: Control of heart rate by the autonomic nervous system. Studies in man on the interrelation between baroreceptor mechanisms and exercise. *Circ.Res.* 19:400-411, 1966
 34. Carter JB, Banister EW, Blaber AP: Effect of endurance exercise on autonomic control of heart rate. *Sports Med.* 33:33-46, 2003
 35. Karvonen J, Vuorimaa T: Heart rate and exercise intensity during sports activities. Practical application. *Sports Med.* 5:303-311, 1988
 36. Lambert MI, Mbamba ZH, St Clair Gibson A: Heart rate during training and competition for long-distance running. *J Sports Sci* 16:S85-S90, 1998
-

-
37. Bagger M, Petersen PH, Pedersen PK: Biological variation in variables associated with exercise training. *Int.J.Sports Med.* 24:433-440, 2003
 38. Borg G: Perceived exertion as an indicator of somatic stress. *Scand.J.Rehabil.Med.* 2:92-98, 1970
 39. Green JM, McLester JR, Crews TR, et al: RPE association with lactate and heart rate during high-intensity interval cycling. *Med.Sci.Sports Exerc.* 38:167-172, 2006
 40. Little T, Williams AG: Measures of exercise intensity during soccer training drills with professional soccer players. *J.Strength.Cond.Res.* 21:367-371, 2007
 41. Ozkan A, Kin-Isler A: The reliability and validity of regulating exercise intensity by ratings of perceived exertion in step dance sessions. *J.Strength.Cond.Res.* 21:296-300, 2007
 42. Chen MJ, Fan X, Moe ST: Criterion-related validity of the Borg ratings of perceived exertion scale in healthy individuals: a meta-analysis. *J.Sports Sci.* 20:873-899, 2002
 43. Gros Lambert A, Mahon AD: Perceived exertion : influence of age and cognitive development. *Sports Med.* 36:911-928, 2006
 44. Banister EW: Modeling elite athletic performance, in MacDougall JD, Wenger HA, Green HJ (eds): *Physiological Testing of the High-Performance Athlete.* vol2.ampaign, IL, Human Kinetics Books, 1991, pp 403-425
 45. Edwards S: *The Heart Rate Monitor Book* Sacramento, CA, Fleet Feet Press, 1993, pp 113-129
 46. Impellizzeri FM, Rampinini E, Coutts AJ, et al: Use of RPE-based training load in soccer. *Med.Sci.Sports Exerc.* 36:1042-1047, 2004
 47. Lucia A, Hoyos J, Santalla A, et al: Tour de France versus Vuelta a Espana: which is harder? *Med.Sci.Sports Exerc.* 35:872-878, 2003
 48. Earnest CP, Jurca R, Church TS, et al: Relation between physical exertion and heart rate variability characteristics in professional cyclists during the Tour of Spain. *Br.J.Sports Med.* 38:568-575, 2004
-

-
49. Foster C, Daines E, Hector L, et al: Athletic performance in relation to training load. *Wis.Med.J.* 95:370-374, 1996
 50. Borg GA: Psychophysical bases of perceived exertion. *Med.Sci.Sports Exerc.* 14:377-381, 1982
 51. Foster C, Florhaug JA, Franklin J, et al: A new approach to monitoring exercise training. *J.Strength.Cond.Res.* 15:109-115, 2001
 52. Foster C: Monitoring training in athletes with reference to overtraining syndrome. *Med.Sci.Sports Exerc.* 30:1164-1168, 1998
 53. Sweet TW, Foster C, McGuigan MR, et al: Quantitation of resistance training using the session rating of perceived exertion method. *J.Strength.Cond.Res.* 18:796-802, 2004
 54. Day ML, McGuigan MR, Brice G, et al: Monitoring exercise intensity during resistance training using the session RPE scale. *J.Strength.Cond.Res.* 18:353-358, 2004
 55. McGuigan MR, Egan AD, Foster C: Salivary cortisol responses and perceived exertion during high intensity and low intensity bouts of resistance exercise. *J Sports Sci Med* 3:8-15, 2004
 56. Jeukendrup A, van Diemen A: Heart rate monitoring during training and competition in cyclists. *J Sports Sci* 16:S91-S99, 1998
 57. Foster C, Heimann KM, Esten PL, et al: Differences in perceptions of training by coaches and athletes. *S Afr J Sports Med* 8:3-7, 2001
 58. Larsson P: Global positioning system and sport-specific testing. *Sports Med* 33:1093-1101, 2003
 59. Townshend AD, Worringham CJ, Stewart IB: Assessment of speed and position during human locomotion using nondifferential GPS. *Med Sci Sports Exerc.* 40:124-132, 2008
 60. Rodriguez DA, Brown AL, Troped PJ: Portable global positioning units to complement accelerometry-based physical activity monitors. *Med Sci Sports Exerc.* 37:S572-S581, 2005

-
61. Larsson P, Henriksson-Larsen K: The use of dGPS and simultaneous metabolic measurements during orienteering. *Med Sci Sports Exerc.* 33:1919-1924, 2001
 62. Schutz Y, Herren R: Assessment of speed of human locomotion using a differential satellite global positioning system. *Med Sci Sports Exerc.* 32:642-646, 2000
 63. Vermeulen AD, Evans DL: Measurements of fitness in thoroughbred racehorses using field studies of heart rate and velocity with a global positioning system. *Equine Vet.J Suppl* 113-117, 2006
 64. Kingston JK, Soppet GM, Rogers CW, et al: Use of a global positioning and heart rate monitoring system to assess training load in a group of thoroughbred racehorses. *Equine Vet.J Suppl* 106-109, 2006
 65. Fitz-Clarke JR, Morton RH, Banister EW: Optimizing athletic performance by influence curves. *J.Appl.Physiol* 71:1151-1158, 1991
 66. Morton RH, Fitz-Clarke JR, Banister EW: Modeling human performance in running. *J.Appl.Physiol* 69:1171-1177, 1990
 67. Busso T, Carasso C, Lacour JR: Adequacy of a systems structure in the modeling of training effects on performance. *J.Appl.Physiol* 71:2044-2049, 1991
 68. Wood RE, Hayter S, Rowbottom D, et al: Applying a mathematical model to training adaptation in a distance runner. *Eur.J.Appl.Physiol* 94:310-316, 2005
 69. Busso T, Denis C, Bonnefoy R, et al: Modeling of adaptations to physical training by using a recursive least squares algorithm. *J.Appl.Physiol* 82:1685-1693, 1997
 70. Busso T, Benoit H, Bonnefoy R, et al: Effects of training frequency on the dynamics of performance response to a single training bout. *J.Appl.Physiol* 92:572-580, 2002
 71. Taha T, Thomas SG: Systems modelling of the relationship between training and performance. *Sports Med.* 33:1061-1073, 2003

-
72. Busso T: Variable dose-response relationship between exercise training and performance. *Med.Sci.Sports Exerc.* 35:1188-1195, 2003
 73. Hellard P, Avalos M, Millet G, et al: Modeling the residual effects and threshold saturation of training: a case study of Olympic swimmers. *J.Strength.Cond.Res.* 19:67-75, 2005
 74. Mujika I, Busso T, Lacoste L, et al: Modeled responses to training and taper in competitive swimmers. *Med.Sci.Sports Exerc.* 28:251-258, 1996
 75. Selye H: *Stress without Distress* Kent, Great Britain, Hodder and Stoughton Ltd, 1974, pp 3-103
 76. Perdrizet GA: Hans Selye and beyond: response to stress. *Cell Stress and Caperones* 2:214-219, 1999
 77. Budgett R: Overtraining syndrome. *Br.J.Sports Med.* 24:231-236, 1990
 78. Derman W, Schwellnus MP, Lambert MI, et al: The 'worn-out athlete': a clinical approach to chronic fatigue in athletes. *J.Sports Sci.* 15:341-351, 1997
 79. Kuipers H, Keizer HA: Overtraining in elite athletes. Review and directions for the future. *Sports Med.* 6:79-92, 1988
 80. Lehmann M, Foster C, Keul J: Overtraining in endurance athletes: a brief review. *Med.Sci.Sports Exerc.* 25:854-862, 1993
 81. Morton RH: Modeling training and overtraining. *J.Sports Sci.* 15:335-340, 1997
 82. Kuipers H: Training and overtraining: an introduction. *Med.Sci.Sports Exerc.* 30:1137-1139, 1998
 83. Uusitalo ALT: Overtraining - Making a difficult diagnosis and implementing targeted treatment. *Phys.Sportsmed.* 29:35-50, 2001
 84. Noakes TD: Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance. *Scand.J.Med.Sci.Sports* 10:123-145, 2000

-
85. Abernethy PJ, Thayer R, Taylor AW: Acute and chronic responses of skeletal muscle to endurance and sprint exercise. A review. *Sports Med.* 10:365-389, 1990
 86. Jones AM, Carter H: The effect of endurance training on parameters of aerobic fitness. *Sports Med.* 29:373-386, 2000
 87. Balaban EP, Snell P, Stray-Gundersen J, et al: The effect of running on serum and red cell ferritin. A longitudinal comparison. *Int.J.Sports Med.* 16:278-282, 1995
 88. Bergman BC, Butterfield GE, Wolfel EE, et al: Evaluation of exercise and training on muscle lipid metabolism. *Am.J.Physiol* 276:E106-E117, 1999
 89. Coggan AR: Plasma glucose metabolism during exercise: effect of endurance training in humans. *Med.Sci.Sports Exerc.* 29:620-627, 1997
 90. Hamilton MT, Booth FW: Skeletal muscle adaptation to exercise: a century of progress. *J.Appl.Physiol* 88:327-331, 2000
 91. Hawley JA, Stepto NK: Adaptations to training in endurance cyclists: implications for performance. *Sports Med.* 31:511-520, 2001
 92. Hawley JA: Adaptations of skeletal muscle to prolonged, intense endurance training. *Clin.Exp.Pharmacol.Physiol* 29:218-222, 2002
 93. Hakkinen K, Alen M, Kraemer WJ, et al: Neuromuscular adaptations during concurrent strength and endurance training versus strength training. *Eur.J.Appl.Physiol Occup.Physiol* 89:42-52, 2003
 94. Angeli A, Minetto M, Dovoio A, et al: The overtraining syndrome in athletes: a stress-related disorder. *J.Endocrinol.Invest* 27:603-612, 2004
 95. Beard J, Tobin B: Iron status and exercise. *Am.J.Clin.Nutr.* 72:594S-597S, 2000
 96. Wilkinson JG, Martin DT, Adams AA, et al: Iron status in cyclists during high-intensity interval training and recovery. *Int.J.Sports Med.* 23:544-548, 2002

-
97. Smith DJ, Roberts D: Effects of high volume and/or intense exercise on selected blood chemistry parameters. *Clin.Biochem.* 27:435-440, 1994
 98. Dickson DN, Wilkinson RL, Noakes TD: Effects of ultra-marathon training and racing on hematologic parameters and serum ferritin levels in well-trained athletes. *Int.J.Sports Med.* 3:111-117, 1982
 99. Bourque SP, Pate RR, Branch JD: Twelve weeks of endurance exercise training does not affect iron status measures in women. *J.Am.Diet.Assoc.* 97:1116-1121, 1997
 100. Meeusen R, Piacentini MF, Busschaert B, et al: Hormonal responses in athletes: the use of a two bout exercise protocol to detect subtle differences in (over)training status. *Eur.J.Appl.Physiol* 91:140-146, 2004
 101. Urhausen A, Gabriel H, Kindermann W: Blood hormones as markers of training stress and overtraining. *Sports Med.* 20:251-276, 1995
 102. Hedelin R, Kenttä G, Wiklund U, et al: Short-term overtraining: effects on performance, circulatory responses, and heart rate variability. *Med.Sci.Sports Exerc.* 32:1480-1484, 2000
 103. Greiwe JS, Hickner RC, Hansen PA, et al: Effects of endurance exercise training on muscle glycogen accumulation in humans. *J.Appl.Physiol* 87:222-226, 1999
 104. Schmitt B, Fluck M, Decombaz J, et al: Transcriptional adaptations of lipid metabolism in tibialis anterior muscle of endurance-trained athletes. *Physiol Genomics* 15:148-157, 2003
 105. Horowitz JF, Klein S: Lipid metabolism during endurance exercise. *Am.J.Clin.Nutr.* 72:558S-563S, 2000
 106. van Loon LJ, Koopman R, Stegen JH, et al: Intramyocellular lipids form an important substrate source during moderate intensity exercise in endurance-trained males in a fasted state. *J.Physiol* 553:611-625, 2003

-
107. Friedlander AL, Casazza GA, Horning MA, et al: Endurance training increases fatty acid turnover, but not fat oxidation, in young men. *J.Appl.Physiol* 86:2097-2105, 1999
 108. Mayes R, Hardman AE, Williams C: The influence of training on endurance and blood lactate concentration during submaximal exercise. *Br.J.Sports Med.* 21:119-124, 1987
 109. Tremblay MS, Copeland JL, Van Helder W: Effect of training status and exercise mode on endogenous steroid hormones in men. *J.Appl.Physiol* 96:531-539, 2004
 110. Bell GJ, Syrotuik D, Martin TP, et al: Effect of concurrent strength and endurance training on skeletal muscle properties and hormone concentrations in humans. *Eur.J.Appl.Physiol* 81:418-427, 2000
 111. Filaire E, Bernain X, Sagnol M, et al: Preliminary results on mood state, salivary testosterone:cortisol ratio and team performance in a professional soccer team. *Eur.J.Appl.Physiol* 86:179-184, 2001
 112. Hawley JA, Spargo FJ: Metabolic adaptations to marathon training and racing. *Sports Med.* 37:328-331, 2007
 113. Kenttä G, Hassmen P: Overtraining and recovery. A conceptual model. *Sports Med.* 26:1-16, 1998
 114. Noakes TD: *Lore of Running*. Cape Town, Oxford University Press Southern Africa, 2001, pp 3-1277
 115. Connolly DA, Sayers SP, McHugh MP: Treatment and prevention of delayed onset muscle soreness. *J.Strength.Cond.Res.* 17:197-208, 2003
 116. Kuipers H: Exercise-induced muscle damage. *Int.J.Sports Med.* 15:132-135, 1994
 117. Cheung K, Hume P, Maxwell L: Delayed onset muscle soreness : treatment strategies and performance factors. *Sports Med.* 33:145-164, 2003
 118. Lambert MI, Marcus P, Burgess T, et al: Electro-membrane microcurrent therapy reduces signs and symptoms of muscle damage. *Med.Sci.Sports Exerc.* 34:602-607, 2002
-

-
119. Semark A, Noakes TD, St Clair Gibson A, et al: The effect of a prophylactic dose of flurbiprofen on muscle soreness and sprinting performance in trained subjects. *J.Sports Sci.* 17:197-203, 1999
 120. Raglin JS, Morgan WP: Development of a scale for use in monitoring training-induced distress in athletes. *Int.J.Sports Med.* 15:84-88, 1994
 121. Rietjens GJ, Kuipers H, Adam JJ, et al: Physiological, biochemical and psychological markers of strenuous training-induced fatigue. *Int.J.Sports Med.* 26:16-26, 2005
 122. Halson SL, Bridge MW, Meeusen R, et al: Time course of performance changes and fatigue markers during intensified training in trained cyclists. *J.Appl.Physiol* 93:947-956, 2002
 123. van Hooff ML, Geurts SA, Kompier MA, et al: "How fatigued do you currently feel?" Convergent and discriminant validity of a single-item fatigue measure. *J.Occup.Health* 49:224-234, 2007
 124. Mazzeo RS: Catecholamine responses to acute and chronic exercise. *Med.Sci.Sports Exerc.* 23:839-845, 1991
 125. Aubert AE, Seps B, Beckers F: Heart rate variability in athletes. *Sports Med.* 33:889-919, 2003
 126. Laukkanen RMT, Virtanen P: Heart rate monitors - state of the art. *J.Sports Sci.* 16:53-57, 1998
 127. Uusitalo AL, Uusitalo AJ, Rusko HK: Exhaustive endurance training for 6-9 weeks did not induce changes in intrinsic heart rate and cardiac autonomic modulation in female athletes. *Int.J.Sports Med.* 19:532-540, 1998
 128. Bonaduce D, Petretta M, Cavallaro V, et al: Intensive training and cardiac autonomic control in high level athletes. *Med.Sci.Sports Exerc.* 30:691-696, 1998
 129. Wilmore JH, Stanforth PR, Gagnon J, et al: Heart rate and blood pressure changes with endurance training: the HERITAGE Family Study. *Med.Sci.Sports Exerc.* 33:107-116, 2001

-
130. Levy WC, Cerqueira MD, Harp GD, et al: Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. *Am.J.Cardiol.* 82:1236-1241, 1998
 131. Pollock ML: The quantification of endurance training programs. *Exerc.Sport Sci.Rev.* 1:155-188, 1973
 132. Wilmore JH, Stanforth PR, Gagnon J, et al: Endurance exercise training has a minimal effect on resting heart rate: the HERITAGE Study. *Med.Sci.Sports Exerc.* 28:829-835, 1996
 133. Perini R, Tironi A, Cautero M, et al: Seasonal training and heart rate and blood pressure variabilities in young swimmers. *Eur.J.Appl.Physiol* 97:395-403, 2006
 134. Thompson PD: Cardiovascular adaptations to marathon running : the marathoner's heart. *Sports Med.* 37:444-447, 2007
 135. Smith ML, Hudson DL, Graitzer HM, et al: Exercise training bradycardia: the role of autonomic balance. *Med.Sci.Sports Exerc.* 21:40-44, 1989
 136. Melanson EL, Freedson PS: The effect of endurance training on resting heart rate variability in sedentary adult males. *Eur.J.Appl.Physiol* 85:442-449, 2001
 137. Jeukendrup AE, Hesselink MK, Snyder AC, et al: Physiological changes in male competitive cyclists after two weeks of intensified training. *Int.J.Sports Med.* 13:534-541, 1992
 138. Waldeck M, Lambert MI: Heart rate during sleep: implications for monitoring training status. *J Sports Sci Med* 3:133-138, 2003
 139. Sadamoto T, Fuchi T, Taniguchi Y, et al: Effect of 8 weeks submaximal conditioning and deconditioning on heart rate during sleep in middle-aged women., in McPherson BD (ed): *Sport an Ageing. Human Kinetics, Champaign*, 1986, pp 233-240
 140. Sedgwick AW, Craig RJ, Crouch R, et al: The effects of physical training on the day and night long-term heart-rates of middle-aged men. *Eur.J.Appl.Physiol Occup.Physiol* 33:307-314, 1974

-
141. Pigozzi F, Alabiso A, Parisi A, et al: Effects of aerobic exercise training on 24 hr profile of heart rate variability in female athletes. *J.Sports Med.Phys.Fitness* 41:101-107, 2001
 142. Skinner JS, Gaskill SE, Rankinen T, et al: Heart rate versus %VO₂max: age, sex, race, initial fitness, and training response-HERITAGE. *Med.Sci.Sports Exerc.* 35:1908-1913, 2003
 143. Short KR, Sedlock DA: Excess postexercise oxygen consumption and recovery rate in trained and untrained subjects. *J.Appl.Physiol* 83:153-159, 1997
 144. Lake MJ, Cavanagh PR: Six weeks of training does not change running mechanics or improve running economy. *Med.Sci.Sports Exerc.* 28:860-869, 1996
 145. Swaine IL, Linden RJ, Mary DA: Loss of exercise training-induced bradycardia with continued improvement in fitness. *J.Sports Sci.* 12:477-481, 1994
 146. Charlton GA, Crawford MH: Physiologic consequences of training. *Cardiol.Clin.* 15:345-354, 1997
 147. Spina RJ, Ogawa T, Kohrt WM, et al: Differences in cardiovascular adaptations to endurance exercise training between older men and women. *J.Appl.Physiol* 75:849-855, 1993
 148. Weir LL, Weir JP, Housh TJ, et al: Effect of an aerobic training program on physical working capacity at heart rate threshold. *Eur.J.Appl.Physiol Occup.Physiol* 75:351-356, 1997
 149. Mier CM, Turner MJ, Ehsani AA, et al: Cardiovascular adaptations to 10 days of cycle exercise. *J.Appl.Physiol* 83:1900-1906, 1997
 150. Spina RJ, Ogawa T, Martin WHI, et al: Exercise training prevents decline in stroke volume during exercise in young healthy subjects. *J.Appl.Physiol* 72:2458-2462, 1992

151. Convertino VA: Heart rate and sweat rate responses associated with exercise-induced hypervolemia. *Med.Sci.Sports Exerc.* 15:77-82, 1983
152. Costill DL, Flynn MG, Kirwan JP, et al: Effects of repeated days of intensified training on muscle glycogen and swimming performance. *Med.Sci.Sports Exerc.* 20:249-254, 1988
153. Houmard JA, Costill DL, Mitchell JB, et al: Reduced training maintains performance in distance runners. *Int.J.Sports Med.* 11:46-52, 1990
154. Coyle EF, Martin WHI, Sinacore DR, et al: Time course of loss of adaptations after stopping prolonged intense endurance training. *J.Appl.Physiol* 57:1857-1864, 1984
155. Zavorsky GS: Evidence and possible mechanisms of altered maximum heart rate with endurance training and tapering. *Sports Med.* 29:13-26, 2000
156. Kingwell BA, Dart AM, Jennings GL, et al: Exercise training reduces the sympathetic component of the blood pressure-heart rate baroreflex in man. *Clin.Sci.(Lond)* 82:357-362, 1992
157. Werle EO, Strobel G, Weicker H: Decrease in rat cardiac beta 1- and beta 2-adrenoceptors by training and endurance exercise. *Life Sci.* 46:9-17, 1990
158. Sylvestre-Gervais L, Nadeau A, Nguyen MH, et al: Effects of physical training on beta-adrenergic receptors in rat myocardial tissue. *Cardiovasc.Res.* 16:530-534, 1982
159. Portier H, Louisy F, Laude D, et al: Intense endurance training on heart rate and blood pressure variability in runners. *Med.Sci.Sports Exerc.* 33:1120-1125, 2001
160. Tulppo MP, Hautala AJ, Makikallio TH, et al: Effects of aerobic training on heart rate dynamics in sedentary subjects. *J.Appl.Physiol* 95:364-372, 2003

-
161. Iwasaki K, Zhang R, Zuckerman JH, et al: Dose-response relationship of the cardiovascular adaptation to endurance training in healthy adults: how much training for what benefit? *J.Appl.Physiol* 95:1575-1583, 2003
 162. al Ani M, Munir SM, White M, et al: Changes in R-R variability before and after endurance training measured by power spectral analysis and by the effect of isometric muscle contraction. *Eur.J.Appl.Physiol Occup.Physiol* 74:397-403, 1996
 163. Loimaala A, Huikuri H, Oja P, et al: Controlled 5-mo aerobic training improves heart rate but not heart rate variability or baroreflex sensitivity. *J.Appl.Physiol* 89:1825-1829, 2000
 164. Carter JB, Banister EW, Blaber AP: The effect of age and gender on heart rate variability after endurance training. *Med.Sci.Sports Exerc.* 35:1333-1340, 2003
 165. Buchheit M, Simon C, Piquard F, et al: Effects of increased training load on vagal-related indexes of heart rate variability: a novel sleep approach. *Am.J.Physiol Heart Circ.Physiol* 287:H2813-H2818, 2004
 166. Buchheit M, Simon C, Viola AU, et al: Heart rate variability in sportive elderly: relationship with daily physical activity. *Med.Sci.Sports Exerc.* 36:601-605, 2004
 167. Buchheit M, Simon C, Charloux A, et al: Heart rate variability and intensity of habitual physical activity in middle-aged persons. *Med.Sci.Sports Exerc.* 37:1530-1534, 2005
 168. Pichot V, Roche F, Gaspoz JM, et al: Relation between heart rate variability and training load in middle-distance runners. *Med.Sci.Sports Exerc.* 32:1729-1736, 2000
 169. Pichot V, Busso T, Roche F, et al: Autonomic adaptations to intensive and overload training periods: a laboratory study. *Med.Sci.Sports Exerc.* 34:1660-1666, 2002
 170. Tulppo MP, Makikallio TH, Seppanen T, et al: Vagal modulation of heart rate during exercise: effects of age and physical fitness. *Am.J.Physiol* 274:H424-H429, 1998
-

-
171. Ramaekers D, Ector H, Aubert AE, et al: Heart rate variability and heart rate in healthy volunteers. Is the female autonomic nervous system cardioprotective? *Eur.Heart J.* 19:1334-1341, 1998
 172. Shetler K, Marcus R, Froelicher VF, et al: Heart rate recovery: validation and methodologic issues. *J.Am.Coll.Cardiol.* 38:1980-1987, 2001
 173. Savin WM, Davidson DM, Haskell WL: Autonomic contribution to heart rate recovery from exercise in humans. *J.Appl.Physiol* 53:1572-1575, 1982
 174. Javorka M, Zila I, Balharek T, et al: Heart rate recovery after exercise: relations to heart rate variability and complexity. *Braz.J.Med.Biol.Res.* 35:991-1000, 2002
 175. Kannankeril PJ, Goldberger JJ: Parasympathetic effects on cardiac electrophysiology during exercise and recovery. *Am.J.Physiol Heart Circ.Physiol* 282:H2091-H2098, 2002
 176. Pierpont GL, Voth EJ: Assessing autonomic function by analysis of heart rate recovery from exercise in healthy subjects. *Am.J.Cardiol.* 94:64-68, 2004
 177. Pierpont GL, Stolpman DR, Gornick CC: Heart rate recovery post-exercise as an index of parasympathetic activity. *J.Auton.Nerv.Syst.* 80:169-174, 2000
 178. Mahon AD, Anderson CS, Hipp MJ, et al: Heart rate recovery from submaximal exercise in boys and girls. *Med.Sci.Sports Exerc.* 35:2093-2097, 2003
 179. Cole CR, Blackstone EH, Pashkow FJ, et al: Heart-rate recovery immediately after exercise as a predictor of mortality. *N.Engl.J.Med.* 341:1351-1357, 1999
 180. Lipinski MJ, Vetrovec GW, Froelicher VF: Importance of the first two minutes of heart rate recovery after exercise treadmill testing in predicting mortality and the presence of coronary artery disease in men. *Am.J.Cardiol.* 93:445-449, 2004

-
181. Morshedi-Meibodi A, Larson MG, Levy D, et al: Heart rate recovery after treadmill exercise testing and risk of cardiovascular disease events (The Framingham Heart Study). *Am.J.Cardiol.* 90:848-852, 2002
 182. Desai MY, Pena-Almaguer E, Mannting F: Abnormal heart rate recovery after exercise: a comparison with known indicators of increased mortality. *Cardiology* 96:38-44, 2001
 183. Desai MY, Pena-Almaguer E, Mannting F: Abnormal heart rate recovery after exercise as a reflection of an abnormal chronotropic response. *Am.J.Cardiol.* 87:1164-1169, 2001
 184. Gaibazzi N, Petrucci N, Ziacchi V: One-minute heart rate recovery after cycloergometer exercise testing as a predictor of mortality in a large cohort of exercise test candidates: substantial differences with the treadmill-derived parameter. *Ital.Heart J.* 5:183-188, 2004
 185. Seshadri N, Gildea TR, McCarthy K, et al: Association of an abnormal exercise heart rate recovery with pulmonary function abnormalities. *Chest* 125:1286-1291, 2004
 186. Kizilbash MA, Carnethon MR, Chan C, et al: The temporal relationship between heart rate recovery immediately after exercise and the metabolic syndrome: the CARDIA study. *Eur.Heart J.* 27:1592-1596, 2006
 187. Tiukinhoy S, Beohar N, Hsie M: Improvement in heart rate recovery after cardiac rehabilitation. *J.Cardiopulm.Rehabil.* 23:84-87, 2003
 188. Streuber SD, Amsterdam EA, Stebbins CL: Heart rate recovery in heart failure patients after a 12-week cardiac rehabilitation program. *Am.J.Cardiol.* 97:694-698, 2006
 189. Myers J, Hadley D, Oswald U, et al: Effects of exercise training on heart rate recovery in patients with chronic heart failure. *Am.Heart J.* 153:1056-1063, 2007

-
190. Giallauria F, Lucci R, Pietrosante M, et al: Exercise-based cardiac rehabilitation improves heart rate recovery in elderly patients after acute myocardial infarction. *J.Gerontol.A Biol.Sci.Med.Sci.* 61:713-717, 2006
 191. Giallauria F, De Lorenzo A, Pilerci F, et al: Long-term effects of cardiac rehabilitation on end-exercise heart rate recovery after myocardial infarction. *Eur.J.Cardiovasc.Prev.Rehabil.* 13:544-550, 2006
 192. Giallauria F, Del Forno D, Pilerci F, et al: Improvement of heart rate recovery after exercise training in older people. *J.Am.Geriatr.Soc.* 53:2037-2038, 2005
 193. MacMillan JS, Davis LL, Durham CF, et al: Exercise and heart rate recovery. *Heart Lung* 35:383-390, 2006
 194. Bunc V, Heller J, Leso J: Kinetics of heart rate responses to exercise. *J.Sports Sci.* 6:39-48, 1988
 195. Hagberg JM, Hickson RC, Ehsani AA, et al: Faster adjustment to and recovery from submaximal exercise in the trained state. *J.Appl.Physiol* 48:218-224, 1980
 196. Sugawara J, Murakami H, Maeda S, et al: Change in post-exercise vagal reactivation with exercise training and detraining in young men. *Eur.J.Appl.Physiol* 85:259-263, 2001
 197. Yamamoto K, Miyachi M, Saitoh T, et al: Effects of endurance training on resting and post-exercise cardiac autonomic control. *Med.Sci.Sports Exerc.* 33:1496-1502, 2001
 198. Otsuki T, Maeda S, Lemitsu M, et al: Postexercise heart rate recovery accelerates in strength-trained athletes. *Med.Sci.Sports Exerc.* 39:365-370, 2007
 199. Brown SJ, Brown JA: Resting and postexercise cardiac autonomic control in trained master athletes. *J.Physiol Sci.* 57:23-29, 2007

-
200. Darr KC, Bassett DR, Morgan BJ, et al: Effects of age and training status on heart rate recovery after peak exercise. *Am.J.Physiol* 254:H340-H343, 1988
201. Bompa TO: *Periodization: theory and methodology of training* (ed4). Champaign, IL, Human Kinetics, 1999, pp 3-412
202. Noakes TD, Myburgh KH, Schall R: Peak treadmill running velocity during the VO₂ max test predicts running performance. *J.Sports Sci.* 8:35-45, 1990
203. Hopkins, WG. A spreadsheet for calculating confidence intervals of correlation coefficients. www.sportsci.org/resource/stats/index.html. 2006.
204. Bland JM, Altman DG: Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1:307-310, 1986
205. Gilman MB, Wells CL: The use of heart rates to monitor exercise intensity in relation to metabolic variables. *Int.J.Sports Med.* 14:339-344, 1993
206. Adopted by the 52nd WMA General Assembly, Tokyo 2004. World Medical Association Declaration of Helsinki: Ethical Principles for Medical Research Involving Human Subjects. <http://www.fda.gov/oc/health/helsinki89.html> . 2004.
207. Snyder AC, Kuipers H, Cheng B, et al: Overtraining following intensified training with normal muscle glycogen. *Med.Sci.Sports Exerc.* 27:1063-1070, 1995
208. Urhausen A, Kindermann W: Diagnosis of overtraining: what tools do we have? *Sports Med.* 32:95-102, 2002
209. O'Connor PJ, Crowley MA, Gardner AW, et al: Influence of training on sleeping heart rate following daytime exercise. *Eur.J.Appl.Physiol Occup.Physiol* 67:39-42, 1993
210. Michael E, Evert J, Jeffers K: Physiological changes of teenage girls during five months of detraining. *Med.Sci.Sports* 4:214-218, 1972
-

-
211. Lamberts RP, Lemmink KAPM, Durandt JJ, et al: Variation in heart rate during submaximal exercise: Implications for monitoring training. *J.Strength.Cond.Res.* 18:103-107, 2004
212. Altman DG: *Practical Statistics for Medical Research* (ed1). Chapman and Hall, London, UK, 1991, pp 1-611
213. Brynteson P, Sinning WE: The effects of training frequencies on the retention of cardiovascular fitness. *Med.Sci.Sports* 5:29-33, 1973
214. Houmard JA, Kirwan JP, Flynn MG, et al: Effects of reduced training on submaximal and maximal running responses. *Int.J.Sports Med.* 10:30-33, 1989
215. Mujika I, Padilla S, Pyne D, et al: Physiological changes associated with the pre-event taper in athletes. *Sports Med.* 34:891-927, 2004
216. Weltman A, Seip RL, Snead D, et al: Exercise training at and above the lactate threshold in previously untrained women. *Int.J.Sports Med.* 13:257-263, 1992
217. Daviglius ML, Stamler J, Pirzada A, et al: Favorable cardiovascular risk profile in young women and long-term risk of cardiovascular and all-cause mortality. *JAMA* 292:1588-1592, 2004
218. Okumiya K, Matsubayashi K, Wada T, et al: A U-shaped association between home systolic blood pressure and four-year mortality in community-dwelling older men. *J.Am.Geriatr.Soc.* 47:1415-1421, 1999
219. Avalos M, Hellard P, Chatard JC: Modeling the training-performance relationship using a mixed model in elite swimmers. *Med.Sci.Sports Exerc.* 35:838-846, 2003
220. Skinner JS, Jaskolski A, Jaskolska A, et al: Age, sex, race, initial fitness, and response to training: the HERITAGE Family Study. *J.Appl.Physiol* 90:1770-1776, 2001
221. Macarthur DG, North KN: Genes and human elite athletic performance. *Hum.Genet.* 116:331-339, 2005
-

222. Heck AL, Barroso CS, Callie ME, et al: Gene-nutrition interaction in human performance and exercise response. *Nutrition* 20:598-602, 2004
223. Haff GG: Roundtable Discussion: Periodization of Training - Part 1. *Strength Cond J* 26:50-69, 2004
224. Houglum JE: Asthma medications: Basic pharmacology and use in the athlete. *J.Athl.Train.* 35:179-187, 2000

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CHAPTER 8

APPENDICES

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APPENDIX 1:
EQUATIONS USED TO QUANTIFY EXERCISE INTENSITY:
1.1 Oxygen consumption reserve (VO₂R)

$$\%VO_2R = \frac{VO_{2ex} - VO_{2rest}}{VO_{2max} - VO_{2rest}} \times 100$$

Where: VO_{2ex} is the average oxygen consumption during exercise, VO_{2rest} is oxygen consumption at rest, VO_{2max} is maximal oxygen consumption.

1.2 Heart rate reserve (HRres)

$$\% HRreserve = \frac{HRe_x - HRrest}{HRmax - HRrest} \times 100$$

Where: HRe_x is the average heart rate of the exercise session, HRrest is resting heart rate, HRmax is maximal heart rate.

EQUATIONS USED TO MODEL THE RELATIONSHIP BETWEEN TRAINING AND PERFORMANCE:
1.3 Predicted performance (Banister et al (1991)⁴⁴)

Predicted performance = Fitness – Fatigue

$$a(t) = k_1w(t)e^{-t/\tau_1} - k_2w(t)e^{-t/\tau_2}$$

Where: k₁ and k₂ are weighting factors (initially k₁ = 1 for fitness and k₂ = 2 for fatigue) such that the fitness impulse (p(t) = k₁w(t)) and the fatigue impulse (f(t) = k₂w(t)) can be calculated by multiplying the training impulse (w(t)) by the appropriate weighting factor (k₁ or k₂).

1.4 Influence curves (Fitz-Clarke et al (1991)⁶⁵)

The influence curve is a line representing the effect of a training impulse at any time (t) on performance at a specific future time (t_p):

$$\text{Impulse response } L(\mu) = k_1e^{-\mu/\tau_1} - k_2e^{-\mu/\tau_2}$$

where $\mu = t_p - t$, is time (in days) before performance.

APPENDIX 2:**BORG 6-20 RATING OF PERCEIVED EXERTION SCALE³⁸**

Rating	Description of Perceived Exertion
6	
7	Very very light
8	
9	Very light
10	
11	Fairly light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Very very hard
20	

APPENDIX 3:

BORG CATEGORY RATIO (CR-10) SCALE⁵⁰ and SESSION RPE SCALE⁴⁹

Rating	Description of Perceived Exertion	
	Category Ratio Scale	Session RPE
0	Nothing at all	Rest
0.5	Very, very weak	
1	Very weak	Really easy
2	Weak	Easy
3	Moderate	Moderate
4	Somewhat strong	Sort of hard
5	Strong	Hard
6		
7	Very strong	Really hard
8		
9		Really, really hard
10	Very, very strong	Just like my hardest race
•	Maximal	

Recording of Session RPE score:

Thirty minutes after every training session the participant uses the above scale to give an overall rating of how hard the whole workout was perceived to be, irrespective of whether it was an interval or continuous session.

APPENDIX 4:**TOTAL QUALITY OF RECOVERY (TQR) SCALE¹¹³**

Rating	Description of Perceived Recovery
6	
7	Very, very poor recovery
8	
9	Very poor recovery
10	
11	Poor recovery
12	
13	Reasonable recovery
14	
15	Good recovery
16	
17	Very good recovery
18	
19	Very, very good recovery
20	

Recording of Recovery score:

Every morning the participant uses the above scale to rate how well he/she feels they have recovered over the previous 24 hours (including the previous night's sleep).

APPENDIX 5:**SCORING FOR ACTION RECOVERY¹¹³**

Category	Maximum Points
Nutrition and hydration	10
Sleep and rest	4
Relaxation and emotional support	3
Stretching and active rest	3
Total	20

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APPENDIX 6:

MUSCLE SORENESS ASSESSMENT ANALOGUE SCALE

Full Name Date

NO PAIN UNBEARABLE PAIN

Front thigh at Rest

Front thigh during activities of daily living

Front thigh during stretching

Back thigh at Rest

Back thigh during activities of daily living

Back thigh during stretching

Calf at Rest

Calf during activities of daily living

Calf during stretching

Click and drag each of the pointers to indicate the extent of pain you feel in the muscles identified in the position/actions described on the left

Record: 1 of 1

Recording of muscle soreness assessment:

Every day, at approximately the same time of day, the participant uses the analogue scale interface above (using Microsoft Access) to give an indication of how sore his/her calves, hamstrings and quadriceps feel at rest (sitting), during activities of daily living (e.g. walking around, up and down stairs) and during stretches (e.g. doing prescribed stretches for each of these muscles). The participant clicks on each of the pointers on the left and drags it across each line to a point between the cues “No pain” (on the extreme left) and “Unbearable pain” (on the extreme right) that represents their muscle sensation at that time.

APPENDIX 7: Results from studies investigating the effect of training on HRV showing the disparity in findings, methodology of recording HRV and prescribed training programmes.

Research paper:		Bonaduce et al ¹²⁸		Levy et al ¹³⁰	Tulppo et al ¹⁶⁰	Iwasaki et al ¹⁶¹	al Ani et al ¹⁶²	Loimaala et al ¹⁶³	Carter et al ¹⁶⁴	Melanson & Freedson ¹³⁶
Type of study		longitudinal	cross-section (detrained vs. untrained)	longitudinal	longitudinal	longitudinal	longitudinal	longitudinal	longitudinal	longitudinal
HR Variability - Time Domain		no change	↑	↑	↑(mean SDNN)	SDNN ↑ at 3 & 6 months, decr at 9 & 12 months	↑ in 9 of 11 subjects, ↓ in 2 (difference between max & min R-R intervals in 1 respiratory cycle)	no change (SDNN & pNN50)	↑ (SDNN)	↑ by week 12 (pNN50 & rMSSD)
HR Variability-Frequency Domain	HFP	no change	↑	↑(absolute spectral value)	↑ in mean of normalized HF & absolute spectral value	no change	absolute spectral value ↑ in 9 of 11 subjects, ↓ in 2	no change	↑	↑ by week 12
	LFP	no change	↑	No information provided	↓ in mean of normalized LF, incr in absolute spectral value	↑ at 3 & 6 months, ↓ at 9 & 12 months	↑	no change	↓	no change
	LFP:HFP	no change	↓	No information	↓	No information	No information	No information	↑	No information
Normalization		No information provided	No information provided	No information provided	normalized by the average R-R interval (CCV%)	HFP & LFP normalized by dividing by total spectral power	No information provided	No information provided	No information provided	No information provided
Duration of measurement		24hr	24hr	rest = 30min, ex = last 2 min of each stage)	24hr	6 min	10min	24hr	supine = 10min, ex = 7min	10 min
Activity during measurement		daily activity & sleep		supine rest & incremental exercise	daily activity & sleep	supine	semi-supine	daily activity & sleep	supine rest & submaximal exercise	supine
Breathing control		No information	No information	No information	No information	12 breaths/min	controlled at own frequency		15 breaths/min	10 breaths/min
Duration of training		5 months	1 mo detraining	6 mo	8 wks	1 yr	6 wks	5 months	12 wks (2x 4wk training, + 2wk taper)	16 wks
Exercise frequency		5x / wk	5x / wk	4-5x / wk	6x / wk	incr over time	daily	control = max 2x / wk, ex groups 1 & 2 = 4-6x / wk	4x / wk	3x / wk
Exercise bout intensity		No information provided	No information provided	incr from 50-60%HRreserve to 80-85%HRreserve	70-80%HRmax	75-85%HRmax	intensity increased to match HR to 85%HRmax	ex grp 1 = 55%VO ₂ max ex grp 2 = 75%VO ₂ max	70-90%HRmax	70%HRreserve in 1 st 2 wks, 80%HRreserve for rest of study
Exercise bout duration		4hr	1hr	45min	mod-intensity grp = 30min high-int grp = 60min	incr from 30-45min 3-4x / wk to 7-9hrs / wk	25min	min 30min at prescribed HR	45-60 min (2 nd 4wk training incr by 20%)	30 min

**APPENDIX 8:
MODIFIED PHYSICAL ACTIVITY READINESS QUESTIONNAIRE**

Name: _____ **Date:** _____

Date of birth: _____ **Age:** _____

E-mail: _____ **Tel:** _____

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

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

If you answered yes to any of the above questions, talk with your doctor BEFORE you become more physically active. Tell your doctor of your intent to exercise and to which questions you answered yes. If you answered no to all questions, you can be reasonably positive that you can safely increase your physical activity gradually. If your health changes so you then answer yes to any of the above questions, seek guidance from a physician.

Participant signature: _____ **Date:** _____

APPENDIX 9:

INJURY AND ILLNESS REPORT

Date: _____

Type of injury/ illness: _____

Area of body (e.g. head/ neck/ trunk/ upper limb/ lower limb): _____

Severity (enter 1 of the following): _____

- 1) Injury/ illness caused discomfort **AFTER** exercise & may have been felt for some time, but did not affect training.
- 2) Injury/ illness caused discomfort **DURING** exercise, but training continued and did not reduce performance.
- 3) Injury/ illness caused more severe discomfort/ pain that **LIMITED** training and performance.
- 4) Injury **PREVENTED** training.

If you entered 1, 3 or 4 for “Severity” provide a timeframe for the injury/ illness (e.g. minutes/ hours/ days): _____

Was any specific/medical treatment required? _____

If Yes, please give details (e.g. bed rest/ supportive brace/ medication/ surgery etc)

LIST OF TABLES

CHAPTER 2:

Table 2.1: Subject characteristics (n = 29; 12 males, 17 females). Group I are the subjects who under-estimated training, group D over-estimated training and group S accurately assessed their training. Data are expressed as mean \pm SD. 68

CHAPTER 3:

Table 3.1: Within-subject correlations between TRIMP and Session RPE method, and between Summated HR Zone and Session RPE method. The number of exercise sessions included in the calculation of the correlation coefficient (r) for each subject is presented. 81

Table 3.2: Subject characteristics for athletes in OVER (n = 6), UNDER (n = 4) and ACCURATE (n = 23) groups, in the correlation between the Session RPE method and the TRIMP method. Groups were based on whether the regression equations over- (OVER), under- (UNDER) or accurately predicted (ACCURATE) the relationship between the methods (pooled data). Data are expressed as mean \pm SD. 82

Table 3.3: Subject characteristics for OVER (n = 5), UNDER (n = 4) and ACCURATE (n = 24) groups, for the correlation between the Session RPE method and the Summated Heart Rate Zones method. Groups were based on whether the regression equations over- (OVER), under- (UNDER) or accurately predicted (ACCURATE) the relationship between the methods (pooled data). Data are expressed as mean \pm SD. 85

CHAPTER 4:

Table 4.1: Subject characteristics. Data are expressed as mean \pm SD. 103

Table 4.2: Changes in absolute heart rate, heart rate recovery (HRr) and percent heart rate recovery (HRr%) during the standardized submaximal running (HIMS) test at week 1 and 2 for Group I, Group S and Group D. Data are expressed as mean \pm SD. 107

CHAPTER 5:

Table 5.1: Average changes in performance and physiological measures after 11 weeks of running training. Values are expressed as mean (\pm SD).	124
Table 5.2: Subject characteristics and physiological changes for Participant 1 after 11 weeks of running training.	127
Table 5.3: Subject characteristics and physiological changes for Participant 2 after a running training programme.	131
Table 5.4: Subject characteristics and physiological changes for Participant 3 after a running training programme.	135
Table 5.5: Subject characteristics and physiological changes for Participant 4 after a running training programme.	139
Table 5.6: Subject characteristics and physiological changes for Participant 5 after a running training programme.	143
Table 5.7: Subject characteristics and physiological changes for Participant 6 after a running training programme.	147
Table 5.8: Subject characteristics and physiological changes for Participant 7 after a running training programme.	151
Table 5.9: Subject characteristics and physiological changes for Participant 8 after a running training programme.	155
Table 5.10: Subject characteristics and physiological changes for Participant 9 after a running training programme.	159
Table 5.11: Subject characteristics and physiological changes for Participant 10 after a running training programme.	163
Table 5.12: Subject characteristics and physiological changes for the Ironman Participant after 32 weeks of prescribed training.	177

LIST OF FIGURES

CHAPTER 1:

Figure 1.1: The relationship between percent maximal oxygen consumption (%VO₂max) and percent maximal workload (%Wmax). 18

Figure 1.2: Schematic representation of the contributions of the parasympathetic and sympathetic nervous systems to heart rate at rest and during increasing intensities of exercise. 22

Figure 1.3: The relationship of the multiplying factor (Y) to increases in heart rate based on the pattern of increased blood lactic acid accumulation during exercise. Adapted from Banister (1991)⁴³. 25

Figure 1.4: Schematic representation of the accumulation and decay of the fitness impulse and fatigue impulse in response to training impulses. The contribution of a training impulse to fatigue is shown proportionally larger than to fitness however the decay time constant of fitness is longer. The difference between the fitness impulse and the fatigue impulse represents predicted performance. Adapted from Banister (1991)⁴³. 31

Figure 1.5: Schematic representation of the General Adaptation Syndrome, depicting three stages of change in the degree of resistance of an organism to a stressor. Adapted from Selye (1974)⁶⁷. 36

Figure 1.6: Heart rate variability can be represented in a time domain in which R-R intervals (ms) and the differences between adjacent R-R intervals are calculated. Adapted from Achten and Jeukendrup (2003)²⁶. 49

Figure 1.7: Heart rate variability represented in the frequency domain, which is the frequency at which the length of the R-R interval changes. Adapted from Achten and Jeukendrup (2003)²⁶. 50

CHAPTER 2:

Figure 2.1: Relationship between self-reported training duration (min.wk⁻¹) and measured training duration (min.wk⁻¹) (n = 29). 69

Figure 2.2: Bland Altman plot of self-reported and measured training duration (n = 29). 70

CHAPTER 3:

Figure 3.1: Relationship between Total Training Load calculated using TRIMP and the Summated HR Zones method. 79

Figure 3.2: Relationship between Total Training Load calculated using TRIMP and the Session RPE method. Groups based on whether the regression equations over-predicted (OVER), under-predicted (UNDER) or accurately predicted (ACCURATE) the relationship between methods. 80

Figure 3.3: Relationship between Total Training Load calculated using the Summated Heart Rate Zone method and the Session RPE method. Groups were based on whether the regression equations over-predicted (OVER), under-predicted (UNDER) or accurately predicted (ACCURATE) the relationship between the methods. 84

CHAPTER 4:

Figure 4.1: Schematic of 2-week study protocol and measurements taken during this period. Training load calculated using TRIMP, HIMS = submaximal shuttle running test, HRr% = 1-minute recovery heart rate expressed as a percent of the heart rate reached at the end of the fourth running stage of the HIMS test, HRS4% = Heart rate at the end of the fourth running stage of the HIMS test expressed as a percent of maximal heart rate. 99

Figure 4.2: Heart rate Interval Monitoring System (HIMS) Test protocol, showing running speeds and durations, interspersed with rest periods. A typical heart rate response to this submaximal test is also shown ($\text{b}\cdot\text{min}^{-1}$), with Stage 4 heart rate and 1-minute heart rate recovery values identified. 101

Figure 4.3: (A) Relationship between TRIMP calculated for week 1 and week 2 and (B) percent change in weekly TRIMP (mean \pm SD) from week 1 to week 2 for Group I (n = 9), Group S (n = 11) and Group D (n = 8). * Group S significantly different from Group I and Group D, and Group I significantly different from Group D ($p < 0.05$). 104

Figure 4.4: Relationship between TRIMP (AU) and Heart rate recovery (%) for (A) week 1, (B) week 2, and (C) the average for the 2 weeks, in Group S (n = 11). 105

Figure 4.5: Percent change in (A) heart rate recovery (HRr%) and (B) stage 4 heart rate (HRS4%) (mean \pm SD) from HIMS 1 (end of week 1) to HIMS 2 (end of week 2) in Group S, Group I and Group D. HRr% = 1-minute recovery heart rate expressed as a percent of the heart rate reached at the end of the fourth running stage of the HIMS test. HRS4% = Heart rate at the end of the fourth running stage of the HIMS test expressed as a percent of maximal heart rate. * Group D significantly different from Group I ($p < 0.05$). 108

CHAPTER 5:

- Figure 5.1:** Relationship between percent change in heart rate recovery (HRr%) after HIMS tests performed at 2-week intervals and accumulated TRIMP for the 2-week periods between HIMS tests. HRr% = 1-minute recovery heart rate expressed as a percent of the heart rate reached at the end of the fourth running stage of the HIMS test. 125
- Figure 5.2:** Percent change in heart rate recovery (HRr%, mean \pm SD) between adjacent HIMS tests performed every 2 weeks after an increase, decrease or no change in TRIMP accumulated over the 2-week period between HIMS tests. 126
- Figure 5.3:** Changes in physiological and outcome measures for Participant 1 after 11 weeks of running training. A - Submaximal heart rate percent (\circ) and heart rate recovery percent (\bullet). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (\square), Hamstring (\times) and Calf (\bullet). D - Monotony accumulated over 2-week periods between HIMS tests. E - TRIMP (AU) accumulated over 2-week periods between HIMS tests. F - TRIMP (AU) for each training session. 129
- Figure 5.4:** Changes in physiological and outcome measures for Participant 1 after 11 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F - TRIMP (AU) for each training session. 130
- Figure 5.5:** Changes in physiological and outcome measures for Participant 2 after 11 weeks of running training. A - Submaximal heart rate percent (\circ) and heart rate recovery percent (\bullet). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (\square), Hamstring (\times) and Calf (\bullet). D - Monotony accumulated over 2-week periods between HIMS tests. E - TRIMP (AU) accumulated over 2-week periods between HIMS tests. F - TRIMP (AU) for each training session. 133
- Figure 5.6:** Changes in physiological and outcome measures for Participant 2 after 11 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F - TRIMP (AU) for each training session. 134
- Figure 5.7:** Changes in physiological and outcome measures for Participant 3 after 10 weeks of running training. A - Submaximal heart rate percent (\circ) and heart rate recovery percent (\bullet). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (\square), Hamstring (\times) and Calf (\bullet). D - Monotony accumulated over 2-week periods between HIMS tests. E - TRIMP (AU) accumulated over 2-week periods between HIMS tests. F - TRIMP (AU) for each training session. 137

- Figure 5.8:** Changes in physiological and outcome measures for Participant 3 after 10 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F – TRIMP (AU) for each training session. 138
- Figure 5.9:** Changes in physiological and outcome measures for Participant 4 after 11 weeks of running training. A - Submaximal heart rate percent (◯) and heart rate recovery percent (●). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (◻), Hamstring (×) and Calf (●). D – Monotony accumulated over 2-week periods between HIMS tests. E – TRIMP (AU) accumulated over 2-week periods between HIMS tests. F – TRIMP (AU) for each training session. 141
- Figure 5.10:** Changes in physiological and outcome measures for Participant 4 after 11 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F – TRIMP (AU) for each training session. 142
- Figure 5.11:** Changes in physiological and outcome measures for Participant 5 after 10 weeks of running training. A - Submaximal heart rate percent (◯) and heart rate recovery percent (●). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (◻), Hamstring (×) and Calf (●). D – Monotony accumulated over 2-week periods between HIMS tests. E – TRIMP (AU) accumulated over 2-week periods between HIMS tests. F – TRIMP (AU) for each training session. 145
- Figure 5.12:** Changes in physiological and outcome measures for Participant 5 after 10 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F – TRIMP (AU) for each training session. 146
- Figure 5.13:** Changes in physiological and outcome measures for Participant 6 after 11 weeks of running training. A - Submaximal heart rate percent (◯) and heart rate recovery percent (●). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (◻), Hamstring (×) and Calf (●). D – Monotony accumulated over 2-week periods between HIMS tests. E – TRIMP (AU) accumulated over 2-week periods between HIMS tests. F – TRIMP (AU) for each training session. 149
- Figure 5.14:** Changes in physiological and outcome measures for Participant 6 after 11 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F – TRIMP (AU) for each training session. 150

- Figure 5.15:** Changes in physiological and outcome measures for Participant 7 after 11 weeks of running training. A - Submaximal heart rate percent (◯) and heart rate recovery percent (●). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (◻), Hamstring (×) and Calf (●). D – Monotony accumulated over 2-week periods between HIMS tests. E – TRIMP (AU) accumulated over 2-week periods between HIMS tests. F – TRIMP (AU) for each training session. 153
- Figure 5.16:** Changes in physiological and outcome measures for Participant 7 after 11 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F – TRIMP (AU) for each training session. 154
- Figure 5.17:** Changes in physiological and outcome measures for Participant 8 after 10 weeks of running training. A - Submaximal heart rate percent (◯) and heart rate recovery percent (●). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (◻), Hamstring (×) and Calf (●). D – Monotony accumulated over 2-week periods between HIMS tests. E – TRIMP (AU) accumulated over 2-week periods between HIMS tests. F – TRIMP (AU) for each training session. 157
- Figure 5.18:** Changes in physiological and outcome measures for Participant 8 after 10 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F – TRIMP (AU) for each training session. 158
- Figure 5.19:** Changes in physiological and outcome measures for Participant 9 after 11 weeks of running training. A - Submaximal heart rate percent (◯) and heart rate recovery percent (●). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (◻), Hamstring (×) and Calf (●). D – Monotony accumulated over 2-week periods between HIMS tests. E – TRIMP (AU) accumulated over 2-week periods between HIMS tests. F – TRIMP (AU) for each training session. 161
- Figure 5.20:** Changes in physiological and outcome measures for Participant 9 after 11 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F – TRIMP (AU) for each training session. 162
- Figure 5.21:** Changes in physiological and outcome measures for Participant 10 after 11 weeks of running training. A - Submaximal heart rate percent (◯) and heart rate recovery percent (●). B - Total Quality of Recovery (0-20 scale). C - Average muscle soreness (%) in Quadriceps (◻), Hamstring (×) and Calf (●). D – Monotony accumulated over 2-week periods between HIMS tests. E – TRIMP (AU) accumulated over 2-week periods between HIMS tests. F – TRIMP (AU) for each training session. 165

Figure 5.22: Changes in physiological and outcome measures for Participant 10 after 11 weeks of running training. G - Absolute heart rate recovery (beats). H - Weekly Monotony. I - Weekly TRIMP (AU). F – TRIMP (AU) for each training session. 166

Figure 5.23: Changes in physiological and outcome measures for the Ironman Participant after 32 weeks of triathlon training. A - Submaximal heart rate percent (◦) and heart rate recovery percent (●). B - Absolute heart rate recovery (beats). C - Average muscle soreness (%) in Quadriceps (◻), Hamstring (×) and Calf (●). D - Weekly Monotony. E – Weekly TRIMP (AU). F – TRIMP (AU) for each training session. 179

CHAPTER 6:

Figure 6.1: Summary of some of the main findings in Chapter 4 and 5 in the context of the General Adaptation Syndrome (Selye, 1974). HRr = heart rate recovery 193