

**TIME COURSE OF PERFORMANCE CHANGES AND FATIGUE  
MARKERS DURING TRAINING FOR THE IRONMAN TRIATHLON**

**BY**

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**THESIS**

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## ABSTRACT

Suboptimal preparation for the Ironman triathlon can have detrimental effects on mental and physical condition. The purpose of this longitudinal investigation was to examine the relationship between a number of performance changes and fatigue markers during training for an Ironman as well as immediately after the event, in an attempt to better understand the effects of ultraendurance training. Eighteen athletes training for the Ironman; South Africa, 2009 were recruited for the study. Over the 6 month data collection period body mass, training load (TRIMP and Session x RPE methods), physiological responses (waking heart rate, postural dizziness, sleep ratings), changes in psychological state (profile of mood states - POMS), reported immunological responses (symptoms of illness), biochemical changes (salivary cortisol and alpha amylase) and performance (8 km submaximal running time trial (TT) and race day performance) were measured. These responses were compared to a control sample (n=15). Results show a significant increase ( $p<0.05$ ) in training load ( $3899.4 \pm 2517.8$ ) four weeks prior to the event. Fatigue scores significantly increased ( $p<0.05$ ) concurrently with this significant increase ( $p<0.05$ ) in training. TT performance did not significantly ( $p<0.05$ ) alter during the time course of training. It was however strongly correlated to training load ( $R^2=0.85$ ) and modestly related to race performance ( $R^2=0.65$ ). The signs and symptoms of upper respiratory tract infections (URTI) were prevalent during the training period, decreasing during the taper and race period. Large standard deviations were found within the majority of the responses. During the final two weeks of preparation, tension scores were significantly increased ( $p<0.05$ ) while training load significantly decreased ( $p<0.05$ ) during the final week of preparation. Cortisol increased significantly ( $p<0.05$ ) immediately post race ( $0.507 \pm 0.15 \mu\text{g} \cdot \text{DL}^{-1}$ ) and 1.5 hours later ( $0.796 \pm 0.23 \mu\text{g} \cdot \text{DL}^{-1}$ ). Overall the results indicate that the POMS questionnaire was a sensitive marker of fatigue and stress associated with ultraendurance training, and that the event itself placed a great deal of stress on the athletes which was illustrated by the post event measures.

## **DEDICATION**

This research is dedicated to my family: Ray, Anne-Marie, Paula and Chris, without their love and support this would never have been possible.

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## CHAPTER I

### INTRODUCTION

#### BACKGROUND TO THE STUDY

Triathlon is a unique and rapidly growing sport consisting of three disciplines: swimming, cycling and running which are performed sequentially (Bently **et al.**, 2007). They are held over varying distances including sprint distance (750m swim/20km bike/5km run) Olympic distance (1500m swim/40km bike/10km run), long distance (2000m swim/80km bike/20km run), and ultra distance including the half Ironman distance (1900m swim/90.1km bike/21.1km run) and the full Ironman distance (3800m swim/180.2km bike/42.2km run) (McHardy **et al.**, 2006). The Ironman triathlon, a classic ultraendurance competition, represents the 'Everest' of triathlon achievement, being the focus of this study.

Although all triathlon events require athletes to compete in three very different disciplines, the challenge of the full Ironman is the vast distances the athletes have to complete in each of these events. Thus the demands placed on them before the allotted 17 hour cut off are tremendous (Bently **et al.**, 2007). However, it is the author's opinion that the most excessive demands placed on these athletes are during the lead up to the event which requires sizable amounts of training, specifically high volumes and an extensive and prolonged training period (Douglas, 1989). While Ironman competitions have received some research focus, a void in research is evident and considerably more research is needed to fully understand the efforts required of these athletes to successfully complete such an event.

While some research has attempted to identify optimal levels of training, most has focused on the phases immediately prior to competition, during competition

and immediately post competition. Considerably less is known about the stress impact of the high training volumes in the many months of training prior to the competition phase. This research therefore sought to place emphasis on, not only the competition and post-competition phase, but also the many phases of training, starting from base-phase training. The study took a longitudinal stance monitoring athletes as early as six months prior to competing in the Ironman South Africa held in Port Elizabeth in April 2009. For many athletes taking part, the rough approximation of the minimum training necessary in order to successfully complete the event has been instructive (Gublin and Gaffney, 1999). In contrast, elite top end finishers and their coaches, who have not been adequately educated on training techniques, try 'out do' their competition by increasing their training load and/or volume to perform at a higher level. As a result, there is a vast range of training regimes found amongst athletes. This highlights the need to investigate a broad range of athletes, from novice to elite, as well as similar performers at all levels, which have very different training loads.

A correctly formulated training regime focused on the systematic practice of a specific exercise (Kuipers, 1987) is essential for improving sporting performance (Smith, 2003). The cardinal rule of basic training adaptation is that performance improvements are achieved by a progressive increase in this sport-specific training stimulus (Rowbottom **et al.**, 1997). Athletes and coaches use the process of 'overload training' which involves sport-specific exercise stress, to provide the alterations or disturbances necessary for adaptation (Smith, 2000). The primary problem with this concept is that many athletes, recreational and/or elite are prone to training at excessively high intensities, durations and/or frequencies with insufficient recovery periods incorporated into their training schedules (Lehmann **et al.**, 1997). Recovery periods are essential as it is during this time when the physiological, muscular and psychological disturbances caused by endurance exercise training are corrected (Virus, 1994). Non-training specific stress factors and exercise tolerance identify the fact that athletes have

different vulnerabilities when performing the same training-related stress factors (Lehmann **et al.**, 1993).

Too little training and competition experience will result in athletes who will be unable to perform at their optimal ability, or who will not be able to complete the event. Therefore, undertraining will likely result in the concerned athletes performing at a reduced performance capability (Neufer, 1989). Further these athletes may not make relevant 'cut-off' times in one, or more, of the three disciplines ending their race prematurely, or after the final cut-off. Although this study did not focus exclusively on overtraining it was expected that most of the athletes would have high training volumes and therefore would be more prone to excessive overload rather than under load. At the overload end of the spectrum, insufficient recovery periods during training will lead to an accumulation of fatigue, which in turn can lead to overtraining and performance decrements (Fry **et al.**, 1991).

The broad term, overtraining, can be divided into different levels of severity which include functional/short term over-reaching, non-functional/extreme over-reaching and in the most severe cases, the overtraining syndrome (OTS) (Coutts **et al.**, 2007). Training programs commonly involve an intentional induction of short term overtraining or functional over-reaching (Fry **et al.**, 1991). The fatigue caused by this form of over-reaching can be reversed by a reduction in training or a small number of days of rest, resulting in an enhanced endurance performance capacity (Jeukendrup **et al.**, 1992; Snyder **et al.**, 1993). If adequate recovery and taper periods are not permitted during an endurance training schedule, functional over-reaching becomes non-functional, causing a wide range of performance reducing symptoms including abnormal hormonal regulation, psychological disturbances, reduced immune system function and sleep disorders (Coutts **et al.**, 2007). If the exposure of these athletes to such a high training stress continues, it is probable that it will result in staleness, burnout or the overtraining syndrome (Lehmann **et al.**, 1993). The symptoms of overtraining include

increased waking pulse rate, 'heavy legs', decreased motivation, due to a decreased enjoyment of exercise, musculoskeletal pain, sleep related disorders, depression, a feeling of dizziness upon standing, dramatic weight loss, increased prevalence of infections, decreased sexual drive and an increase in the level of effort required to perform exercise training (Hooper and McKinnon, 1993; Eichner, 1995).

Overtraining has been extensively researched (Kuipers and Keizer, 1988; O'Toole, 1989; Fry **et al.**, 1991; Hooper and McKinnon, 1993; Eichner, 1995; Derman **et al.**, 1996; Rowbottom **et al.**, 1997; Birch and George, 1998; Urhausen and Kindermann, 2002 and Coutts **et al.**, 2007) yet despite this, there is still a lack of understanding of relevant and appropriate diagnostic tools (Urhausen **et al.**, 1995). The tools which have been propounded for the diagnosis of the OTS, and more importantly the early indicators of the onset of the OTS, are confounding due to the multiple symptoms of the syndrome (Fry **et al.**, 1991). However the 'gold standard' of overtraining remains a decrement in sports-specific performance. It is contended that diagnostic measurement parameters that are inexpensive and practical to administer during rest or during submaximal exercise would be preferable due to the limited influence that they have on their training routine (Urhausen and Kindermann, 2002). The challenge is therefore to uncover the most suitable markers of excessive bodily stress which are not only noninvasive, but cost efficient. It is therefore important to consider tests that cause minimal interruption to the training process, as well as reduce any additional stress placed on the athletes.

Therefore measurements that can be performed at rest, due to their limited interference, were utilized for this study. These tests include heart rate and body mass changes, sleep related disorders, signs and symptoms of illness, psychological measures such as the profile of mood states (POMS) and subjective complaints as well as enzyme, metabolic and hormonal markers found in salivary samples (Urhausen and Kindermann, 2002). Hormonal analysis, in the

past, has been problematic due to the invasive nature of blood and urine sampling as well as the logistical issues concerned with sample collection. However, due to advancements in technology, the use of saliva/drool sampling for the biochemical analyses of hormones has become a viable and non-invasive option (Levine **et al.**, 2007). While there are a number of subjective symptoms of overtraining, hormone analyses has the ability to objectively quantify the effect of somatic, psychological and physical stresses on the body (Linas **et al.**, 1980), therefore making it a useful tool for the quantification of bodily stress associated with high levels of training.

It would be preferable to be able to measure all diagnostic markers of inappropriate training at rest. However decreases in sport-specific performance still remains the best diagnostic overtraining tool as it highlights the stress that the training undertaken places on the athlete (Urhausen and Kindermann, 2002). A decrease in performance shows excessive stress to the relevant neuromuscular and cardiovascular systems. Submaximal time trial tests can be useful to assess performance decrements and assist in the prevention of the OTS (Coutts **et al.**, 2007b). Furthermore as it is not viable to make an accurate and reliable prediction of stress, fatigue and performance from a once off measurement, a longitudinal follow-up of these markers was considered more accurate and valid (Lac and Maso, 2004). Due to the interrelationship of the bodily systems as well as the overlapping symptoms of over-reaching (functional and non-functional) and overtraining, it is probable that there may never be one definitive diagnostic parameter of the overtraining syndrome (Fry **et al.**, 1991). However the identification of a number of 'key' parameters measured during the long training period would allow for a practical method of diagnosis for athletes and coaches alike. Assess a wide range of athletes training for an Ironman event can be beneficial to gain insight into a wide range of athlete performance levels.

## **STATEMENT OF THE PROBLEM**

While there has been an increased participation in ultraendurance sporting events, such as the Ironman, there has been little research focus placed on the impact of training for events of this nature. Further, there has been little improvement in the diagnostic tools available for the longitudinal analyses of the signs and symptoms of inappropriate training. Likewise, many studies have focused on either the physiological or psychological measures while, in this study, a holistic approach was adopted in order to quantify the physiological, psychological, hormonal, immunological, and performance changes during a longitudinal analysis of the different phases of training for an ultraendurance Ironman triathlon as well as the immediate and post competition phase.

## **RESEARCH HYPOTHESIS**

It was expected that during training in the months prior to the ultraendurance triathlon, Ironman, in Port Elizabeth, South Africa (2009), the physiological, psychological, performance and hormonal parameters would be affected by changes in training load. It was further expected that performance changes would be affected by alterations in training during the different phases of competition preparation. Additional changes were expected during the competition period itself.

## **STATISTICAL HYPOTHESIS**

1. The first null hypothesis proposed is that (a) the physiological responses, (b) the psychological responses, (c) the immunological responses, (d) the stress hormone responses Cortisol( $x$ ) and (e) Alpha amylase( $y$ ), and (f) the performance responses of the athletes will remain unchanged

during training for the Ironman competition, surrounding the competition and after the competition.

(a)  $H_0: \mu \text{PHY}_{(1)} = \mu \text{PHY}_{(2)} = \mu \text{PHY}_{(3)} = \mu \text{PHY}_{(4)} = \mu \text{PHY}_{(5)} = \mu \text{PHY}_{(6)}$

$H_a: \mu \text{PHY}_{(1)} \neq \mu \text{PHY}_{(2)} \neq \mu \text{PHY}_{(3)} \neq \mu \text{PHY}_{(4)} \neq \mu \text{PHY}_{(5)} \neq \mu \text{PHY}_{(6)}$

(b)  $H_0: \mu \text{PSY}_{(1)} = \mu \text{PSY}_{(2)} = \mu \text{PSY}_{(3)} \dots = \mu \text{PSY}_{(24)}$

$H_a: \mu \text{PSY}_{(1)} \neq \mu \text{PSY}_{(2)} \neq \mu \text{PSY}_{(3)} \dots \neq \mu \text{PSY}_{(24)}$

(c)  $H_0: \mu \text{HORx}_{(1)} = \mu \text{HORx}_{(2)} = \mu \text{HORx}_{(3)} = \mu \text{HORx}_{(4)} = \mu \text{HORx}_{(5)} = \mu \text{HORx}_{(6)} = \mu \text{HORx}_{(7)} = \mu \text{HORx}_{(8)} = \mu \text{HORx}_{(9)}$

$H_a: \mu \text{HORx}_{(1)} \neq \mu \text{HORx}_{(2)} \neq \mu \text{HORx}_{(3)} \neq \mu \text{HORx}_{(4)} \neq \mu \text{HORx}_{(5)} \neq \mu \text{HORx}_{(6)} \neq \mu \text{HORx}_{(7)} \neq \mu \text{HORx}_{(8)} \neq \mu \text{HORx}_{(9)}$

(d)  $H_0: \mu \text{HORY}_{(1)} = \mu \text{HORY}_{(2)} = \mu \text{HORY}_{(3)} = \mu \text{HORY}_{(4)} = \mu \text{HORY}_{(5)} = \mu \text{HORY}_{(6)} = \mu \text{HORY}_{(7)} = \mu \text{HORY}_{(8)} = \mu \text{HORY}_{(9)}$

$H_a: \mu \text{HORY}_{(1)} \neq \mu \text{HORY}_{(2)} \neq \mu \text{HORY}_{(3)} \neq \mu \text{HORY}_{(4)} \neq \mu \text{HORY}_{(5)} \neq \mu \text{HORY}_{(6)} \neq \mu \text{HORY}_{(7)} \neq \mu \text{HORY}_{(8)} \neq \mu \text{HORY}_{(9)}$

(e)  $H_0: \mu \text{PER}_{(1)} = \mu \text{PER}_{(2)} = \mu \text{PER}_{(3)} = \mu \text{PER}_{(4)} = \mu \text{PER}_{(5)}$

$H_a: \mu \text{PER}_{(1)} \neq \mu \text{PER}_{(2)} \neq \mu \text{PER}_{(3)} \neq \mu \text{PER}_{(4)} \neq \mu \text{PER}_{(5)}$

2. The second null hypothesis proposed is that the responses from the athletic sample will be the same as those of the control sample.

(a)  $H_0: \mu \text{PHY}_{(\text{CON})} = \mu \text{PHY}_{(\text{A1-A6})}$

$H_a: \mu \text{PHY}_{(\text{CON})} \neq \mu \text{PHY}_{(\text{A1-A6})}$

(b)  $H_0: \mu \text{PSY}_{(\text{CON})} = \mu \text{PSY}_{(\text{A1-A24})}$

$H_a: \mu \text{PSY}_{(\text{CON})} \neq \mu \text{PSY}_{(\text{A1-A24})}$

(c)  $H_0: \mu \text{HORx}_{(\text{CON})} = \mu \text{HORx}_{(\text{A1-A9})}$

$H_a: \mu \text{HORx}_{(\text{CON})} \neq \mu \text{HORx}_{(\text{A1-A9})}$

(d)  $H_0: \mu \text{HORY}_{(\text{CON})} = \mu \text{HORY}_{(\text{A1-A9})}$

$H_a: \mu \text{HORY}_{(\text{CON})} \neq \mu \text{HORY}_{(\text{A1-A9})}$

Where:

- PHY = Physiological responses (Resting and exercising heart rate)
- PSY = Psychological responses (Sleep ratings, subjective comments, Profile of Mood States, training effort and muscle discomfort ratings)
- HORx = Hormonal responses (Salivary Cortisol)
- HORY = Hormonal responses (Salivary Alpha Amylase)
- PER = performance responses (Submaximal time-trial results)
- CON = control group (age and gender matched sedentary control group)
- A = athlete group (Ironman athletes used for the project)
- (#) = Sampling period defined as by the Appendix C

## **DELIMITATIONS OF THE STUDY**

The study was delimited to 32 subjects (18 athletes, 15 controls) aged 24-61 years. The athletes were training for the Ironman ultraendurance triathlon in Port

Elizabeth, South Africa in April 2009. Experience in triathlons and the length of training period prior to the event was not considered a limiting factor. The primary focus of the project was the investigation of the relationship between training load and a number of physiological, psychological, hormonal and performance-based markers, which could aid in the early identification of the signs and symptoms of suboptimal training. Training programs were independently selected by the athletes/coaches involved, and the author did not produce or influence training regimes partaken by the athletes. A control sample of 15 aged and gender matched sedentary (defined by the ACSM's guidelines (Balady **et al.**, 2000)) individuals were used for comparative purposes. As prior participation in Ironman events was not a limiting factor, a number (n=5) of the subjects were novices.

Dependent variables examined included: a number of daily subjective complaints pertaining to sleep ratings and perceived training ratings; the Profile of Mood States (POMS); salivary alpha-amylase and cortisol hormonal changes; and performance data from a submaximal running time trial. The longitudinal nature of the project required the experimental group to commit to a maximum of six months of data collection and compliance to the data collection procedures was stressed. The age matched sedentary control group (n=15) was recruited to make comparisons to the athlete sample. This control took part in data collection for a one month period (April). The data obtained for the control group included information from the daily log and a weekly POMS questionnaire and a weekly salivary hormone collection. This control group was limited do to poor subject participation and drop outs from the study.

## **LIMITATIONS OF THE STUDY**

Due to the limitations of available and willing subject numbers and the difficulty in attaining a large sample, the subjects were not randomly selected from the Ironman athlete population. This competition experience and the training performed therefore limited the validity of the results found, however, while these

subjects were of a wide range of ages and performance abilities, this did increase the scope of the study.

The longitudinal, hands-off nature of the project inevitably minimised control over possible extraneous variables which could have affected the results of the study. This included the reliability of the completion of the daily training log, POMS questionnaire, as well as the sampling of the salivary hormones. However due to the committed nature of the sample of athletes being tested and the numerous previous habituation and orientation briefings, the effect of these external variables would have been limited. Constant contact (telephonic, email, short message) was maintained with the subjects throughout the data collection, this ensured that subjects did not become complacent with recording data. Due to the less committed nature of the control sample, the entire group was based in Grahamstown to ensure data was correctly and successfully recorded.

The national distribution of the athletes involved in the project (Johannesburg, Port Elizabeth and Grahamstown) limited the control the author had over the logistics concerning the drool samples. While the Port Elizabeth samples were collected by the author, the Johannesburg samples were moved from their collection point to their storage site in Pretoria by a specialised logistics company. While defined instruction was given to this company regarding sample handling, no guarantee could be ensured over compliance to these guidelines, perhaps affecting the results of the hormone analyses.

While the age and sex of the two samples (athlete and control) were matched, the stature and mass range was not. This could possibly limit the reliability of comparing two samples due to a difference in, for example, body composition.

Due to injury, financial constraints, and personal reasons a large number of the subjects pulled out of the Ironman competition and therefore the research project during its course. This could not be controlled, although, additional subjects were recruited, where possible, to increase the subject numbers.

Financial constraints limited the frequency of the hormone sampling. An increase in funds would have seen more frequent sampling of the hormones, increasing the reliability of the hormone analyses.

There were a large number of limitations concerning the hormone analyses during the project. While the training phase samples were performed following an overnight fast, the post event samples were limited by the logistical inability to prescribe nutritional intake during the event, which could have impacted the hormonal results, recovery and performance. These are however standard with any hormone sampling. Qualified and experienced guidance and assistance while performing the alpha-amylase assays and cortisol immunoassays, was gained through experts from the Biochemistry Department at Rhodes University, Grahamstown, South Africa.

Weather on the day of the event could not be controlled, and thus the extreme conditions experienced could have affected the performance of the athletes. Extreme weather conditions also resulted in the collapse of the medical tent at the finish. Due to this venue being the post-race saliva collection point, certain athletes were unable to perform either one or both of the post-race samples.

## CHAPTER II

### REVIEW OF RELATED LITERATURE

#### INTRODUCTION

Ultraendurance exercise has been defined as a bout of exercise lasting for longer than four hours (O'Toole and Douglas, 1989). An example is triathlon which is a relatively new sport having originated around 30 years ago (Ackland *et al.*, 1997). It is also one of the fastest growing participation sports in the world, with a growth in the U.S.A. alone of 1500 registered members in 1982 to over 58 000 in 2005. In addition to this, there are currently 120 national federations which are affiliated to the International Triathlon Union (I.T.U.) (Gosling *et al.*, 2007). Triathlons comprise three main disciplines including swimming, cycling and running. In addition, the sport is competed over a number of distances; shown in Table I (McHardy *et al.*, 2006). These vary greatly from the short, explosive sprint distance event to the ultraendurance full Ironman event, which was the focus of this particular study (shaded text in Table I).

**Table I: Triathlon race distances**

<b>Standard Distance Events</b>	<b>Discipline</b>		
	<b>Swim</b>	<b>Bike</b>	<b>Run</b>
Sprint	0.75 km	20 km	5 km
Olympic	1.5 km	40 km	10 km
Long	2.0 km	80 km	20 km
<b>Ultradistance Events</b>	<b>Discipline</b>		
	<b>Swim</b>	<b>Bike</b>	<b>Run</b>
Half Ironman	1.9 km	90.1 km	21.1 km
<b>Full Ironman</b>	<b>3.8 km</b>	<b>180.2 km</b>	<b>42.2 km</b>

The Ironman triathlon was created with the endeavour of crowning the 'fittest' endurance athlete on the planet. In the past, the majority of the contestants involved in ultraendurance triathlons were professionals, however, over the past decade, with its increasing popularity, the participants have been mainly amateurs rather than elite professionals (O'Toole and Douglas, 1989). Elite athletes are defined as those holding a world ITU ranking of less than 125<sup>th</sup> (Bently **et al.**, 2002).

Regardless of performance level the commitment to training for an event such as the Ironman triathlon is a sizable one (Douglas, 1989) and the athletes involved in the race elicit extraordinary athletic capabilities to successfully complete the three consecutive disciplines before crossing the finish line. It is therefore necessary to focus on not only the elite few, but also the amateur athletes making up the majority of the field. The elite professionals take just over eight hours to finish while the amateurs of the field can be competing up until the 17 hour cut-off.

Successful triathletes are those who have the ability to perform each discipline of a triathlon at an optimal intensity, which does not create unnecessary fatigue that could negatively impact performance in the subsequent discipline (O'toole **et al.**, 1989). It has been well established that a correctly formulated training schedule can result in an improvement in an athlete's performance (Smith, 2003). The cardinal rule of basic training adaptation is that performance improvements are achieved by the progressive increase in a sport-specific training stimulus (Rowbottom **et al.**, 1997). The performance of repeated bouts of a specific exercise over a period of time will cause numerous changes to an athlete's physiological ability (Jones and Carter, 2000) as well as alterations to the cellular balance of the body (Birch and George, 1998), resulting in improved performance ability.

Athletes and coaches use this process of ‘overload training’ which involves sport-specific exercise stress/training load, above which the athlete is familiar and comfortable, in order to provide the alterations or disturbances necessary for adaptation (Smith, 2000). The primary concern is that many athletes, recreational or elite are prone to training at excessively high intensities, durations and/or frequencies with insufficient recovery periods incorporated into their training schedules (Lehmann **et al.**, 1997).

## **ADAPTATIONS TO ENDURANCE AND ULTRAENDURANCE TRAINING**

Endurance and ultraendurance exercise training results in marked adaptations to the cardiovascular, respiratory and neuromuscular systems of the body, improving performance ability (Jones and Carter, 2000). There are a number of current physiological models which are available to aid in the understanding of training and enhanced endurance and ultraendurance performance. Noakes (2000) reviews five models of enhanced performance ability (Table II). Although they are separately proposed models, it is unlikely that the factors which are responsible for improvements are restricted to a single model, but are rather a combination of a number of these models (Noakes, 2000). Gaining a greater understanding of these endurance adaptation models will create an awareness of the point at which there are no longer improvements in endurance and ultraendurance performance capacity, aiding in the diagnosis of suboptimal training technique.

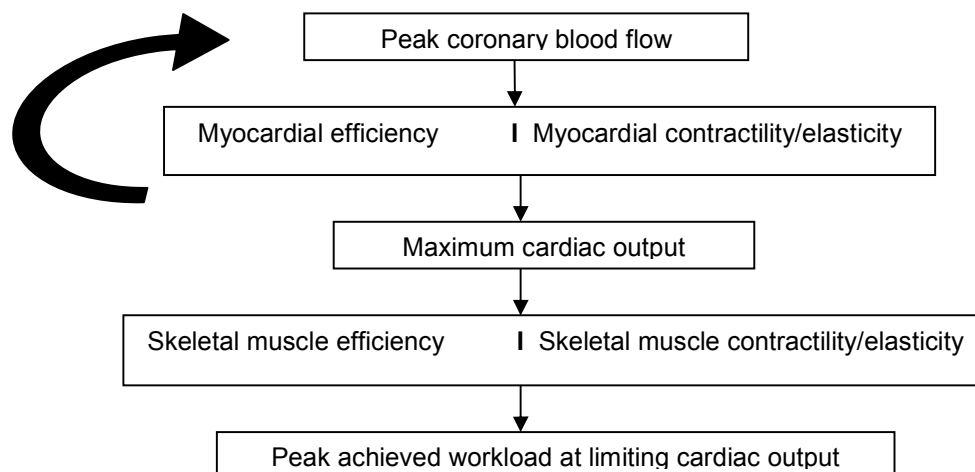
**Table II: Physiological models of enhanced endurance and ultraendurance performance**

<b>Number</b>	<b>Model</b>
<b>I</b>	The cardiovascular/anaerobic model
<b>II</b>	The energy supply/depletion model
<b>III</b>	The muscle recruitment model
<b>IV</b>	The biomechanical model
<b>V</b>	The psychological/motivation model

## CARDIOVASCULAR/ANAEROBIC MODEL

The cardiovascular/anaerobic model states that an individual's endurance performance capacity is defined by the ability to deliver unusually large volumes of oxygenated blood to active muscles (Noakes, 2000). It suggested that exercise training increases the body's maximum capacity to utilise oxygen by increasing the heart's ability to pump blood (increased cardiac output), enhancing the ability of muscles to consume oxygen as well as increasing capillarisation and mitochondrial mass (Noakes, 2000). The greater coronary blood flow results in an increased cardiac output and therefore a greater exercise capacity (Noakes, 2000). These factors all aid in the reduction of exercise-induced anaerobic metabolism, reducing blood lactate concentrations which in turn allows for continued muscle contraction and therefore a slower rate of fatigue (Noakes, 2001). The model holds that there is a level of exercise known as the 'anaerobic threshold' above which the muscles work without adequate blood supply, resulting in fatigue (Noakes **et al.**, 2004).

The major limitation with this model is that if the heart's capacity limits oxygen supply to exercising muscles, then the first organ affected by oxygen deficiency would be the heart itself (Noakes, 2000). As the heart is dependant for its blood supply on its own pumping capacity, any exercise that places demand on the heart without an increase in cardiac output or coronary flow jeopardises the heart's blood supply. Further decreases in coronary blood flow place the heart at risk of developing irreversible myocardial ischaemia (Noakes, 2000). Therefore the first physiological function to undergo a plateau during progressive exercise must be coronary blood flow followed, as a result, by all other physiological functions (Noakes, 1998). While the cardiovascular system can indeed set the limitations for an athlete's maximal exercise performance capabilities, its limit on performance during submaximal exercise, where oxygen supply is adequate, should be further explained (Noakes, 2000). Figure 1 shows the physiological factors which have been described by the cardiovascular model as responsible for the limitation in exercise performance.



**Figure 1: Physiological factors which have a limiting role on maximal exercise capacity**

(Taken from Noakes, 2001)

There are a number of key cardiovascular parameters which have been found to be reflective of endurance training status and include: maximal oxygen uptake ( $VO_{2max}$ ), exercise economy, the fractional utilization of  $VO_{2max}$  ( $\%VO_{2max}$ ) (O'Toole and Douglas, 1995), the lactate/ventilatory threshold and oxygen uptake kinetics (Whipp *et al.*, 1982). The improvement in one or more of these parameters should result in increased endurance performance ability, thus causing a rightward shift in the velocity-time curve, allowing exercise at a given intensity for a longer period of time (Jones and Carter, 2000).

### **Maximal rate of oxygen uptake ( $VO_{2max}$ )**

$VO_{2max}$  reflects the maximal rate of oxygen uptake at which an individual can perform (Jones and Carter, 2000) and has been utilized for many years as a predictor of maximum performance capability (O'Toole and Douglas, 1995; Whipp *et al.*, 1982; Costill *et al.*, 1973b; Saltin and Astrand, 1967). While  $VO_{2max}$  has the ability to represent the maximum performance potential of a triathlete, it does not make up the sole determinant of endurance performance (McArdle *et al.*, 2001). The use of  $VO_{2max}$  has been found to be an indifferent predictor of endurance performance in a group of athletes whose abilities are relatively

homogenous (Noakes, 2000), where athletes with similar  $VO_{2max}$  values can vary greatly in ultraendurance ability (Suriano and Bishop, 2009). This poor prediction ability has stemmed from the inability of the cardiovascular/anaerobic model to predict fatigue resistance of athletes during submaximal exercise (Noakes, 2000). It has been found that athletes that are involved in multisport activities have lower values for maximum oxygen uptake when compared to single sport competitors (Zalcman **et al.**, 2007). This is elaborated by O'Toole **et al.** (1987) who found that there was no significant relationship between  $VO_{2max}$  and Ironman athlete finish times.

Measures of running economy and the fractional utilization of maximal capacity have a greater capacity to represent the ability of how close a triathlete is to reaching their full potential as an athlete (O'Toole and Douglas, 1995). These measures may therefore have a greater ability to analyse changes in performance capacity.

### **Exercise economy**

Exercise economy has been previously defined as the rate of oxygen uptake required by an individual at an absolute exercise intensity (Jones and Carter, 2000). There are however considerable individual differences between athletes with very similar maximal performance capabilities (Morgan and Craib, 1992; Conley and Krahenbuhl, 1980). Athletes with superior economy are at a considerable advantage during endurance and ultraendurance exercise as they require a lower percentage of maximal effort for any given exercise bout. In the past, elite athletes with lower  $VO_{2max}$  values have been found to compensate for this with an excellent exercise economy (Jones and Carter, 2000; Londeree, 1986). Although these parameters are important in the determination of endurance exercise performance it is important to take into consideration that during the training and completion of an Ironman triathlon, psychological factors, training/race tactics and environmental conditions play an integral role in the athlete's endurance exercise performance (Jones and Carter, 2000).

### **Percentage $VO_{2max}$**

While  $VO_{2max}$  can be used for describing the maximal limit for exercise, it has been suggested that parameters measured during submaximal exercise are more reliable predictors of ultraendurance performance (Costill 1973b; Coyle **et al.**, 1988). The fractional utilization of maximal capacity or the percentage of maximal oxygen uptake ( $VO_{2max}$ ) is representative of the effects of both  $VO_{2max}$  and the economy of motion (O'Toole and Douglas, 1995). Due to the fact that the stress induced on the majority of the physiological systems is quantified on a relative, rather than an absolute intensity level, the percentage of  $VO_{2max}$  can also be used as an indicator of how close an athlete can perform to their maximal ability during competition (Costill, 1973b). During endurance training, improvements in both  $VO_{2max}$  as well as exercise economy will aid in reducing the percentage at which athletes complete the same relative exercise bout, thus improving performance (Jones and Carter, 2000). The relationship between these two variables of aerobic training status can be usefully applied in the measurement of athletic capability during the course of an Ironman training regime (Jones, 1998; Morgan **et al.**, 1989). Although these are useful and valid measures of performance, there is, however, little practicality of continual  $\%VO_{2max}$  and  $VO_{2max}$  testing. Not only is it time consuming and logistically improbable, for the purpose of this study, but the tests themselves put the athletes under added, undue stress.

### **Lactate/ventilatory threshold**

The relationship between exercise intensity, blood lactate levels, and the ventilatory threshold of an individual has been found to be a powerful predictor of endurance exercise performance (Jones and Doust, 1998). The lactate/ventilatory threshold (LT/VT) graph shows that endurance exercise training is the source of a rightward shift of these variables towards higher power output ability and is indicative of well constructed endurance performance programmes, thus indicating its importance as a predictor of endurance performance (Wells and Pate, 1988). During exercise of a 'moderate' intensity

(below lactate threshold), and at the intensity where Ironman athletes spend the most of their training and competition, pulmonary oxygen uptake increases exponentially to achieve steady state in approximately 2-3 minutes (Jones and Carter, 2000). Submaximal exercise (below LT) causes a rise in the oxygen deficit increasing blood lactate, however these elevated levels will return to resting levels as exercise continues. Exercise that is above the LT will result in a steady state blood lactate level which is elevated above that of a resting level (Roston **et al.**, 1987). Although the maximum lactate steady state (MLSS) has been referred to as the 'gold standard' as a measure of endurance exercise capability (Hill and Smith, 1999; Poole **et al.**, 1988), steady-state  $\text{VO}_2$  has not been found to change significantly following a period of endurance exercise training (Whipp, 1994), therefore limiting MLSS as a predictor of endurance performance.

## ENERGY SUPPLY/DEPLETION MODEL

### **Energy supply**

The energy supply model proposes that it is the inability of the body to deliver energy substrates to active muscle which explains the limitation of performance. It also states that it is the inability of the body to supply adenosine triphosphate (ATP) to the active musculature, at the required rate, to maintain exercise (Noakes, 2000). Therefore the adaptations to endurance exercise training would include an increase in the body's ability to store energy substrates, as well as the enhancement of the relevant metabolic pathways. Thus, the major purpose of endurance training is the improvement of energy delivery systems which increases exercise economy (Snell, 1997). The argument against this model is that complete depletion of ATP will result in muscular rigor, a phenomenon which does not occur, even during forced contractions in ischaemic conditions (Fitts, 1994).

## Energy depletion

Costill **et al.** (1973a) stated that the depletion of carbohydrate stores is a limiting factor of endurance exercise performance. This statement is supported by a number of findings that suggest that fatigue is associated with a depletion of muscle and liver glycogen stores (Fitts, 1994; Bosch **et al.**, 1993). That hypoglycaemia associated with liver glycogen depletion can be reversed allowing exercise to continue (Noakes, 2000; Cogan and Coyle, 1987) and that carbohydrate loading, prior to, and consumption during training and racing aid in the prevention of fatigue (Hawley **et al.**, 1997; Bosch **et al.**, 1996) shows that depletion of energy stores cannot fully explain fatigue. Further, Noakes (2000), states that there is little evidence that training improves endurance performance ability exclusively by increasing carbohydrate stores. Further, studies which have demonstrated enhanced performance due to high fat feeding suggest that reduced carbohydrate stores are not the limiting factor (Brouns and van der Vusse, 1998).

It is difficult, however, using the energy depletion model, to explain ultraendurance performance during the 42km run leg of an Ironman triathlon event. This is due to the assumption that exercise at a moderate intensity is not at all possible after a marked depletion in muscle glycogen (Noakes, 2001; 2000). Top end Ironman athletes were expected to have near total muscle glycogen depletion on completion of the 180.1km bike leg of the event and yet they were able to run at  $16\text{km}\cdot\text{h}^{-1}$  for an additional 160min after the bike leg (Bosch **et al.**, 1993). If the energy depletion model is correct, then it is physiologically impossible for these Ironman athletes to complete the run leg of the Ironman at such speeds (Noakes, 2001). This is due to the inability of humans to store the adequate carbohydrate to provide energy for such exercise intensities and durations (Noakes, 2001).

## MUSCLE RECRUITMENT MODEL

### **Muscle recruitment and central fatigue model**

While the first two models are based on energy substrate delivery in blood or by the glycolytic and oxidative pathways, there is an alternative model which states that it is not the rate of supply of energy to the muscles, but rather the processes involved in the skeletal muscle (recruitment, contraction and excitation) which limit performance ability (Noakes, 2000). This model claims that the brain affects the stimulation of the skeletal muscles by altering the quantity of neural impulses which reach them, therefore inducing fatigue during exercise (Noakes, 2000).

Although fatigue of skeletal muscle has primarily been associated with the dysfunction of the contraction process itself (peripheral fatigue), the failure of the central nervous system (CNS) recruitment of skeletal muscle constitutes the make up of the central fatigue model (Noakes, 2000; Davis and Bailey, 1997). Peripheral fatigue involves impairments in neuromuscular transmission, dysfunction within the sarcoplasmic reticulum, availability of metabolic substrates as well as actin-myosin cycling (Coggan and Coyle, 1991; Enoka and Stuart, 1992; Balog *et al.*, 1994). Central fatigue has been defined as fatigue associated with alterations in the functioning of the CNS, which cannot be adequately explained by dysfunctions within the muscle itself. The debilitating fatigue that accompanies the overreaching/overtraining syndrome has little to do with the skeletal muscle itself, therefore it is probable that the CNS plays a significant role in fatigue (Davis and Bailey, 1997).

Noakes (2000) argued two interpretations of the central fatigue model: One is that changes in neurotransmitters induce fatigue as a natural progression during endurance exercise such as the Ironman training and racing. Alternatively the role of the central nervous system may be necessary to protect the body under a number of stressful conditions (Noakes, 1998, 1997). These are required to prevent myocardial ischaemia, heart failure (in chronic heart patients) caused by a fall in blood pressure, heatstroke, and hypoglycaemia resulting in glucopaenic

brain damage. The mechanism of control during endurance exercise is through the regulation of recruitment and/or the contraction coupling of skeletal muscle (Noakes, 2000).

The plateau that was in the past said to cause the termination of the process of increased coronary blood flow and thus cardiac output, would result in a continuously progressing myocardial ischaemia, worsening with prolonged exercise duration (Noakes, 2000). The fact that this myocardial ischaemia does not occur suggests that there is another process which controls the limit of coronary blood flow (Raskoff **et al.**, 1976). Therefore there needs to be an alternative hypothesis which states that the limit of enhanced endurance performance, a result of sport specific training, may be the result of a central nervous system fatigue (Davis and Bailey, 1997; Noakes, 2000).

This alternative hypothesis also known as the central governor hypothesis, states that as the heart is at the greatest risk of developing a state of oxygen deficiency, there must be a protective mechanism which exists to restrain the use of skeletal muscles during extremely strenuous (duration and/or intensity) exercise (Noakes, 2000). This 'governor' monitors the state of oxygenation of the heart, brain and diaphragm, in aid of preventing an oxygen deficiency to these vital organs (Noakes, 2001), terminating the point of maximum cardiac output when the development of progressive myocardial ischaemia occurs. When the 'safe' limits of adequate oxygenation are reached, and before the heart becomes ischaemic, the motor cortex in the brain is stimulated. The consequence of this is that skeletal muscle recruitment is prevented from rising any further or it falls, this limits work output of the skeletal muscle, bringing about the onset of fatigue (Noakes, 2000). Therefore the maximum exercise capacity that an individual can perform is a process that is subconsciously coordinated by the brain, and is limited by peak coronary blood flow and therefore the supply of oxygen to the heart (Noakes, 2001).

### **Muscle power model**

According to this model, the ability of muscle to generate force, or muscular contractile capacity, is not equal in all humans. Therefore, athletes with greater contractility will have superior athletic capacity (Noakes, 2000). Although there are few studies which isolate athletes, it is important to remember that strength of muscle does not necessarily result in enhanced performance (Widrick *et al.*, 1996; Fitts *et al.*, 1989). Rather, it has been found that endurance training usually reduces muscle contractility, highlighting the issue of the application of strength specificity in endurance sports (Costill *et al.*, 1992).

### **BIOMECHANICAL MODEL**

Biomechanical factors have been found to contribute to the efficiency of human movement. Included in these biomechanical factors are the ability of an athlete to produce force, differing limb lengths between individuals and body weight distributions. More efficient muscle will aid in the enhancement of endurance exercise performance (Noakes, 2000), while differences in anthropometry and somatotype of athletes may affect exercise economy (Noakes, 2001). This exercise economy is sports-specific therefore the triathlon is unique as it has a three discipline makeup. While an athlete may be best suited to running, it may come at a cost on the swim or bike leg, and vice versa (Noakes, 2001). The more efficient and economical muscles are, the greater their ability to enhance endurance exercise performance by slowing down the accumulation of metabolites which cause fatigue. Further, this will reduce the rate of rise in body temperature which delays the attainment of the body's 'critical' temperature, which halts exercise (Noakes, 2000).

The thermoregulation of the body's core temperature, which is regulated by sensory feedback to the hypothalamus from thermoreceptors in the peripheral and central nervous system (Cheuvront and Haymes, 2001), is influenced by a number of factors during endurance exercise events including environmental conditions and adequate hydration. Due to the prolonged nature of the Ironman

triathlon, athletes are exposed to the environmental elements for up to 17 hours, during which extreme environmental conditions can severely limit an athlete's ability to transfer heat away from the body through the heat gradient, thereby decreasing the time to 'critical' temperature and the onset of fatigue (Cheuvront and Haymes, 2001). The percentage of  $VO_{2max}$  at which the athletes are performing, as well as body mass, are strongly correlated (positive) to body temperature, therefore it is beneficial for 'top end' athletes, performing at higher speeds (especially in the running discipline), to have a reduced body mass, therefore reducing these athletes rate to fatigue (Cheuvront and Haymes, 2001). To aid in the maintenance of the integrity of the thermoregulatory system, adequate hydration is paramount during endurance exercise. The consequence of dehydration is excess strain on the thermoregulatory system and therefore, a decrease in endurance performance capacity. Endurance training has been shown to have an impact on exercise economy, allowing athletes to compete at the same exercise intensities with lower overall heat expenditure and this is even more evident in athletes who are already well trained (Svedenhag and Sjodin, 1985).

#### PSYCHOLOGICAL MODEL

Due to the holistic nature of human performance it is important to take cognisance of the psychological component. The psychological model states that it is a conscious effort which maintains exercise performance ability, and that a dysfunction at any point along the continuum from the brain to the muscle will result in fatigue (Davis and Bailey, 1997). Recent literature has shown that the cognitive orientation plays an important role in the performance of athletes involved in endurance sports (Baker *et al.*, 2005). After decades of research, literature continues to support the fact that there is a relationship between performance-related cognitive awareness and the performance of athletes involved in endurance sports (Masters and Ogles, 1998). It has been found that during an Ironman triathlon, expert/elite ultraendurance triathletes reported thoughts that were more related to performance, while amateur athletes elicited

thoughts that were unrelated to performance (Baker **et al.**, 2005). Elite level athletes can also be characterised by proactive personality traits, predisposing them to higher levels of performance (Seibert **et al.**, 1999). The influence of cognitive function and decision making in ultraendurance sports is not clear-cut, however focusing on performance during, as well as training this thought process, will aid athletes' ability to perceive performance relevant stimuli (Baker **et al.**, 2005). Changes to psyche or mood state of athletes training for an ultraendurance event can result in changes to their performance (Davis and Bailey, 1997). The profile of the mood states (POMS) has been found not only to assist in the diagnosis of the overtraining syndrome but also the identification of athletes who are at risk of overtraining (Hooper and Mackinnon, 1995).

There are countless psychological benefits of endurance training; these include an increase in positive state of mind, reduced tension and anxiety, decreased depression, increased quality of life, positive personality traits, an increased resistance to stress, improved mental functioning and fewer minor medical complaints (Noakes, 2001).

## **ULTRAENDURANCE TRAINING SPECIFIC TO THE IRONMAN**

Although arriving at the finish line on race day is an immensely difficult feat to accomplish, one often overlooks the huge amount of preparation that the athletes involved have pursued for the many months up until the event. The majority of athletes begin a formal training schedule approximately eight months prior to competition (O'Toole, 1989). It is therefore important to study this training/preparation period in an attempt to optimise training techniques for competitors (Cheuvront and Haymes, 2001). A large number of studies have focused on either the event itself and/or the period post-competition (Neubauer **et al.**, 2008; Bentley **et al.**, 2007; Bentley **et al.**, 2002; Ginsburg **et al.**, 2001; O'Toole and Douglas, 1995; O'Toole, 1989; O'Toole and Douglas, 1989; O'Toole **et al.**, 1989; O'Toole **et al.**, 1987), while few have studied the progress of

athletes during training or preparation. Although Coutts and co-workers (2007) studied the practical tests for monitoring performance and recovery in triathletes, their study focused on Olympic distance events rather than the Ironman event illustrating the need for a longitudinal analysis of Ironman athletes.

Athletes have been shown to average a weekly training schedule of 21 hours, with average weekly training distances in the disciplines of 8km swimming, 330km cycling and 75km running (Ginsburg *et al.*, 2001). There is however wide variation in the training distances and paces. This is to be expected due to the large variation in the performance capacities of the athletes. Gublin and Gaffney (1999) compared the training regimes of athletes training for the Ironman World Championships in Kona, Hawaii and those of athletes training for other Ironman events. The findings show that many of the athletes are concerned with an approximation of the minimum amount of training necessary to complete the event successfully.

### **World Championships: Kona, Hawaii**

In 1978 the first Ironman event took place in Honolulu, Hawaii. Only 12 males, and no females, finished the race in a winning time of 11 hours and 46 minutes. The Ironman in Hawaii moved to Kona in 1981 and remains there as the World Championships course to this day. Between the years 1985 and 1988 a group of research scientists, collectively known as LABMAN (O'Toole, 1989), gathered training information and finish line information from 323 volunteering Ironman athletes (235 males and 88 females). The LABMAN research focused part of its attention on the athletes training for the Ironman in Kona Hawaii. The extreme training load of the triathletes taking part in this is illustrated by the immense distances and intensities trained in each discipline. These triathletes swam an average of  $12\text{km}\cdot\text{wk}^{-1}$  at a mean pace of  $20\text{min}\cdot\text{km}^{-1}$  and cycled an average of  $365\text{km}\cdot\text{wk}^{-1}$  with a mean pace of  $29\text{km}\cdot\text{h}^{-1}$ . The minimum weekly cycling training distance covered by each athlete was 24km while the maximum training distance recorded was 700km. These athletes ran an average of  $72\text{km}\cdot\text{wk}^{-1}$ , with the

lowest weekly mileage of 7km and the highest weekly mileage of 160km (O'Toole, 1989). Over the period between 1981 and 2007 the performances of triathletes, at Kona, was combined by Lepers in (2008). The results of this study showed that since 1981 performances amongst elite, male and female, triathletes have improved. While these initial decreases in race time were most likely due to increased participation, training, racing strategy and nutrition habits, there has been a plateau in performance since the late 1980's.

### **Non-World Championship events**

The typical training performed by elite athletes may not be representative for the athletes training for non-World Championship events (Gublin and Gaffney, 1999). Findings taken from athletes training for the Austrian Ironman event, show that athletes did not complete such high training loads as those competing in the Ironman in Kona. The results of this research also indicate that, discipline specific race distance training was not necessary prior to race day. Non-World Championship athletes' mean weekly training distances were recorded for swimming, cycling and running as, 8.8km (18.1min.km<sup>-1</sup>), 270km (31.8km.h<sup>-1</sup>) and 58.2km (4.55min.km<sup>-1</sup>) respectively (Gublin and Gaffney, 1999).

The search for 'optimal' training techniques tends to become problematic due to the large inter-individual differences found between athletes. This difference is accentuated by the differences in strengths of athletes throughout the three disciplines, often leading to aspiring athletes following training programs set out for elite athletes at the top of the sport (O'Toole, 1989). In addition, elite athletes often try to 'out do' their competition with new and improved training techniques. Due to advances in technology these training practices can be made individually relative although the training variables that O'Toole (1989) found to have the most influence on the finishing times of the Ironman were: experience in triathlons, number of months of training as well as the number of triathlons completed prior to the race. Athletes that trained for a longer period of time, had longer single training sessions and averaged greater training mileage and

number of sessions per week generally were the best performers in Ironman triathlons (O'Toole, 1989). Although O'Toole's (1989) research shows that there is a correlation between amount of training and ultraendurance triathlon performance, there is no mention of over-reaching or the overtraining syndrome, two feared aspects of training especially due to the long training duration for an Ironman.

## **QUANTIFICATION OF TRAINING**

Recording the daily changes in a number of performance and fatigue based markers has been proposed as an effective method of assessing training load and therefore aiding in the diagnosis of the OTS (Brown **et al.**, 1983; Rushall, 1990; Hooper **et al.**, 1995; Hug **et al.**, 2003). While distance, duration and speed of training performed gives absolute values which can be used in the comparison of training performed, each individual athlete will respond in a different manner to the same load of training (Borresen and Lambert, 2008). It is therefore imperative to determine the relative load of training being performed by each athlete. While there are a number of different methods of quantifying training load, the two selected which were most suited to this research were the objective Training Impulse (TRIMP) method, and the more subjective Session-RPE method. The ability to quantify a complex interaction such as training load into a simple single figure has become appealing due to its practical application (Borresen and Lambert, 2008).

### **TRAINING IMPULSE (TRIMP) METHOD**

The majority of the training completed by Ironman athletes is performed in a steady state nature. Therefore it is necessary to identify a method of training load quantification that is best suited to this type of training. The heart rate response of an athlete to a bout of exercise, coupled with the duration of such exercise, was suggested to be a possible method for objectively quantifying the load of

training performed during an exercise session. The TRIMP method for calculation of training load was therefore developed by Banister **et al.** (1991) taking into account these two variables, thereby providing one unit for the load of training performed by an individual athlete during a specific session (Borresen and Lambert, 2008).

#### SESSION-RATING OF PERCEIVED EXERTION (RPE) METHOD

As a large number of athletes are unable to, or choose not to, perform their training with the aid of a heart rate monitor, it is important to ensure that training intensity and therefore it is necessary to be able to quantify training load during these sessions without a heart rate value. The Session-RPE method developed by Foster **et al.** (1996) enables this quantification by simply multiplying the duration of the session with the athlete's global intensity, in RPE, of the session. This uncomplicated method was designed to encourage athletes to take into consideration 'how they perceived the work out' to aid in to quantification of training load, a method which has been considered a valid and reliable measure of training load during aerobic exercise sessions (Borresen and Lambert, 2008).

#### COMPARISON OF METHODS

A comparison between these two training methods showed that the methods had a correlation of  $r=0.76$  (95% confidence interval: 0.56 to 0.88) (Borresen and Lambert, 2008). While the relationship between these two methods only accounted for 58% of the variance (Borresen and Lambert, 2008), it is important to take into account the complex interactions affecting perceptual responses to physical effort when analysing such RPE-based methods of quantification. These interactions include hormone concentrations, personality traits, environmental conditions and psychological state of the athlete involved (Williams and Eston, 1989). Research by Borresen and Lambert (2008) has showed that the TRIMP method overestimates training load in comparison to the Session-RPE method in athletes who spend more time training at higher intensities. Athletes who train at

predominantly lower intensities have shown underestimated training loads compared to the Session-RPE method.

## **SUBOPTIMAL TRAINING**

Enhanced endurance capacity is achieved by the improvement of the body's ability to tolerate the stressful demands of training as well as competition (Fry **et al.**, 1991). This increased ability to tolerate stress comes from the application of several training stimuli, creating the overload necessary for adaptation to occur (Matveyev, 1981). Following this period of overload, recovery is essential as it is during this time where the disturbances caused by exercise are corrected, resulting in overcompensation and enhanced athletic ability (Virus, 1994). However an insufficient application of the training stimulus, or too much recovery time, can lead to a lack of performance progress or detraining (Neufer, 1989). Inter-individual differences between athletes suggests that they require different recovery and regeneration periods. Non-training specific stress factors and exercise tolerance help to identify the fact that athletes have different vulnerabilities when exposed to the same training-related stress factors (Lehmann **et al.**, 1993). These issues are further complicated by the fact that where, previously, it was seen as adequate to train once a day, athletes now train as regularly as twice, or more, times per day (Hug **et al.**, 2003).

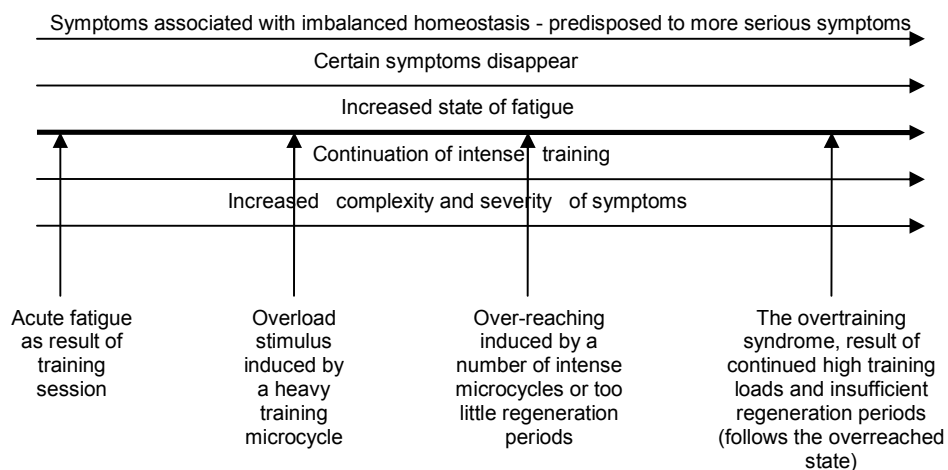
Due to the immense amount of training being performed by Ironman athletes there is a great deal of concern surrounding the issue of balance between training/competition and recovery. An imbalance between these two components will result in performance decrements caused by either insufficient training or overtraining. Too little training and competition experience will result in athletes who will be unable to perform at their optimal efficiency. Insufficient recovery will lead to neuromuscular, sympathetic, metabolic, and psychological and adrenal overload, resulting in an accumulation of fatigue and the onset of overtraining (Fry **et al.**, 1991).

In an attempt to enhance the understanding of the correlation between training inputs and performance outcomes, Morton (1997) studied the effects of modelling training using a dose-response tool similar to that used in pharmacological studies. The primary interest with this dose response was to design a training program which achieves the desired goal of maximizing the performance potential of athletes at a known future date (competition date), while at the same time minimizing the risk of overtraining. The tool takes into account three factors: fitness, fatigue and performance, with a dose of exercise resulting in an increase in fitness and fatigue. Fatigue is noticeable immediately after the completion of a bout of exercise, while fitness is not immediately perceived (Morton, 1997). The positive balance between these two factors over a period of time will result in an increase in performance ability.

The broad term, overtraining, which has many other terms including overwork, failing adaptation, overstress, staleness, stagnation, chronic fatigue and burnout (Eichner, 1995), can be divided into different levels of severity which include: functional/short term over-reaching, non-functional/extreme over-reaching and in the most severe cases, the overtraining syndrome (Coutts **et al.**, 2007). It is a commonly occurring problem due to a number of reasons. Two of these reasons are that the relationship between training load and that of performance encourages athletes to excessively increase their training loads in attempt to gain minimal improvements in performance (Foster, 1998). These small increases in performance are of even greater concern to elite athletes as it is these increases that define top class competitive results (Foster **et al.**, 1996).

The second reason put forward is that of the instinctive response of both athlete and coach to increase training load following poor competition results (Foster, 1998). On numerous occasions poor performance is often the result of fatigue caused by overreaching and a further increase in training is the most inappropriate response, resulting in a further development of the overtrained state (Foster, 1998).

There is no indication that overtraining can result in irreversible damage to the athletes involved, however, it does suggest a greater prevalence of injury, a weakened immune function and a premature end to the athlete's career, with a recovery period of weeks up to months required for complete recovery (Hooper and Mackinnon, 1995; Mackinnon and Hooper, 1994, Morgan **et al.**, 1987). There is however no evidence for a guideline to the difference between overreaching and overtraining. Rather, there is a continuum, portrayed in Figure 2, from a training state where the symptoms of overtraining do occur, to where they do not occur (Fry **et al.**, 1991).



**Figure 2: The continuum of the signs and symptoms of overtraining**  
(Adapted from Fry **et al.**, 1991)

### OVER-REACHING

Training programs commonly involve intentional or, in some cases, an unintentional induction of short term overtraining or functional over-reaching (Fry **et al.**, 1991). This training overload is considered by many coaches as a part of the normal training process (Meeusen **et al.**, 2006). Although many coaches insist that it is necessary to induce a state of over-reaching for performance

enhancements during training, the general consensus has been that over-reaching should be avoided due to the unpredictable outcome (Urhausen and Kindermann, 2002).

### **Functional over-reaching**

The application of functional over-reaching can be achieved by manipulating the athletes' training intensity, frequency and/or duration (Derman **et al.**, 1996). These altered training patterns combined with appropriate periods of recovery have been found to result in an increase in performance and a heightened work capacity due to supercompensation (Fry **et al.**, 1991).

### **Non-functional over-reaching**

If adequate recovery and taper periods are not permitted by a training schedule, functional over-reaching becomes non-functional causing a wide range of performance reducing symptoms as well as abnormal hormonal regulation, psychological disturbances, reduced immune system functions and sleep disorders (Coutts **et al.**, 2007). This fatigue caused by over-reaching can be reversed by a reduction in training or a few days of full rest (Snyder **et al.**, 1993; Jeukendrup **et al.**, 1992). However, if further exposure to the training stress occurs, without a manipulation of the training balance, it can induce staleness, burnout or the overtraining syndrome (Lehmann **et al.**, 1993).

### **THE OVERTRAINING SYNDROME**

The overtraining syndrome regarded by Urhausen and Kindermann (2002) as one of the most feared complications faced by athletes and coaches, has been defined as a state of chronically depressed performance which is accompanied by one, or more, of the more serious symptoms (Frey **et al.**, 1991). However due to the wide variation caused by inter-individual and intra-individual differences of athletes (Urhausen and Kindermann, 2002), the onset of the overtraining syndrome has a large number of diagnostic symptoms. These symptoms include

increased waking pulse rate, 'heavy legs', decreased motivation due to a decreased enjoyment of exercise, musculoskeletal pain, sleep related disorders, depression, a feeling of dizziness upon standing, dramatic weight loss, increased prevalence to infections, decreased sexual drive, an increase in the level of effort required to perform exercise training as well as a decrease in exercise performance (Eichner, 1995; Hooper **et al.**, 1993). Most researchers use the 'German approach' (Israel, 1976), and further divide the overtraining syndrome into two defined types, relating them to the sympathetic nervous system (basedowoid) and parasympathetic nervous system (addisonoid) (Urhausen **et al.**, 1995). Although these types of overtraining have been divided, a combination of the two often exists, the sympathetic type prevalent during the early stages of overtraining, with a shift to the parasympathetic form after a period of time (Urhausen **et al.**, 1995).

The sympathetic type of overtraining, related to anaerobic sports, has been determined to be the likely consequence of an imbalance between training/competition and recovery but is related to an exposure to an excessively high intensity training schedule along with a number of accompanying psycho-emotional, non-training/competition related, stress factors (Lehmann **et al.** 1998). Symptoms of sympathetic overtraining include an increased pulse rate and blood pressure at rest, dramatic reduction in body mass, decreased rate of recovery of heart rate after a given load, loss of appetite and altered mood states (Kindermann, 1986).

The parasympathetic type of overtraining, observed in endurance 'aerobic' sports (Lehmann **et al.**, 1997), referred to as the 'modern type' of overtraining, is much more frequently observed than the sympathetic form, and is related to the long-term exposure to excessively high training volumes with inadequate recovery periods (Lehmann **et al.**, 1998). The symptoms of parasympathetic overtraining have been reported as a decreased sport-specific performance capacity, a reduced resting blood pressure, progressive anaemia, unaccustomed appetite, hypoglycaemic symptoms during exercise, altered mood state, sleep disorders,

persistence high fatigue ratings as well as disturbed immune and reproductive function (Foster and Lehmann, 1997; Urhausen **et al.**, 1995; Lehmann **et al.**, 1993; Kuipers and Keizer, 1988; Bompa, 1983; Kerezty, 1971). It is however important not to discount the effects of everyday life stress as these will play an integral role in the development of the OTS (Lehmann **et al.**, 1993).

Once the overtraining syndrome has been 'diagnosed' or developed, it is essential that athletes take care in aiding their recovery to a non-overtrained state. Some practical suggestions to aid recovery, are to rest for 6-12 weeks including very light training and cross training during this rest period, consult a therapist who specializes in Rational Emotive Behaviour Therapy as well as speaking to athletes who did continue to overtrain once diagnosed with the overtraining syndrome (Birch and George, 1998). Further training in this overtrained state is counterproductive and has the ability to induce chronic fatigue and Fatigued Athletes Myopathic Syndrome (FAMS) (Noakes, 2001; Derman **et al.**, 1996). The major diagnostic symptoms of chronic fatigue include an onset of persistent or reoccurring fatigue that has been present for a minimum of six months. The individual's state of fatigue does not improve with bed rest and has the ability to reduce daily physical activity to 50% of a relative 'normal' (Derman **et al.**, 1996).

Recently, a trend has been recognized amongst those athletes suffering from FAMS. These athletes have a history of high volume exercise training, a wide range of skeletal muscle related symptoms (delayed onset muscle soreness (DOMS), stiffness, tenderness, and cramps). They do not fulfil the symptoms of chronic fatigue and have not been able to identify a diagnosis (Derman **et al.**, 1996).

## RECOVERY

To avoid the symptoms of overtraining, recovery should be administered systematically (Lehmann **et al.**, 1993). Recovery encompasses both acute (several hours) and chronic recovery (in excess of two weeks) in response to

training (Lehmann **et al.**, 1993; Halson **et al.**, 2002). It has been put forward by Kellmann and Gunther (2000) that recovery should be more than a passive resting period after a systematic increase in training load, and that this process of restoring homeostasis is often overlooked.

The requirement of both of these types of recovery is necessary to avoid the development of the OTS. Acute recovery assists in the prevention of non-functional overreaching, while chronic recovery is required to allow for adequate regeneration to avoid the onset of the OTS (Lehmann **et al.**, 1993). While it is important that a training regime provides an athlete with the adequate training necessary to complete an event, the manipulation of the intensity and volume of a training regime is often required to optimise performance outcomes (Kellmann and Gunther, 2000). Training should be monitored continuously so that it can be manipulated as soon as negative symptoms are evident, this allows athletes to obtain a balance between training and recovery (Morgan **et al.**, 1987).

## **DIAGNOSIS OF SUBOPTIMAL TRAINING**

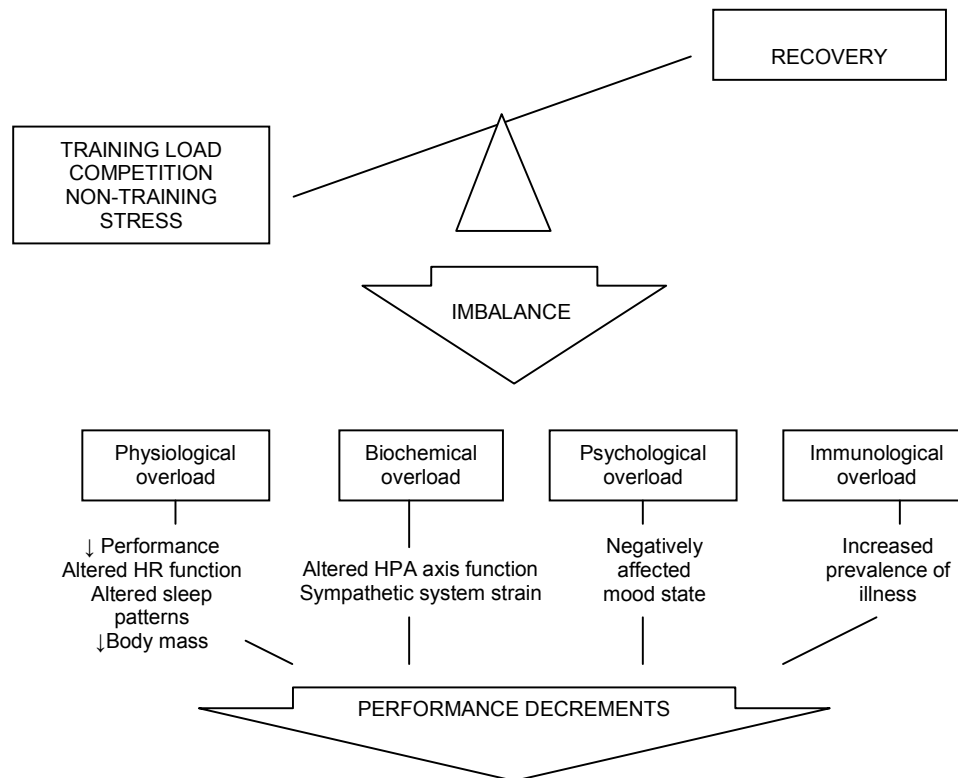
For many years it has been, and still is, a challenge for sports scientists to propose accurate and reliable markers of suboptimal training resulting in fatigue and overtraining (Lac and Maso, 2004). It is impossible for coaches to develop a single 'optimal' training program based on a subjective perception of the athlete's ability. This is due to individual differences in load tolerance and recovery capacity found between athletes. This is further complicated by highly motivated athletes intentionally covering any of the signs and symptoms of overtraining (Hooper and Mackinnon, 1995). Due to the serious nature of the symptoms of overtraining, with a required recovery period of 3-6 months up to approximately a year (Lehmann **et al.**, 1993; Kuipers and Keizer, 1998), the prevention and diagnosis of fatigue of overtraining is therefore extremely important (Maglisho, 1982; Javorek, 1987). The major concern with the parameters which have been propounded for the diagnosis of the OTS is that the results of studies performed

have either been inconsistent or there have been too few studies to propose definite conclusions (Hooper **et al.**, 1995).

Over recent years, researchers have made a concerted effort to enhance their predictive capabilities of exercise performance during ultraendurance triathlons (Laursen and Rhodes, 2001). Many authors have attempted to enhance this ability by the use of a number of key physiological variables which promote performance during endurance exercise (Langill and Rhodes, 1993; Butts, **et al.**, 1991; Dengel **et al.**, 1989; O'Toole, 1989). Laboratory studies have found that the factors affecting endurance performance include maximal oxygen uptake ( $VO_{2\text{ max}}$ ), economy of movement as well as the rate of oxygen uptake ( $VO_2$ ) and anaerobic threshold (AT) (Sleivert and Rowlands, 1996; Dengel **et al.**, 1989). These physiological variables have been identified as predictors of performance ability during endurance exercise. However, they have also been reported to differ from the actual performance intensity of athletes during competition (Hoogeveen and Schep, 1997; Nichols **et al.**, 1997). Therefore the results of laboratory based physiological measurements are not highly correlated to performance during ultraendurance triathlon events due to variability in athlete training status, athlete experience and extraneous factors such as thermal regulation, hydration and energy homeostasis (Laursen and Rhodes, 2001).

There are a large number of proposed diagnostic markers and theories which have been hypothesized to account for the symptoms of overtraining (Smith, 2000). It is therefore more effective to take a holistic approach in the identification of the signs and symptoms of overtraining. Tests for these symptoms should be performed taking into consideration physiological, perceptual, biochemical and immunological domains (Figure 3). It must also be acknowledged that psychological stress may have a significant impact on an individual's risk of overtraining. The large numbers of symptoms which have been identified in each of these domains have been described in previous literature and illustrate the complexity of the symptoms (Fry **et al.**, 1991). Therefore it is important to identify

a number of selected parameters, within these domains, that have the ability to distinguish between the changes associated between optimal training and those associated with the overtraining syndrome (Kreider **et al.**, 1998).



**Figure 3: The mechanisms underlying the overtraining syndrome**  
(Adapted from Hug **et al.**, 2003)

Although the knowledge of central pathomechanisms of the overtraining syndrome has significantly increased over the past decades, there is still a large demand for accurate tools for the diagnosis of the OTS (Urhausen and Kindermann, 2002). The diagnostic markers which have been propounded have been less frequently based on well founded experimental observations but rather on personal observations (Urhausen **et al.**, 1995). The use of regular testing or longitudinal follow-up of athletes has been put forward as the best, if not the only, method of recording progress in performance (Lac and Maso, 2004). Therefore, performing a prospective analysis of athletes during training for an Ironman

event, rather than taking a retrospective approach, will provide the necessary basis for the early identification of the signs and symptoms of overtraining thereby providing the necessary basis for the prospective nature of this study.

However due to the lack of objective parameters available for the diagnosis of overtraining there are complications regarding which parameters to use during the longitudinal follow up of athletes (Urhausen **et al.**, 1995). The tools for the diagnosis of overtraining can be divided into two groups; they include parameters which can be tested at rest and those which need to be performed during exercise (Urhausen and Kindermann, 2002). Regardless of the problems involved and the lack of diagnostic markers of the OTS, it is important that the parameters available are the most accurate, reliable, and cost efficient methods for diagnosing suboptimal training. This particular study included a number of markers to aid in the identification of suboptimal training. Markers should include documentation of performance and training changes, as athletes should be monitoring training on a daily basis (Hug **et al.**, 2003).

It has been proposed by a number of researchers that training and overtraining is most effectively monitored by the athletes themselves (Brown **et al.**, 1983; Rushall, 1990; Hooper **et al.**, 1995). While the results gained from self analysis studies has not yet been determined as an accurate and reliable preventative tool for the OTS, the information gained is extremely valuable to determine the response of athletes to exercise loads (Hooper and Mackinnon, 1995).

## PHYSIOLOGICAL / PERFORMANCE RELATED DIAGNOSTIC MARKERS OF OVERTRAINING

### **Performance**

A decrease in performance has become the universal measure of the diagnosis of suboptimal training (Smith, 2000). Therefore it can be used as a simplistic method of identifying the OTS. The issue, however, with using decreased performance outputs as a diagnostic tool is that once performance has begun to

decline the athlete has already progressed into a state of overtraining. It is therefore important to find a suitable variable that can be used to diagnose suboptimal training before performance is negatively affected. A proposed method for the early diagnosis of the OTS is by the completion of a log book, recording all relevant information pertaining to the athletes training status (Hooper **et al.**, 1995).

The focus of this study was to measure the reliability of a number of tools used for the diagnosis of OTS. These tools were assessed from the early phases of the training period through to finishline of the event. These measures were collected to determine the most suitable early indicator of the signs and symptoms of suboptimal training. Race day samples were collected to assess the effects training and race pacing on selected stress hormone levels. Hooper and Mackinnon (1995) stated that it is important that there should be criteria developed to aid in the diagnosis of the OTS. This is due to the broad range available where performance decrements, which are a result of overtraining, have been found to range from 0.7 – 15% (Hooper **et al.**, 1993; Costill, 1986; Barron **et al.**, 1985). Certain researchers have even found that a plateau in performance, when considered with a number of other symptoms, is adequate to diagnose overtraining (Hooper **et al.**, 1995; Kuipers and Keizer, 1988). The problem with this is that all of the domains which affect human performance are influenced at differing rates and to different degrees, thus further complicating the diagnosis of overreaching and overtraining (Kreider **et al.**, 1998).

### **Time Trial**

Coutts **et al.** (2007) stated that progressively monitoring acute changes in a triathlete's performance, by the use of a running time trial, may aid in the prevention of obtaining an overtrained state. They also stated that future studies should examine the relationship between the practical tests of performance and the athlete's exercise capacity over prolonged period to justify their sensitivity. This performance should be recorded using a standard measure. The 3km

maximal pace running time trial has been found to be an accurate measure of performance during training for Olympic distance triathlons (Coutts **et al.**, 2007). Due to the Olympic distance focus of the Coutts **et al.** (2007) study, the use of the 3km time-trial may be justified. However maximal tests may place ultraendurance athletes in an unaccustomed training intensity, which may put them at risk of overtraining. The greatest concern with the continual assessment of performance by the use of physical tests, is that they can be overly stressful and therefore, in turn, promote the onset of the overtraining themselves (Coutts **et al.**, 2007). Therefore, the performance of tests which are issued to the athletes for the diagnosis of overtraining should be performed at a greater distance and at a submaximal pace. Mark Allen under the guidance of Philip Maffetone, used an 8km submaximal time trial to assess performance levels during the training period. The use of a submaximal time trial placed Mark to less stress than a time trial of maximal nature (Noakes, 2001).

Along with decreased performance there are a number of other physiological performance markers which can be used to aid in the diagnosis of suboptimal training. Among many others these include: need for prolonged recovery, changes in heart rate at rest, exercise and recovery, decreased body mass, sleep pattern disturbances and a generally increased prevalence of aches and pains (Fry **et al.**, 1991).

### **Heart rate changes**

Heart rate measures are a simple and effective method of monitoring training and competition intensity as well as changes in average values at rest or exercise, and can therefore be useful in determining performance adaptations during training (Roalstad, 1989). Athletes progressing into a state of overtraining have been found to show an increase in their wakening heart rate with an elevation of 5-10bt.min<sup>-1</sup>. Noakes (2001) suggested that this increase is likely due to a heightened activity of the sympathetic nervous system which reflects the increased stress on the body, as well as an insufficient recovery period between

training sessions. The measurement of heart rate after standardized submaximal exercise has been found to be a viable method for the continual assessment of athletic performance and overtraining, however there is little literature to support this (Costill, 1986). An increased heart rate during submaximal exercise, therefore decreasing performance (time-trial), has the ability of indicating infectious disease and reduced exercise tolerance (Urhausen and Kindermann, 2002)

### **Sleep patterns**

Sleep is an important facet of any training program and is necessary for athletes to fully recover and regenerate. Altered sleep patterns during training have been described by Urhausen **et al.** (1998) as a possible early indicator of overtraining, with disturbances in these self-analysis patterns being present well before performance decrements and other signs and symptoms of overtraining become apparent (Hooper **et al.**, 1995). Moderate training has been found by Santos **et al.** (2007) to improve the quality of sleep. However, exercise at higher intensities and durations which do not entail adequate rest, may have an adverse effect on these sleep patterns. Sleep disorders including overall short sleep time as well as poor sleep quality, have been found to be common sleep related disorders associated with overtraining (Smith, 2000). Sleep deprivation has a number of effects on athletic performance with both aerobic and anaerobic pathways affected, as are fatigue and recovery processes. All these effects are dependent on the timing and the length of the waking state, but many of them are seen after only few hours of sleep deprivation (Davenne 2009).

### **Body mass variations**

Inadequate caloric intake during endurance training can lead to chronic weight loss as well as decrements in performance ability (Applegate, 1989). This is largely due to the difficulty of matching energy intake with the high energy expenditure associated with ultraendurance training. Therefore athletes are advised to record body mass on a daily basis to aid in assessing caloric intake

adequacy (Applegate, 1989). Intense exercise, which reflects an anabolic process, is associated with elevated cortisol levels and decreased free testosterone concentrations (Aldercreutz **et al.**, 1986). The ratio between these two hormones (cortisol:testosterone) has been found to be entirely opposed to the requirements of protein anabolism, thus aiding in the understanding of weight loss in overtrained athletes (Kindermann, 1986).

#### BIOCHEMICAL RELATED DIAGNOSTIC MARKERS OF OVERTRAINING

Historically the use of salivary hormone analysis has been problematic. However, advancements in technology have made it possible to accurately assess the biomarkers in saliva, in a non-invasive manner (Levine **et al.**, 2007).

#### **Cortisol and the Hypothalamic-Pituitary-Adrenal (HPA) axis**

Over the past decade there has been a growing interest in the role of the hypothalamic-pituitary-adrenal (HPA) axis with the signs and symptoms of suboptimal training and the overtraining syndrome (Lac **et al.**, 1997; Urhausen **et al** 1998; Lac and Maso, 2004; Grossi **et al.**, 2005). The HPA axis is a three gland component of the endocrine system and is stimulated by acute and/or chronic stress (Bugajski **et al.**, 1999). When the body is exposed to physiological and/or psychological stress the hypothalamus is stimulated by the central nervous system (McArdle **et al.**, 2001). This causes a secretion of corticotrophin-releasing hormone (CRH) from neurons of the paraventricular nuclei of the hypothalamus (Moidel **et al.**, 2006). In response, corticotrophs of the anterior pituitary gland secrete adrenocorticotrophic hormone (ACTH) which in turn causes the secretion of cortisol from the zona fasciculata and zona reticularis of the adrenal cortex (Levine **et al.**, 2007; Moidel **et al** 2005).

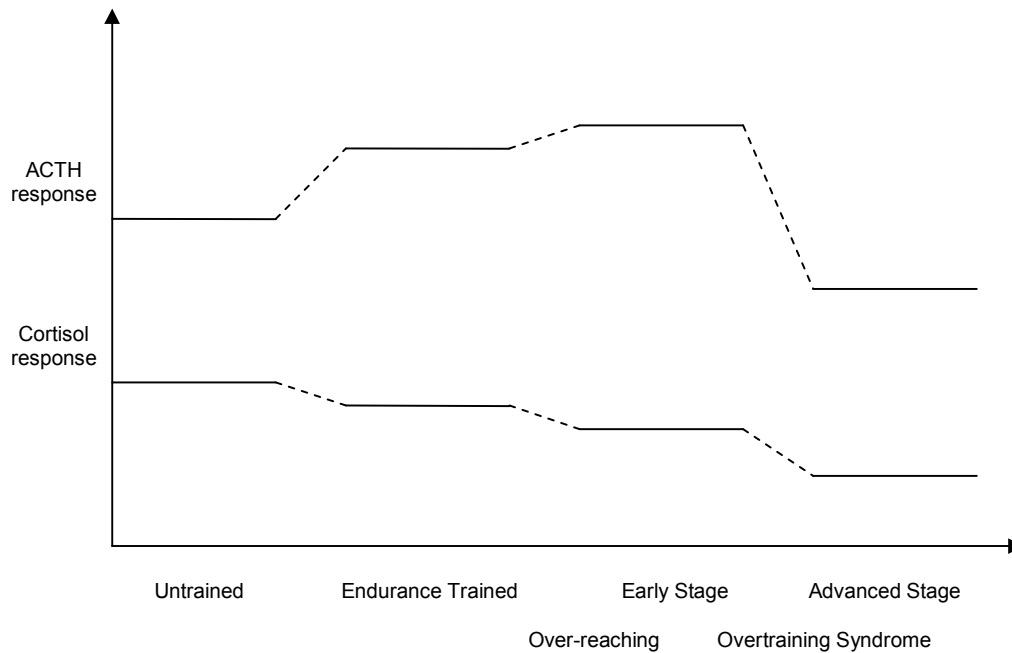
Research by Wittert **et al.** (1996) and Persson **et al.** (1980) shows that ultraendurance exercise may have an effect on the functioning of the hypothalamus and the autonomic nervous system, therefore affecting the HPA axis and causing decreased adrenal responsiveness (Wittert **et al.**, 1996). Since the overtraining syndrome has been considered a stress response to exercise

Hooper **et al.** (1995) found that it was not surprising that changes in adrenal hormones accompanied the symptoms of overtraining. Due to the negative feedback nature of the HPA axis, elevated circulating cortisol levels lead to the suppression of CRH and ACTH and the reduction of cortisol production (Levine **et al.**, 2007). However a number of findings in the past have found evidence of reduced adrenal responsiveness and hypothalamic-pituitary-adrenal dysfunction amongst athletes during heavy training, over-reaching and overtraining (Noakes, 2001; Urhausen, 1998; Lehmann **et al.**, 1992).

This dysfunction and reduced adrenal responsiveness, which can be seen in Figure 4, has been found to be due to an incomplete regeneration of the hormones and can be determined by an increased compensatory ACTH response (Lehmann **et al.**, 1998). This compensation is no longer evident in the early stage of the parasympathetic overtraining syndrome, therefore causing cortisol responsiveness to decrease (Lehmann **et al.**, 1992). In the advanced stage of overtraining, both the ACTH and cortisol responses are significantly reduced (Barron **et al.**, 1985). This depression of adrenal response has been interpreted as a protective mechanism by the body, either due to the negative feedback nature of the HPA axis induced by cortisol or by the depletion of the ACTH pool (Urhausen **et al.**, 1998; Viru, 1985).

Cortisol is the end product of the HPA axis and the major glucocorticoid of the adrenal cortex and it promotes the breakdown of amino acids (in all cells except the liver), acts as an insulin antagonist, promotes triglyceride breakdown and supports hormones such as glucagon and growth hormone in the process of gluconeogenesis (McArdle **et al.**, 2001). It therefore plays a pivotal role in providing the required bodily response to physiological and psychological stress (Levine **et al.**, 2007). Cortisol turnover, or the difference between its production and removal from the body, provides sports scientists with a convenient means by which to study the response to exercise (McArdle **et al.**, 2001). Factors such as individual difference, exercise duration and intensity, level of training status,

nutritional intake, and the circadian rhythm cause a substantial amount of variability in cortisol turnover, causing the analysis of collected cortisol to be a challenging process (Davis *et al.*, 2000; Suay F *et al.*, 1999).



**Figure 4: Schematic presentation of the alterations in adrenal responsiveness through the continuum from untrained to the overtraining syndrome**

(Adapted from, Lehmann *et al.*, 1998)

Although cortisol has been used extensively in human studies there is still debate on which method of measurement is most accurate, free or total cortisol (Levine *et al.*, 2007). A commonly used method of free cortisol measurement is that of salivary cortisol (Kammerer *et al.*, 2007). The validity of the salivary samples for measurement of free plasma cortisol has been extensively researched (Kirschbaum and Hellhammer 1994, 1989) and has been found to be an accurate and reliable indicator of HPA activity (Grossi *et al.*, 2005). Salivary cortisol has also been suggested as the preferred method of cortisol sampling during rest, recovery and exercise due to its non-invasive nature and its ease of repeated sampling on numerous occasions (Neary *et al.*, 2002). Gender differences

associated with corticosteroid responses have been found, these differences have been found to be significant during times of competition (Kivlighan **et al.**, 2005). Cortisol levels for males have been found to decrease significantly from 20 - 40 minutes after competition, while female's show no significant changes.

However, 'at home' testing comes with a number of issues which could act as drawbacks to the sampling and include: compliance and sample variability; subjects providing an insufficient quantity of saliva; samples being provided after the consumption of any liquids and/or solids which may affect reliability results and the contamination the saliva by blood due lesions in the mouth (Grossi **et al.**, 2005; Guttling **et al.**, 2005; Ashman **et al.**, 2002). However, even with these drawbacks, salivary cortisol is still a practical and useful measurement technique (Levine **et al.**, 2007). Single cortisol samples have failed to illustrate the correlation between overtraining and hormonal levels, therefore a longitudinal approach, with several samples, is more appropriate for an accurate analysis (Grossi **et al.**, 2005). Awakening cortisol levels have been found to be suited to determining subtle dysregulation in the activity of the HPA axis without being influenced greatly by a number of factors such as age, sleep duration and using an alarm clock (Pruessner **et al.**, 1997).

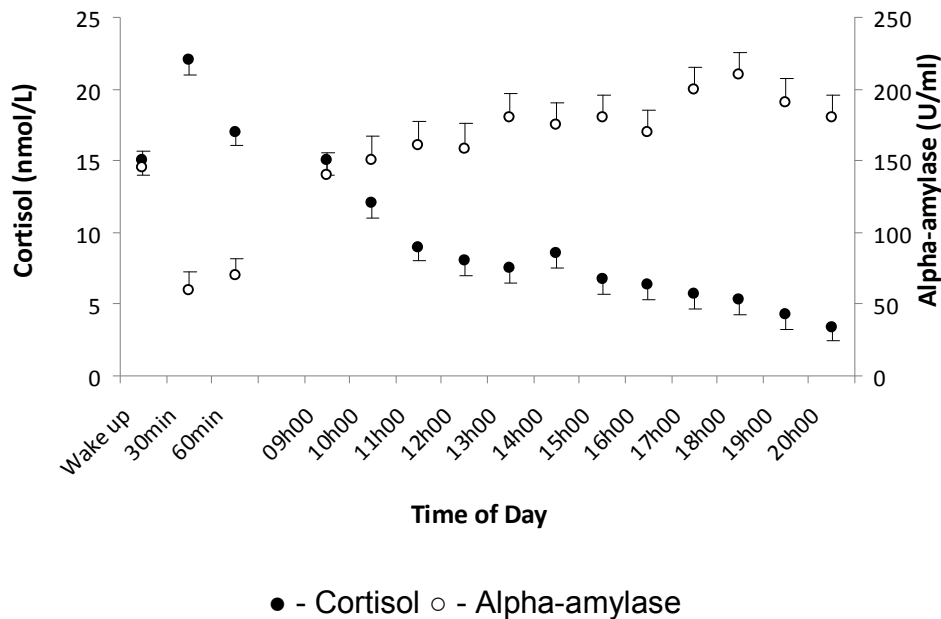
### **Alpha-Amylase and sympathetic activity**

Alpha-amylase, one of the principle salivary proteins, is a calcium containing metalloenzyme which is produced by the serous acinar cells of the parotid and submandibular glands (Takai **et al.**, 2004). Amylase makes up 10-20% of the total salivary gland protein production which is primarily synthesized in the parotid gland (Zakowski and Bruns, 1985). It is well accepted that salivary cortisol is a useful parameter for the assessment of the functioning and reactivity of the HPA axis (Kirschbaum and Hellhammer, 1994). However, it has been suggested that the use of salivary cortisol alone, may not be adequate in gaining a comprehensive understanding of the human stress response (Granger **et al.**, 2007).

Alpha-amylase, which has been identified as an accurate and reliable measure of sympathetic adrenal medullar system (SAM) activation (Granger **et al.**, 2007; Bosch **et al.**, 1996) is increased by a number of physiologically and psychologically based stress tests (Chatterton **et al.**, 1996). The activation of the human stress response by these stressors causes a string of neuron-endocrine and nervous system responses (van Stegeren **et al.**, 2008). The SAM as well as the catecholamines (epinephrine and norepinephrine) in conjunction with the glucocorticoids plays a pivotal role in homeostasis during periods of stress. Therefore, it can be used as a suitable indicator of autonomic activity as salivary secretion is a product of the stimulation of salivary glands by the sympathetic and parasympathetic nervous systems (Garrett, 1999). Chrousos and Gold (1992) argue that the stress response has at least two major principles: the HPA axis involving the secretion of cortisol into circulation, as well as the activation of the sympathetic nervous system and the concurrent release of catecholamines. Therefore the simultaneous measurement of alpha-amylase and cortisol would be complimentary in providing a more comprehensive evaluation of the stress response (Kirschbaum and Hellhammer, 1989). While it is clear that both the HPA and SAM work in coordination to produce the changes associated with stress, alpha-amylase and cortisol levels do not seem to correlate (Chatterton **et al.**, 1996; Kivlighan and Granger, 2006; Nater **et al.**, 2006). It has been considered that increase in SAM activity have been associated with stressors that are controllable, where the increases in HPA axis activity are related to emotionally stressful or uncontrollable situations (Henry, 1992; Kivlighan and Granger, 2006)

It is critical to have an understanding of two major concepts prior to the use of hormones as biological markers of stress. These include the diurnal course of hormones as well as the determinants of this course (Nater **et al.**, 2007). Figure 5 shows an example of the diurnal course of both cortisol and alpha-amylase. An understanding of the diurnal course of alpha-amylase makes it possible to choose an appropriate time to collect samples, as well as selecting an

appropriate sampling strategy (Nater **et al.**, 2007). It has been found in a number of studies (Rantonen and Meurman, 2000; Artino **et al.**, 1998) and illustrated in Figure 5 (Nater **et al.**, 2007) that alpha-amylase has a distinct diurnal rhythm, with a strong decrease in alpha-amylase production immediately post awakening; thereafter there is a steady increase throughout the day. The diurnal profile of alpha-amylase has also been found to be robust to change against influencing factors; it is however influenced by age (Nater **et al.**, 2007). There are no effects of eating and drinking on the levels of alpha-amylase, although drinking a CHO rich beverage has previously been associated with increased levels (Squires, 1953), and the use of an alarm clock, which is independent of quality of sleep, has been found not to significantly affect the diurnal course of alpha-amylase. This robust nature of the hormone makes it a useful measure, especially for the purpose of field studies, where extraneous factors affect the reliability of the results.



**Figure 5: Diurnal course of salivary alpha-amylase and cortisol concentrations (data shows mean and standard deviations)**  
(Taken from Nater **et al.**, 2007)

There has also been a relationship found between physiological activity (Steerenberg **et al.**, 1997), chronic stress and stress reactivity on the diurnal course of alpha-amylase, showing that increased stress levels elicit increased momentary salivary alpha-amylase levels (Nater **et al.**, 2007, Nater and Rohleder, 2009). The effects of age are evident, with there being a less pronounced increase over the day with an increasing age (Nater **et al.**, 2007).

#### PSYCHOLOGICAL RELATED DIAGNOSTIC MARKERS OF OVERTRAINING

Training for an event such as the Ironman triathlon requires not only physical energy but also emotional energy. While there is much doubt over the objective nature of physiological markers to diagnose the suboptimal training (Fry **et al.**, 1991; Hooper **et al.**, 1995; Urhausen and Kindermann, 2002), there has been a reoccurring agreement by sports scientists that psychological markers including impaired mood state and subjective complaints can be used, in a prospective manner, to assess the signs and symptoms of overtraining (Urhausen and Kindermann, 2002; Foster, 1998; Hooper **et al.**, 1997; Verde **et al.**, 1992; Gutmann **et al.**, 1984; Morgan **et al.**, 1987). Nideffer (1988) stated that psychological stress can undoubtedly be a major factor in contributing to the aetiology of the OTS, while O'Brian (1988) emphasised that overstressing an athlete, mentally and/or physically will result in a state of overtraining. These signs and symptoms include increased negative mood factors such as tension, depression, anger, fatigue and confusion and decreased positive mood factor, vigor, (Hooper **et al.**, 1997). These mood states make up the emotional parameters of the profile of mood states (POMS) questionnaire.

#### **Profile of mood states (POMS)**

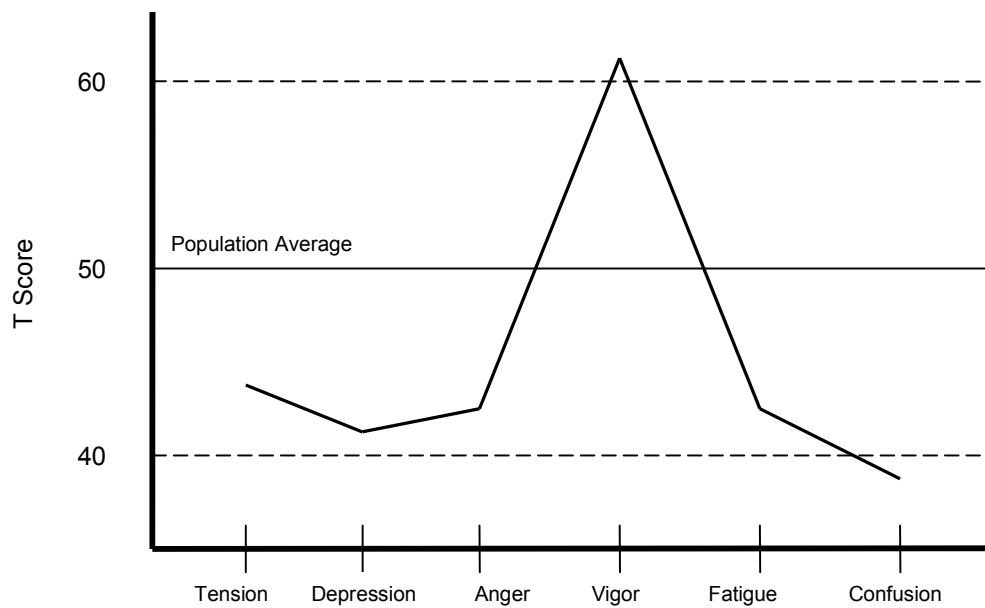
The Profile of Moods States questionnaire was first published in 1971 (Lambert and Borresen, 2006). It was developed as a self-completed test of mood, including mood state as well as emotion. Further, changes in mood state could be gained by taking a prospective analysis approach (McNair **et al.**, 1971). While the questionnaire was first developed for counselling and therapy patients, it has

been manipulated to be utilised in sporting scenarios, and has become a widely used psychological diagnostic marker of overtraining. The questionnaire has been extensively used to study the relationship between mood states and athletic performance (Verde **et al.**, 1992; Urhausen and Kindermann; 2002; Silva **et al.**, 2008). The psychological results gained have been shown to be the one simple measure of overtraining which shows a consistent response (Verde **et al.**, 1992). Physical activity has been related to both improvements and decrements in mood states (O'Connor and Puetz, 2005). During training, mood states have been found to directly relate to the changes in training volume and intensity (Hooper **et al.**, 1997). It has been reported that elite and highly trained athletes score below the average population scores on a number of the POMS scales (tension, depression, anger, fatigue and confusion), and approximately one standard deviation above the average population for the positive mood state, vigor (Morgan **et al.**, 1987). This profile has been referred to as the 'iceberg profile' due to the shape of the graph shown in Figure 6.

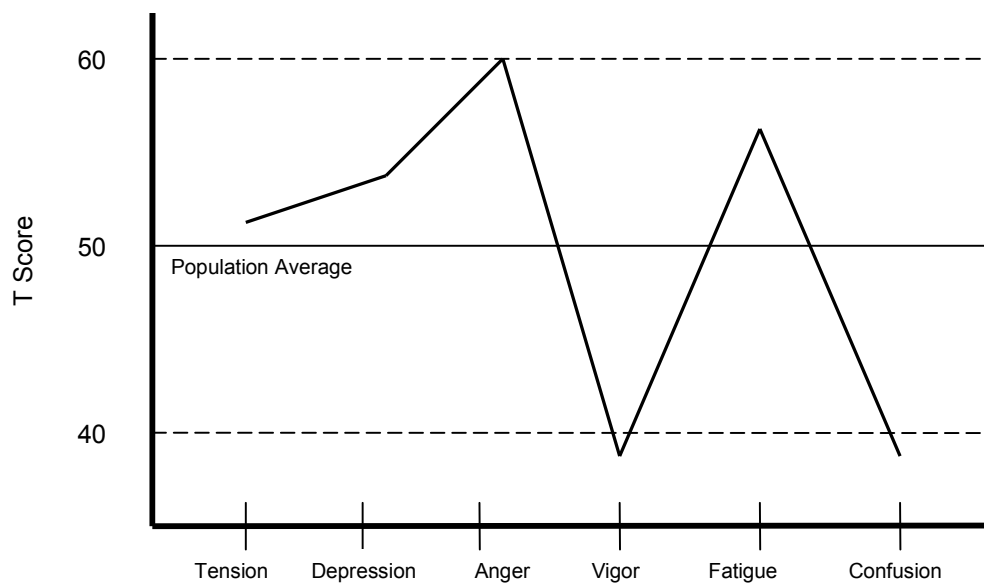
### **Iceberg profile**

The classical iceberg profile (Figure 6) has been found to be influenced by overreaching and overtraining; the changes which have been proposed as a result of overtraining includes a significantly decreased score for vigor (positive mood score) and a significantly increased score for fatigue (Verde **et al.**, 1992). The other mood states including tension, depression, anger and confusion, have found to be negatively affected by excessive training, however they do not show significantly different results to 'normal' athletes (Verde **et al.**, 1992). Overtraining has been found to significantly affect the mood profile of an athlete showing a dramatically differed profile known as the 'inversed iceberg profile' which can be seen in the Figure 7 (McNair **et al.**, 1991; Morgan **et al.**, 1987). The scores can also be used to determine a total or global mood score, calculated by; sum of the negative mood scores, minus the positive mood score (McNair **et al.**, 1991).

The POMS questionnaire has been used to aid in the prevention of overtraining in a number of athletes, in a number of sporting disciplines (Eichner, 1995). A consistent finding has been the profound change in global/total mood amongst overtrained athletes (Morgan **et al.**, 1983), however due to individual difference this pattern varies considerably from athlete to athlete (Smith, 2000).



**Figure 6: The 'iceberg profile' of a 'normal' athlete**  
(Taken from Morgan **et al.**, 1987)



**Figure 7: The inversed 'iceberg profile' of an overtrained athlete**  
 (Taken from Morgan *et al.*, 1987)

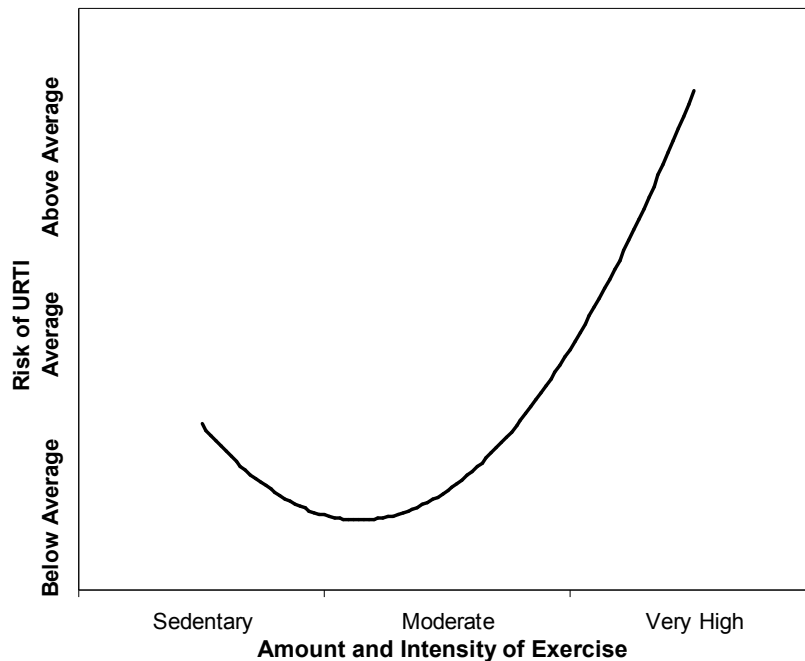
Psychological stress related to overtraining has been found to contribute to negative alterations in the nervous regulation. These stresses may not only be a result of training and competition, but may also be influenced by peer pressure and other unrelated life stresses (Birch and George, 1999). The changes in mood state that occurs due to a result of overtraining are associated with a number of cardiovascular, enzymatic, endocrine, hormonal and hypothalamic changes, which when observed in conjunction with a reduction in functional capacity (Morgan *et al.*, 1986).

#### OVERTRAINING AND OVERTRAINING

Athletes get sick for a number of reasons; these reasons are related to an exposure to pathogenic bacterial or viral agents (Foster, 1998). Anecdotal evidence has suggested that there is a prevalence of illness amongst overtrained athletes (Fry *et al.*, 1991; Lehmann *et al.*, 1993; Smith, 2000), therefore resulting in an increased susceptibility to colds, influenza, allergies, increased healing

time, and headaches as well as gastrointestinal disorders (Fry **et al.**, 1991). There is also an increased prevalence of self reported incidences of infections of the upper respiratory tract after bouts of strenuous exercise (Gabriel **et al.**, 1998), indicating the need to longitudinally record any changes in signs and symptoms of infection.

During high volume training mild tissue trauma to active musculature occurs. Adequate recovery allows for adaptation and an improvement in athletic performance (Margonis **et al.**, 2007). In the case where this adaptive process is not permitted and insufficient recovery is allowed, muscle trauma develops into a more severe state. Athletes therefore develop an inflammation (muscle damage) response to excessive exercise (Smith, 2000). This overtraining-induced muscle damage is associated with the inflammatory response which has been characterised by an increased susceptibility to infectious disease and the function of the immune cells (Margonis **et al.**, 2007). Newsholme (1991) proposed a theory that excessive exercise resulted in a reduced blood levels of the amino acid, glutamine. This reduction was responsible for the frequently observed impaired immune system function (Smith, 2000). The relationship between exercise involvement and upper respiratory tract infections (URTI) has been modelled using the 'J' curve (Figure 8), which proposes that while there is a decrease in URTI risk with moderate exercise, there is an increase in risk for URTI during periods of excessive exercise loads (Nieman, 1994b).



**Figure 8: The ‘J’ shaped curve illustrating the relationship between varying exercise load and risk of URTI**  
 (Taken from Nieman, 1994)

The reasons behind the high prevalence of illness amongst overtrained athletes remain unclear; however they are most likely due to the impairment of the immune system (Smith, 2000; Fry **et al.**, 1991). Although the function of the immune system is enhanced by moderate exercise, the excessive exposure to high intensity and high volume exercise that is associated with the OTS has an immunosuppressant function (Nieman, 1994a).

#### ADDITIONAL RISK FACTORS

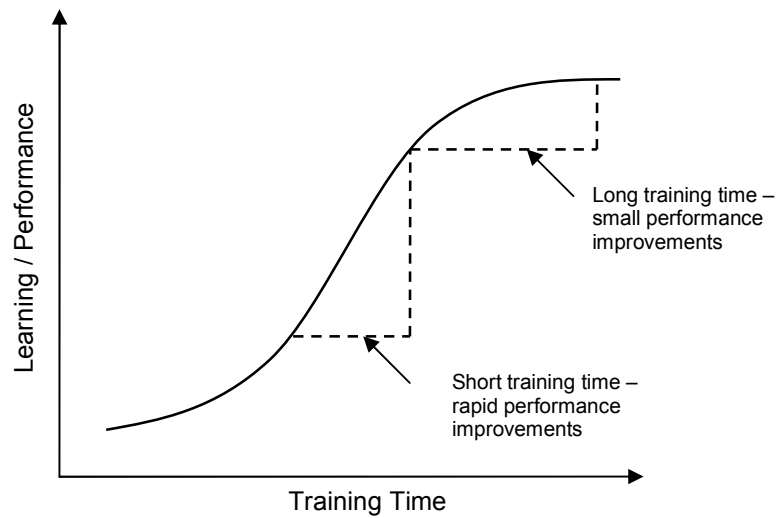
##### **Performance level**

Athletes at all levels of performance ability there are risks of developing the signs and symptoms of overtraining, and it is this disorder that often prevents many sportsmen and sportswoman from becoming elite (Fry **et al.**, 1991). It is often the case that inexperienced, amateur athletes attempt to follow training programs

designed for elite level athletes, inevitably resulting in the development of the OTS (Falsetti, 1983). Due to the 'after the fact'/retrospective role, described by O'Toole (1989), that science has played in research, there is a need for sports scientists to take a prospective approach to research, aiding in the determination of diagnostic tools for prevention of the OTS.

Athletes, amateur or elite, who are training without coaching for an event such as the Ironman triathlon, place themselves at undue risk for developing the symptoms of overtraining (Noakes, 2001; Fry *et al.*, 1991). A coach or sports scientist understands the progression of the OTS and can therefore adjust a training program, where necessary, to suit individual adaptations to training loads. The consequent failure to include adequate periods of regeneration into training regime, disturbing the balance training/competition and recovery, will result in the OTS (Fry *et al.*, 1991).

Throughout the training progression athletes follow the classical learning curve, as can be seen in Figure 9. This shows that amateur athletes have significant increases in performance in a relatively short period of training time with only moderate investments in training. In contrast, more experienced elite athletes have small gains in performance over substantially longer periods of training. These small gains are a 'fine tuning' of skills rather than a learning of new skills (Feigley, 1984). This puts the elite athletes at a greater risk of overtraining due to their perceived need to train harder for small improvements in performance (Feigley, 1984). This decreased performance feedback from training provides a potent source of demotivation.



**Figure 9: The classical learning curve**

(Taken from Feigly, 1984)

### **Experience**

It has been frequently observed that athletes taking part in ultraendurance events tend to be older than athletes taking part in shorter events (Glance **et al.**, 2002; Speedy **et al.**, 2001). There is also more variation (20-50 years) than those athletes taking part in single sport activities which has been attributed to athletes turning to triathlon after years of single-sport participation (Roalstad, 1989). Athletes with an increased number of years of participation in any sport have an enhanced awareness of the consequences of training and participation in a sport (Feigley, 1984). This experience, which shows greater maturity and emotional balance, is paramount in an athlete's understanding of their body as the aches and pains associated with elite level training are substantially greater than at an amateur athlete level.

### **NUTRITION**

Competition in ultraendurance events, as well as the training prior to these events, places athletes under huge physiological demands. Although athletes

undertake extensive training regimes during the preparation for these demands, a number of competitors do not complete the race (Zalcman **et al.**, 2007). Nutrition during this type of ultraendurance sport, training and competition, is paramount for performance (Bentley **et al.**, 2007).

During the extensive training nutritional (macronutrient and energy) intake is imperative to performance (Zalcman **et al.**, 2007). The ingestion of adequate carbohydrates (CHO), proteins and fats is necessary to prevent chronic fat weight loss and impaired performance (Applegate, 1989), maintain glycogen stores for recovery and regenerate and build tissue (Zalcman **et al.**, 2007), as well as supply the essential fatty acids and vitamins (Position of the American Dietetic Association, Dieticians of Canada, and the American College of Sports Medicine, 2000). Endurance athletes manipulate their dietary intake to meet the requirements of often multiple day training sessions and competition (Holly **et al.**, 1986). These multiple sessions place excessive stress on the individuals involved and even a high CHO diet may not adequately replace glycogen stores. This is due to the 24-hour period required for normalisation of muscle glycogen (Costill **et al.**, 1971).

Carbohydrate (CHO) intake and overtraining interrelate. Eichner (1995) studied the effects of CHO intake and overtraining, finding that amongst a group of swimmers (n=12), who doubled their training distances for a period of ten days, only four of these athletes became fatigued and swam slower than usual times. The fatigued athletes were found to be the athletes who habitually consume less carbohydrate and therefore had lower muscle glycogen stores, putting them at a greater risk of overtraining. This under-consumption of the necessary quantities of CHO has been found to be common amongst athletes who train under the 'slim to win' approach, whereby athletes intentionally reduce CHO intake to lose body mass for performance purposes. However this body mass loss comes from a loss of muscle mass causing performance impairment due to the use of protein

stores for energy production rather than muscle glycogen stores (Burke **et al.**, 2001).

Grandjean (1989) found that the intake of CHO amongst athletes generally tended to be lower than necessary, with runners, gymnasts and swimmer's intake being a major concern. Ultraendurance triathletes however, have been found to eat more wisely due to the endurance athletes having the tendency to 'graze' on snack foods, with 30% of daily energy intake coming from these snacks (Houtkooper, 1992).

Increased CHO intake in preparation for a competition with the ultraendurance nature of the Ironman requires athletes to maximise their CHO stores prior to the race in order to reduce time to fatigue (Noakes, 2001). One of the most popular methods of increasing muscle glycogen stores is by carbohydrate loading or carbohydrate supercompensation (McArdle **et al.**, 2001). While this process does assist in maximising CHO stores, there are a number of negative aspects behind its practice. CHO is a heavy fuel in comparison to fats, storing 3g of water for every gram of stored CHO. Any extra load adds to the energy cost of locomotion such as running and may outweigh the positive effects of the additional stored muscle glycogen (McArdle **et al.**, 2001).

## **SUMMARY**

Ultraendurance exercise training results in a number of physiological, psychological, hormonal and performance adaptations. These changes can be monitored in a longitudinal nature over a period of time to assess whether or not these adaptations are optimal in sport-specific performance. Due to the immense volume of training performed by the athletes participating in the Ironman triathlon, there is a great deal of concern surrounding the balance between optimal training and suboptimal training techniques. The basic principle of training is overload; however this principle can lead to excessive overload, resulting in functional overreaching and the overtraining syndrome. The present study therefore sought

to gain further insight into the training and associated consequence of that training in a group of athletes of varying performance levels.

## CHAPTER III

### METHODOLOGY

#### INTRODUCTION

Successful completion of the Ironman triathlon requires a great deal of training which usually begins approximately eight months prior to the event (O'Toole, 1989). Previous research has focused primarily on the time period immediately prior to, and including, the event (Barron **et al.**, 1985; O'toole **et al.**, 1989; Dengel **et al.**, 1989; Applegate, 1989; Speedy **et al.**, 2001; Laursen and Rhodes, 2001; Ginsburg **et al.**, 2001; Bentley **et al.**, 2002; Bentley **et al.**, 2007; Neubauer **et al.**, 2008). However, due to the immense volume of training performed by the athletes, it is pertinent that more information in the months of intensive training is obtained in order to better understand the impact this has on athlete well-being and performance.

Over the past decade the majority of the athletes taking part in the Ironman have been amateurs rather than elite professionals (Butts **et al.**, 1991; Coutts **et al.**, 2007b). Regardless of this, a great deal of the research has been concentrated on World class athletes competing at the World championships in Kona, Hawaii (O'Toole and Douglas, 1989; Ginsburg **et al.**, 2001; Lepers, 2008). In addition, there has been a significant difference found between training for the World Championships and training for other Ironman triathlon events around the world (Gublin and Gaffney, 1999). This highlights the importance of including an athletic sample which covers a broad range of athletes, from amateur to elite. The present study therefore analysed a number of performance changes and fatigue markers, in a wide range of athletes, in the pursuit of a holistic, efficient, accurate and cost effective assessment of the impact of training for an event of this nature. Although the attempted diagnosis of the overtraining syndrome

(OTS) was an important component of this study, the main purpose was to assess specifically what training athletes were actually doing in preparation for the event as well as what impact these individual's training programmes had on well-being, training and certain physical, psychophysical and performance responses.

## **'PILOT STUDIES**

Several pre-test studies were conducted prior to the start of the experimental data collection period. These studies were performed to provide insight to the selection and manipulation of the experimental protocol and procedure of the project. Initial consultations were conducted with researchers from the University of Cape Town (UCT), Tshwane University of Technology (TUT) and the University of Kwa-Zulu Natal (UKZN), Westville Campus. These individuals were contacted due to their previous research experience with either the Ironman triathlon and/or the phenomenon of the overtraining syndrome. The consultations proved invaluable in the identification of the existing void in the literature regarding training techniques, the overtraining syndrome and the Ironman triathlon.

The 'pilot' testing stage was also used to decide upon the tests that would be used for the study. Consultations held with researchers from UKZN further aided in the development of the longitudinal nature of the project. This included the duration of the hormone collection period and the frequency of collection throughout the project. This sampling frequency was however restricted by the financial resources available. During the pilot testing the phases of training used to determine training loads in relevant phases were decided upon, these included the Base, Heavy, Taper and Race phases. The use of an age and sex-matched control sample, to compare to the results of the athletic sample, was also decided during this period.

## **EXPERIMENTAL DESIGN**

Jones and Carter, (2000) stated that understanding the adaptations resulting from endurance training would enable the identification of the point at which there were no longer improvements and possibly decrements in performance capacity, highlighting suboptimal training techniques. While the single best predictor of suboptimal training appears to be a decrement in performance, there are a large number of predictors which can be included and which have typically been investigated with respect to the diagnosis of the overtraining syndrome (Smith, 2000). With respect to overtraining research, several diagnostic markers have been developed which can be extended to the assessment of suboptimal training. It has been contended that these need to include a broad range of parameters. It is therefore imperative to take a holistic approach in the identification of the signs and symptoms of suboptimal training and/or overtraining. The present study considered this by taking into consideration the physiological, psychological, biochemical and immunological domains. The significance of taking this approach was that certain tests could be more sensitive diagnostic markers of suboptimal training than other tests and therefore could have a more effective diagnostic power than markers from other domains. Furthermore, the permutation of two or more of these tests could prove to possess a powerful diagnostic combination.

Athlete progress was assessed using a number of tests throughout the data collection period. Most of these tests have been used in an overtraining context and are therefore explained as such. This is despite the fact that this study was exploratory in nature and assessed numerous levels of athletes training for the same event. Whether these athletes would get overtrained was not certain and therefore the purpose was to assess whether training was optimal (or suboptimal) with the possibility that some athletes may have become overtrained. Tests included a day-to-day graded training log, which was adapted from the Sports Science Institute of South Africa's training diary to monitor signs and

symptoms of overtraining. Derman **et al.** (1997) used this day to day training log to identify the possible markers of chronic fatigue, showing that continual assessment is paramount in the diagnosis of non-functional overreaching and the overtraining syndrome. The training log used in the 1997 study, which already integrated a number of variables used to assess the athletes during training (these included; 'resting' heart rate, quality and quantity of sleep, body mass, postural dizziness, symptoms of illness and general mood), was adapted for use in the current study by increasing its detail to suit an ultraendurance triathletes training schedule, namely multiple daily training sessions.

Heart rate (HR) measures can be useful in determining physical and emotional stress, be it acute or chronic. It is however difficult to obtain measures of true resting HR, therefore, the measurement of waking HR has been found to be the most accurate method due to the limited number of factors which could influence the individual prior to recording (Noakes, 2001). The use of 'resting' HR as a diagnostic marker of the OTS has been questioned in past research, where contradictory results have been found (Urhausen and Kindermann, 2002). However, an increase in submaximal exercise heart rate, resulting in decreased performance, has the ability of indicating infectious disease, bodily stress as well as reduced exercise tolerance, a possible prelude to the OTS (Urhausen and Kindermann, 2002). This illustrates the importance of following the HR changes of athletes during exercise and at rest in a prospective manner over the duration of the project.

During periods of high training load, adequate macronutrient and energy intake must be sustained, this is especially important for carbohydrates and proteins (Zalcman **et al.**, 2007). While ultraendurance triathletes express a keen interest in the benefits of optimized nutrition habits (Burke **et al.**, 1991), inadequate caloric intake during endurance training does occur due to the difficulty for triathletes to match the requirements of daily training needs. This leads to reduced muscle glycogen stores, reduced levels of protein necessary for tissue

regeneration and reduced fat stores resulting in chronic weight loss and therefore possible decrements in performance ability (Applegate, 1989). This shows the importance of monitoring body mass changes when formulating optimal training techniques for ultraendurance athletes (Zalcman **et al.**, 2007). As such, in the present study, athletes recorded body mass on a daily basis as an aid in assessing caloric intake adequacy (Applegate, 1989).

To allow for adequate training time during the day, the majority of athletes are required to spend early hours of the day training. This disturbs the diurnal rhythm of daily life and can result, to some extent, in sleep deprivation. This deprivation of sleep is stressful and may be further effected by excessive physical and emotional stress (Shephard, 1984). Not only does the limited time available for the required long training sessions affect the sleep patterns of the athletes training for the Ironman but a number of authors (Urhausen **et al.**, 1998; Urhausen and Kindermann, 2002) have found that sleep disorders are associated with overtraining. Therefore a measurement of quality and quantity of sleep was included on the day to day training log.

Due to the diverse nature of the training regimes followed by the athletes, the quantification of training performed or training load was necessary. Although training load is not the only training-related variable to contribute to performance enhancements or decrements, its quantification was essential to successfully relate training load to performance and fatigue markers. This quantification was performed by using two methods; the objective TRIMP (Training Impulse) method developed by Banister and co-workers (1991) and the subjective 'Session RPE' (Rating of Perceived Exertion) method developed by Foster **et al.** (1996). This subjective method was developed to simplify the quantification of training load, and was aptly used where subjects could not train with a heart rate monitor, usually during the majority of swim training sessions (Borresen and Lambert, 2008).

While it has been an issue for sports scientists to agree upon an accurate and reliable physiological marker of fatigue, psychological stress has been determined as a major contributing factor to the aetiology of overtraining (Nideffer, 1988). There has been a re-occurring agreement by sports scientists that psychological markers such as impaired mood state and subjective complaints can be used, in a prospective manner, to assess the signs and symptoms of overtraining (Gutmann **et al.**, 1984; Morgan **et al.**, 1987; Verde **et al.**, 1992; Hooper **et al.**, 1997; Foster, 1998; Urhausen and Kindermann, 2002). This has been shown by the profound change in global/total mood amongst overtrained athletes (Morgan **et al.**, 1983). However, due to individual difference, this pattern varies considerably from athlete to athlete (Smith, 2000). Therefore, general mood state was recorded in the training log on a daily basis by the athletes and the changes were analysed over a longitudinal period.

The Profile of Mood States (POMS) questionnaire, extensively used in studies of exercise, fitness and sports science has over 250 citations in these fields, and has been used repeatedly to aid in the prevention of the development of the overtraining syndrome in a number of athletes and sporting disciplines (Eichner, 1995). Physical activity has been related to changes in mood states (O'Connor and Puetz, 2005), and mood states have been found to directly relate to the changes in training volume and intensity (Hooper **et al.**, 1997). These psychological results have shown to be one of the most reliable, yet simple, measures of overtraining (Verde **et al.**, 1992). Therefore to add to the subjectively graded general mood state score found on the day to day training log the POMS questionnaire was completed weekly.

Monitoring acute changes in an athlete's performance has been found to aid in the prevention of entering an overtrained state (Derman **et al.**, 2007). It has also been stated that future studies should examine the relationship between the practical tests of performance and the athletes exercise capacity over a prolonged period (Coutts **et al.**, 2007b). Mark Allen, a six time winner of the

Ironman World Championship gauged performance improvements or decrements using an 8km submaximal time-trial (Noakes, 2001). The intensity of the time-trial was determined by the Maffetone formula. This formula was derived by the well known triathlon coach Phillip Maffetone (1996) renowned for his advancements in heart rate based training. A submaximal intensity time trial was selected in place of a similar test of maximal intensity, largely because of the unforgiving nature of an 'all out' maximum intensity time trial, to which ultraendurance athletes are also not habitually accustomed. Further, maximal testing could place subjects at a greater risk of overtraining-related illness and injury (Coutts **et al.**, 2007).

While hormone sampling has been problematic in the past (Schwartz **et al.**, 1998), the use of salivary hormone analysis has allowed a non-intrusive manner in which to analyse these biological markers of stress (Levine **et al.**, 2007). Ultraendurance exercise has been found to have an effect on the functioning of the hypothalamus and the autonomic nervous system, therefore affecting the Hypothalamic Pituitary Adrenal axis, causing decreased adrenal responsiveness (Persson **et al.** 1980; Wittert **et al.**, 1996). Salivary cortisol was therefore selected as it has been suggested that the use of free cortisol, 30 minutes post awakening, has been identified as a reliable marker of HPA activity (Grossi **et al.**, 2005). Further, the use of an additional hormonal indicator of the human stress response, or sympathetic adrenal medullar system (SAM) activation would allow for a more comprehensive evaluation of the stress response of ultraendurance training (Kirschbaum and Hellhammer, 1989). The hormones used for the purpose of the study were therefore salivary cortisol and salivary alpha amylase. They were collected on selected days during the training period encompassed by the projects data collection phase (November 2008 – April 2009). The sample dates can be seen on the sample collection data sheet (Appendix C). These hormones were selected in conjunction with the Head of Research at Tshwane University of Technology's sports science department, as predictors of metabolic stress and the results were used to aid in the determination of the validity of the

psychological questionnaire, training diary information and time-trial performance as markers of suboptimal training techniques

### DESIGN MATRIX

The project included two groups, an athlete sample and a control sample. These two samples were compared during two periods, namely the training period and the competition period. The athlete sample was analysed longitudinally over a six month period (five months training; one month competition), while the control sample was analysed for a period of one month and compared to all months of the athlete sample. During this period training, psychological, biochemical, immunological and performance responses were assessed. As limited changes to responses were expected amongst the control sample during data collection, a statistician was consulted to assist with the comparison between the control sample's data and the athlete's data.

**Table III: Two-by-two Matrix used to establish the experimental design**

	Training Period	Competition Period
<b>Athlete</b>	Training Physiological Psychological Biochemical Immunological Performance Responses	Physiological Psychological Biochemical Immunological Performance Responses
<b>Control</b>	Physiological Psychological Biochemical Immunological Responses	Physiological Psychological Biochemical Immunological Responses

(Training Period encompasses all measurements taken from November to the day prior to the race)

(The competition period encompasses the Race day and the two weeks post race)

## **MEASUREMENTS AND EQUIPMENT PROTOCOL**

Athletes (n=18) were handed a hard copy folder as well as an electronic data collection file. This file can be seen in the Appendix C. Control subjects (n=15) were handed a hard copy of the data file. This was largely because these individuals were locals in the area. This file included the daily training log, POMS questionnaire and space to record the details from the submaximal time trial and the hormone analysis. There were a number of measurements which needed to be documented. The control group completed an abridged version of the training log which did not include space to fill out training details (Appendix D). In a similar fashion, these control subjects completed a weekly POMS questionnaire, and produced salivary samples at three points. The control sample were taken from a population of individuals who perform equal to, or less than the American College of Sports Medicine's (ACSM) guidelines for minimum weekly activity, being 30 minutes of moderate exercise on most, if not all of the days of the week (Balady **et al.**, 2000). Due to the sedentary nature of the control group, significant exercise-related changes in psychological and hormonal variables were not expected and therefore it was not necessary to analyse data from these subjects for the full duration of the project. Rather, these subjects were analysed over the Ironman period itself (1<sup>st</sup> April – 19<sup>th</sup> April), producing saliva samples on three occasions (5<sup>th</sup>, 12<sup>th</sup> and 13<sup>th</sup> April).

## **TRAINING LOG**

An adapted version of a training log designed by the Sports Science Institute of South Africa, previously used by Derman **et al.** (1997) to study clinical fatigue in athletes (Appendix C), was completed by the participants. This was carried out on a daily basis for the duration of the data collection period. The training log encompasses a range of markers to aid in the diagnosis of the OTS, these include ten categories which were divided into three periods of measurement. Those measured in the morning on awakening (waking heart rate, hours of sleep,

sleep rating and body mass), those measured throughout the day (postural dizziness, general mood rating and symptoms of illness), and those recorded after the completion of training sessions (training details, perceived training effort and muscular discomfort during training).

The majority of triathletes will begin aerobic conditioning or base training approximately eight months prior to the relevant Ironman competition (O'Toole, 1989). In the case of the 2009 Ironman South Africa, this would have been in August 2008. This study, however, focused more on the periods of higher training load which follows the initial base training phase. Data collection therefore began in November 2008 (five months prior to the event). Individual differences found in training patterns and durations varies from athlete to athlete, therefore athletes were required to submit a proposed training regime for their preparations for the event in 2009. This information made it possible to determine predicted training loads for the athletes taking part in the study, as well as aiding in the determination of the appropriate dates for the salivary hormone collection.

The continuum of training was therefore divided into three phases (Table IV), determined during the pilot testing phase of the project, to aid in the identification of the phase of training during which athletes were at most risk for developing signs and symptoms of suboptimal training and possibly the OTS (Fry *et al.*, 1991). The base to heavy training phase encompasses training performed prior to, and during, their heavy training regime. Measurements were taken during a reduced exercise intensity training period (recovery week) at the beginning of November in an attempt to reflect a reference level on which to quantify the subsequent measurements. Exercise involvement prior to this sample was recorded as a reference level to Ironman specific conditioning. The base phase usually consists of approximately the first three to four months of training. The majority of the training sessions performed during the base phase are of low intensity, with a progressively increasing frequency and duration throughout the training phase (Noakes, 2001). During the next phase of training, the heavy

training phase, volume and frequency of training sessions continue to increase with the addition of high intensity sessions closer to the event, therefore placing athletes at greater risk of developing the OTS (Lehmann *et al.*, 1993). Sampling during this phase occurred at the beginning of the following months: January, February and March. The taper period for the race began approximately midway through March and is demonstrated by a marked decrease in training load in preparation for race day (Mujika *et al.*, 2000). The race and recovery phase constitutes the race day as well as the two weeks post event.

**Table IV: Phases of training proposed by the study (base, heavy, taper and race phase)**

Training Phases					
November	December	January	February	March	April
Base to Heavy training				Heavy training to Taper	Race to recovery

RECORDED ON AWAKENING

### **Waking Heart Rate**

The measurement of waking heart rate (HR) was recorded each morning prior to subjects getting out of bed. After waking, subjects were instructed to lie relaxed for approximately five minutes. HR was then either taken by manual method, which was either palpation at the carotid artery or at the radial artery. To take HR by manual method, subjects placed their index and middle fingers on the relevant artery and palpated for a pulse. Once this was achieved athletes counted the number of beats during a one minute period (Figure 10). Alternatively subjects used a heart rate monitor. In the case where subjects opted to use HR monitors to record waking HR, subjects had to fit the electrode strap around the chest at the level of the inferior border of the pectoralis major and in line with the apex of

the left ventricle, situated slightly to the left of the mid-centre of the chest. The HR data, collected by the strap, was then transmitted to the watch which serves as means of display for the HR data.



**(a) Carotid artery**



**(b) Radial artery**

**Figure 10: Manual method heart rate palpation sites**

#### **Ratings of Sleep (Quality and quantity)**

Sleep quality or the level of sleep disturbance is just as important as the quantity of sleep obtained. This allows for the subjective rating of the level of sleep deprivation when attempting to quantify the restorative quality of the sleep state. It was therefore important to take into consideration these two variables of sleep. Therefore sleep quality was graded from 1 – 4 according to a subjective rating performed by the subjects (Table V).

**Table V: Grades of quality of sleep**

<b>Grade</b>	<b>Quality</b>
<b>1</b>	Uninterrupted sleep, feeling refreshed.
<b>2</b>	Interrupted sleep, however <u>do not</u> require more sleep
<b>3</b>	Interrupted sleep, <u>do</u> require more sleep
<b>4</b>	Interrupted sleep, feeling terrible

Sleep quantity or the hours of sleep was recorded and rounded off to the nearest 30 minutes, e.g. 6h30, 7h30, 8h00. Any sleep other than that of overnight sleep (e.g. afternoon sleep) was added to the hours of sleep obtained during that day. This figure was filled in on the daily training log.

### **Body Mass**

Subjects were required to measure their body mass, dry and nude, on a daily basis. Therefore, changes in body mass could be determined during the course of the project. The measurement was performed after awakening, prior to consuming anything per mouth such as food or liquids. If necessary, subjects went to the toilet and then recorded body mass. Where possible, mass was measured on the same scale throughout the data collection period to avoid discrepancies in scale calibration.

RECORDED THROUGHOUT THE DAY

### **Postural Dizziness**

Dizziness or light-headedness felt when standing from a seated or lying position is a proposed symptom of the overtraining syndrome and is usually associated with poor nutrition habits (Derman **et al.**, 1997). Therefore it was included in the day-to-day training log and subsequently graded throughout the day according to the level of occurrence. The grading was performed according to the levels 1 – 4 shown in Table VI.

**Table VI: Grades of postural dizziness**

<b>Grade</b>	<b>Dizziness</b>
1	No dizziness
2	Slight dizziness, disappears within 2 seconds
3	Marked dizziness, time taken to orient one's self
4	Marked dizziness, requiring sitting or lying down again

### **General Mood Rating**

Although mood rating was extensively covered by the Profile of Mood States (POMS) questionnaire, general mood ratings were also recorded. As opposed to weekly, general mood rating was obtained daily (Table VII).

**Table VII: General mood ratings**

<b>Grade</b>	<b>Mood</b>
1	Good mood
2	Feeling sad or down at times
3	Feeling sad or down most of the time
4	Feeling sad and down all of the time

### **Symptoms of Illness**

Ironman athletes are at risk of immunosuppression and therefore have a greater risk of falling ill and showing symptoms of illness, this has been identified on cyclists by Halson **et al.** (2003). Athletes were required to record any illness suffered using the relevant number(s) found in Table VIII. Athletes not suffering from illness recorded their score as a zero. Symptoms suffered that were not illustrated on the list were recorded as 8 (other) and a brief description of the symptoms was given.

**Table VIII: Symptoms of illness**

<b>Number</b>	<b>Symptom of Illness</b>
1	Sore throat
2	Cough
3	Nausea and/or vomiting
4	Diarrhoea
5	Stomach ache
6	Headaches
7	Fatigue
8	Other_____

#### RECORDED AFTER TRAINING

Once training sessions were completed, athletes were required to record the details of their training sessions. This information included training details as well as the subjective measures of perceived training effort and feelings of muscle discomfort for each session. These details were used to calculate the training load for each of the disciplines trained.

#### **Training Details**

All daily training details were recorded for each discipline; this included training distances (km) and durations (minutes) as well as the average and maximum heart rates ( $\text{bt}\cdot\text{min}^{-1}$ ) for the session. Brick sessions (running directly after cycling) were divided into appropriate disciplines (Appendix C). Heart rate results (average and maximum) for swim training were not recorded due to the difficulties with transmitter strap positioning caused by water drag. However, where possible, HR results from swim training were recorded.

#### **Perceived Training Effort**

It has been noted that possibly the best gauge of overtraining is the way in which an athlete perceives the difficulty of training (Eichner, 1995). Therefore the perceived effort of training throughout a training session could be used as a

reliable marker of the diagnosis of an overtrained state and was rated by the athletes in this study. It allows an indication of subjective feeling of perceived training effort and should be graded and recorded (Table IX).

**Table IX: Perceived effort of training**

<b>Grade</b>	<b>Perceived effort</b>
1	Felt good throughout the entire session
2	Felt good at times during the session
3	Felt tired, somewhat difficult session
4	Difficulty completing the session, very difficult

### **Muscle Discomfort**

Strenuous exercise damages muscle and the consequences of high volumes of exercise include muscle pain, soreness, stiffness and a loss of strength (Eichner, 1995). Greater muscle discomfort during exercise sessions possibly indicates that athletes have been exposed to exercise of an unaccustomed load. The magnitude of active strain imposed on the muscle fibres precipitates the damage occurred by the muscle and the resulting soreness (McArdle **et al.**, 2001). It was therefore imperative that athletes recorded any discomfort, stiffness and/or pain, experienced during training sessions. This grading aided in the determination of excessive increases in training load and the adaptation of athletes to these loads. Discomfort should be graded according to Table X.

**Table X: Perceived muscle discomfort during training**

<b>Grade</b>	<b>Pain/Stiffness</b>
1	No stiffness, no pain
2	Mild stiffness, mild pain – session unaffected
3	Moderate stiffness, moderate pain – difficulty completing session
4	Severe stiffness and pain – unable to complete session

## TRAINING LOAD

The quantification of training performed by the athletes was done using the TRIMP and Session-RPE methods. The equations used to calculate training load can be seen below.

### **TRIMP equation** (Banister *et al.*, 1991)

$$\text{Male: Duration (min)} \times (\text{HR Ex} - \text{HR Rest}) / (\text{HR Max} - \text{HR Rest}) \times 0.64e^{1.92x}$$

$$\text{Female: Duration (min)} \times (\text{HR Ex} - \text{HR Rest}) / (\text{HR Max} - \text{HR Rest}) \times 0.86e^{1.67x}$$

Where:

HR Ex = Average heart rate during exercise session

HR Rest = Heart rate during rest (waking heart rate)

HR Max = Maximum heart rate achieved during exercise session

$e = 2.712$

$x = (\text{HR Ex} - \text{HR Rest}) / (\text{HR Max} - \text{HR Rest})$

### **Session-RPE equation** (Foster *et al.*, 1996)

Duration (min) x session RPE

Due to the subjects recording a subjective 1–4 grading of perceived training effort, rather than a Borg's (1-10) RPE reading, the result (1-4), was multiplied by a factor of 1.67. This was in order to obtain a relative RPE value to be used in the Session-RPE equation.

## **PROFILE OF MOOD STATES (POMS) QUESTIONNAIRE**

This method of psychological measurement, which has been used extensively in previous sports-related literature (Verde *et al.*, 1992; Urhausen and Kindermann; 2002; Silva *et al.*, 2008), was used in the present study to determine the psychological mood state changes. The POMS score was recorded on a weekly basis throughout the data collection period. Subjects scored their moods felt

during the week and rated them accordingly on the accumulative score sheet (Appendix C).

The score sheet listed 65 feelings which were rated by the subjects from 0 – 4 in a multiple choice fashion (Table XI). The rating selected (0 - 4) correlated to the level that each subject had been experiencing throughout the week prior to completion of the questionnaire. From these ratings an assessment of six mood states can be made. These mood states included five negative and one positive mood state; namely “tension”, “depression”, “anger”, “fatigue”, “confusion” and “vigor” respectively (McNair and Heuchert, 2003). Only simple instructions were required for subjects to successfully complete the accumulative score sheet provided, therefore making POMS an ideal psychological analysis tool for the quantification of mood state throughout the project’s duration.

**Table XI: Levels of rating for the POMS questionnaire**

<b>Rating</b>	<b>Level of experience</b>
0	Not at all
1	A little
2	Moderate
3	Quite a bit
4	Extremely

### **8KM SUBMAXIMAL RUNNING TIME TRIAL**

A simple yet accurate measure of performance is to undertake a sport-specific time trial which is measured at the same level of intensity during each attempt over a period of time. An issue with the continual assessment of performance by the use of physical tests is that they can be overly stressful and therefore, in turn, promote the onset of the overtraining themselves. Performance was therefore recorded using a monthly, 8km submaximal pace running time trial.

The results were then used to record any changes in performance during the months of training prior to the Ironman South Africa, 2009. The reason for the selection of a running time trial, and not a swimming or cycling time trial, was due to the ease at which it could be performed. This is due to a number of the athletes taking part in the study being committed to work-related travel, and thus the logistics of bicycle transport and the location of a swimming pool may have made it difficult to perform either one of these time trials while away from home. Running also holds the least amount of room for influence from extraneous factors such as weather.

To ensure that subjects completed each time trial at the same relative intensity, average heart rate (HR) was monitored during the time trial session. Due to the individual differences between the subjects, each subject performed the run at a relative average submaximal HR. This HR was calculated by using the Maffetone '180 minus' formula (Appendix C). Athletes were encouraged to perform the time trial on the same course and preferably on a flat gradient at each attempt. The time trial, applied in a similar fashion to the training regime of the six time World Ironman champion, Mark Allen, was performed by the subjects on a personally selected day during the testing period (provided they were approximately 4 weeks apart), with the intensity restricted by a heart rate range specified by the Maffetone formula (Noakes, 2001).

### **SALIVARY HORMONE ANALYSIS**

Due to technological advancements it is now possible to accurately assess biomarkers of stress in saliva, a non-invasive measurement technique which does not require trained staff and specialised equipment during collection (Levine *et al.*, 2007). The measurement of salivary hormones has been widely used in endocrinology, psychobiology and behavioural medicine research studies (Kirschbaum and Hellhammer, 1994) and has been found to be a useful indicator of physical, mental and emotional stress.

Saliva sampling is a risk free method of analyzing hormones, which involves the collection of saliva using either a salivette (cotton swob) or ‘drool’ technique. The present study analysed two salivary hormones, cortisol and alpha-amylase; these have been found to be the ‘complimentary couple’ in hormonal analyses of the human stress response, focusing on the Hypothalamic-Pituitary-Adrenal (HPA) axis as well as the sympathetic nervous system respectively.

These hormones were sampled at nine points, by most athletes, during the course of the study (Table XII), and at three points by the control sample. These sampling points, which show a reflection of the subject’s hormonal changes throughout training, around the race itself as well as the two weeks following the race, were decided upon in conjunction with the head of research from the Tshwane University of Technology’s Sport Science Department who has experience with this type of analyses. In some instances subjects were unable to deliver the samples on the exact day defined by the project, however, the next possible opportunity available for sampling was used.

**Table XII: Hormonal sampling dates outlined by the study for the duration the training for an Ironman Triathlon**

October	December	January	February	March/April	
Training for Ironman				Ironman	
T1 - Baseline Assessment during reduced training <b>November</b>	T2 - <b>December</b>		T5 - <b>March</b>		T8 – <b>Finish line</b>
	T3 - <b>January</b>		T6 - <b>April</b>		T9 - <b>1.5h post finish</b>
	T4 - <b>February</b>		T7 - <b>April (day prior to race)</b>		

There are a number of issues surrounding the collection of saliva for biochemical analysis: the samples’ susceptibility to changing environmental conditions (temperature); the presence of blood in the saliva; as well as compliance to the instructions given prior to the testing. Consequently the importance of correct

sampling procedures was made clear to all subjects during a subject project orientation as well as being printed in a document of information handed out to all subjects.

#### HORMONE COLLECTION

Hormone samples were produced by subjects 30 minutes post waking on assigned days during the study, as well as immediately post race, and an hour and a half after finishing the Ironman 2009. Samples were collected by tilting the head forward, allowing the saliva to pool on the floor of the mouth, and then passing the saliva through a short straw into a 2ml Corning® polypropylene cryogenic vial (Figure 11). Approximately 2ml of saliva was necessary to comfortably complete all necessary analyses. Samples had to be kept refrigerated at -2°C before being delivered to an assigned 'drop-off' point in respective cities. These samples were transported on ice to the relevant drop-off points. They were allowed a maximum of 48 hours at this temperature (between -2 and -8°C) before they were collected from these 'drop-off' points and stored at -68.4°C until the analysis took place.



**Figure 11: Corning® 2ml polypropylene cryogenic vials used in hormone sample collection**

#### HORMONE ANALYSIS

Hormone analysis kits for the purpose of this research were purchased from Salimetrics® in the United Kingdom. Cortisol was measured using a competitive

immunoassay which has been designed specifically for the quantitative measurement of salivary cortisol. Alpha-amylase was measured using an assay kit designed specifically for the kinetic measurement of salivary alpha-amylase. On the selected day of assay, samples were thawed (on ice) and the reagents for the analyses were brought to room temperature. They were then analysed following the Salimetrics Icc<sup>®</sup> procedural guidelines.



**Figure 12: Author pipetting saliva samples during the hormone analyses**

### **Alpha-Amylase analysis**

Once the microtitre plate layout had been determined samples were thawed, then vortexed and centrifuged at 300 revolutions per minute (rpm) for 15 minutes. Samples were then pipetted into the appropriate wells, making sure not to include any particulate matter, as this could have had an adverse affect on the reaction. Samples were diluted to a concentration (1:200) with an amylase diluent (phosphate buffered solution containing a non-mercury preservative). This was performed by preparing a 1:10 dilution of the samples and amylase diluent. 10 $\mu$ L of the sample was pipetted to 90 $\mu$ L of the diluent. 10 $\mu$ L of this 1:10 dilution was

further diluted with 190µL of amylase diluent (Figure 12). The alpha-amylase substrate solution (ready-to-use liquid preparation of 2-chloro-p-nitrophenol linked with maltotriose, sodium azide, at 0.01%, was added as a preservative) was heated, for a minimum of 20 minutes, to 37°C. Alpha-amylase controls were supplied, one high concentration and one low concentration. 8 µL of these controls as well as the diluted saliva samples were pipetted (reverse pipetting technique used to prevent bubbles) into the appropriate wells. 320µL of the preheated substrate solution was added, simultaneously, to each well using a multichannel pipette. To produce the most accurate results, duplicates were analysed for all samples, only one microtitre strip was tested at a time, pipette tips were discarded after each use to avoid contamination and wells containing bubbles were repeated. The plate was then immediately placed in a plate reader and mixed at 500-600 rpm and incubated at 37°C. Optical density was read (405nm filter) in centre measurement kinetic mode at one minute and again at three minutes.

To calculate the activity of alpha-amylase in each sample (U/mL) the following calculations were performed

$$\frac{\Delta\text{Abs./min} \times \text{TV} \times \text{DF}}{\text{MMA} \times \text{SV} \times \text{LP}} = \text{U/mL of alpha-amylase activity in sample}$$

Where:

$\Delta\text{Abs./min}$  = Absorbance difference per minute

TV = Total assay volume (0.328mL)

DF = Dilution factor

MMA = Millimolar absorptivity of 2 chloro-p-nitrophenol (12.9)

SV = Sample volume (0.008mL)

LP = Light path = 0.97 (specific to plate received with the kit)

$$\frac{\Delta\text{Abs./} 2\text{min} \times 0.328\text{mL} \times 200}{12.9 \times 0.008\text{mL} \times 0.97} = \Delta\text{Abs.} \times 328 = \text{U/mL of alpha-amylase activity in sample}$$

### **Cortisol analysis**

Once the setup and layout of the anti-cortisol coated microtitre plate was determined, the appropriate wells were then pipetted accordingly with 25 $\mu$ L of either: six cortisol concentration standards (3.000, 1.000, 0.333, 0.111, 0.037 and 0.012 $\mu$ g/dL) based in a saliva-like matrix containing a non-mercury preservative. One low and one high concentration cortisol control (# and #  $\mu$ g/dL respectively), as well as the samples (saliva). Wells kept open for blanks as well as marked non-specific binding wells (NSBs) which were not coated with the anti-cortisol antibody were pipetted with 25 $\mu$ L of assay diluent (phosphate buffered solution containing a pH indicator and a non-mercury preservative). A 1:1600 dilution of the Horseradish peroxidase enzyme conjugate was prepared by adding 15 $\mu$ L of the conjugate to 24mL of the assay diluent. This solution was mixed immediately and 200 $\mu$ L was pipetted into each well using a multichannel pipette. The plate was mixed on a plate rotator for five minutes at 500rpm and then incubated at room temperature for 55 minutes. A 10x wash buffer was diluted with deionised water before being used to wash the Microtitre plate. This wash was performed by gently squirting the wash buffer into each well with a squirt bottle - this process was repeated four times. Once the plate had been blotted dry 200 $\mu$ L of tetramethylbenzidine (TMB) was pipetted into each well using a multichannel pipette. The plate was again mixed on a plate rotator for five minutes at 500rpm and incubated for 25 minutes in the dark at room temperature. After this incubation period, the stop solution (sulphuric acid solution) was added to the plate using a multichannel pipette. The plate was mixed for three minutes at 500rpm, wiped clean with a moistened lint free cloth and dried before reading in the plate reader at 450nm. The reading was taken within ten minutes of adding the stop solution. All wells were performed in duplicate to ensure the accuracy of the samples.

To calculate the concentrations of the controls and the unknown samples the following calculations were performed:

- 1) The average optical density (OD) of the duplicate wells was calculated
- 2) The average OD from the NSB wells was then subtracted from the average OD of the blank, standard, control and sample wells
- 3) The percent bound (B/Bo) for each standard, control and sample was calculated by dividing the average OD by the average OD for the zero (Bo)
- 4) The 4-parameter sigmoid minus curve fit was then used to calculate the concentrations

Due to the 'hands off' nature of the study, and the fact that the data recording and sampling was performed by the subjects themselves rather than a Doctor or Sports Scientist, it was imperative that subjects were appropriately educated regarding the correct method of hormone collection. This was to make certain that when the sampling was performed, it was done so correctly and accurately in order to provide reliable and valid results. To ensure that subjects were familiar with the method of sampling a comprehensive information document was handed out for subjects to read (Appendix C). Further, prior to data collection, this information was explained to each of the subjects in an organised meet, either in Johannesburg, Port Elizabeth and Grahamstown.

## **EXPERIMENTAL PROCEDURE**

Once a broad experimental design for the study had been setup, a meeting with the race director of the Ironman South Africa was held. The meeting covered the nature of the study, avoiding conflict with the Ironman<sup>®</sup> and Triangle Sports<sup>®</sup> brands, as well as finding a suitable venue for subject recruitment. The race director approved the research and allowed the use of the 2008 pre-race registration and exhibition as a subject recruitment and informal information briefing venue.

## SUBJECT RECRUITMENT

During the 2008 exhibition, both male and female athletes, were approached and informed about the possibility of involvement in an Ironman-related research project. Those interested were given a flyer (Appendix A) and a brief explanation of the intentions of the study as well as the expectations of the subjects themselves. Contact details of the interested athletes were recorded. Two weeks after the Ironman 2008, an informative Electronic-mail (E-mail) was sent to all those athletes who showed interest during the exhibition. This included a letter of information and a more detailed explanation of the project requirements and procedures (Appendix B and C). Athletes who maintained interest were asked to reply to the E-mail stating their intention to be a part of the study. From this recruitment process an ultraendurance triathlete sample (n=136; 103 male and 33 female), of ranging abilities, was selected for the study. All of the subjects taking part in the research project from this point had to be intending to compete in the 2009 Ironman, South Africa. A number of athletes were unsure of participation in 2009, therefore signed up to take part in the research, but did not follow through with formal preparation and competition. Previous participation in full Ironman events was not a limiting factor for signing up for the study, therefore a number of the final subjects (n=5) were novice athletes training for their first Ironman event.

From the 136 athletes who signed up in the initial registration sample, 36 athletes showed continued interest by replying to the E-mail containing the letter of information and the design of the study. The majority of the athletes who replied to the E-mail were based in Gauteng and the Eastern Cape provinces of South Africa. Therefore, for logistical reasons, hormone sample collection sites were set up in Johannesburg (Gauteng), Port Elizabeth and Grahamstown (both Eastern Cape). Two triathlon institutes were approached for their assistance for the duration of the project. Troisport<sup>®</sup> of Johannesburg and Triangle Sports<sup>®</sup> of Port Elizabeth agreed to aid in the collection, as a drop-off point, of the hormonal samples on a monthly basis.

The control group was selected in February as a comparative group to the athlete sample. The members of this group were selected as a sedentary control defined by the ACSM's guidelines. The sample was made up from a Grahamstown population where the primary research was based.

## SUBJECT CHARACTERISTICS

**TableXIII: Subject characteristics for the experimental and control groups**

	Athlete			Control		
	X	SD	CV (%)	X	SD	CV (%)
<b>Age (yr)</b>	34.7	10.8	31.1	37.8	9.7	25.7
<b>Stature (mm)</b>	1796*	62	3.5	1738	73	4.2
<b>Mass (kg) April</b>	79.8*#	9.6	12.1	90.5	17.8	19.7
<b>Mass (kg) November</b>	82	10	12.2			
<b>BMI (November)</b>	25.6	2.1	8.2	30.2	5.2	17.2
<b>BMI (April)</b>	24.9	2.8	11.2			

(\* Denotes significant differences to control group)

(# Denotes significant differences to mass in November)

(BMI = Body Mass Index)

(SD = Standard Deviation; CV = Coefficient of Variation)

The control group used for the comparison with the athlete sample was sex and age matched (10 male and 5 female). Therefore, the mean age of the control group ( $37.8 \pm 9.7$ )yr was not significantly different ( $p < 0.05$ ) to the athlete group ( $34.7 \pm 10.8$ )yr. The mean mass of the control group ( $90.5 \pm 17.8$ )kg was not significantly different ( $p < 0.05$ ) to the mean starting mass of the athlete group ( $82 \pm 10$ )kg, however, it was significantly different ( $p < 0.05$ ) to the mean mass of the athletes immediately prior to racing ( $79.8 \pm 9.6$ )kg. The athletes ( $1796 \pm 62$ )mm were significantly taller ( $p < 0.05$ ) than the controls ( $1738 \pm 73$ )mm. The control

sample had a body mass index (BMI) of  $30.2\text{kg}\cdot\text{m}^{-2}$  indicating that they were defined as obese and fell into a high health risk category. The BMI readings for the athlete sample decreased from  $25.6\pm 2.1\text{kg}\cdot\text{m}^{-2}$  to  $24.9\pm 2.8\text{kg}\cdot\text{m}^{-2}$  over the training period. The control sample also possessed a higher variation in mass and stature amongst the group. This was illustrated by higher standard deviations (Table XIII).

#### INFORMATIVE BRIEFINGS

Meetings were held in Johannesburg, Port Elizabeth and Grahamstown. Athletes were encouraged to attend these meetings which would brief them on the experimental design and data collection procedure for the duration of the project. TUT assisted in the storage and collection of the Gauteng-based athlete's hormonal samples and were therefore present at the meet in Johannesburg. These briefings were in aid of ensuring that subjects received written and verbal instruction on the requirements of the project. While these instructions focused on the familiarisation with the methodological concerns of the fulfilment of the day-to-day training log, POMS score sheet, and 8km submaximal time trial, there was a great deal of emphasis placed on the procedure involved with the hormone sampling technique. Once familiarised with the protocol of the project, subjects were handed a hormone collection kit. This included ten, 2ml Corning® polypropylene cryogenic vials marked with the relevant sampling date as well as the subject's individual reference number. The instruction of the correct sampling and the pre-storage routine was given to the subjects verbally and in writing to ensure proper technique was followed. Once athletes had been extensively informed of the requirements, design and procedures of the project, relevant questions were answered. Following this, subjects gave their voluntary, written and witnessed informed consent (Appendix E) to a research design and protocol which had been approved by the Rhodes University Ethics Committee. Subjects were given the option of completing a hard copy or electronic data file for the study (Appendix C). Those athletes unable to meet at the organised times were met personally at another arranged time and venue.

Correspondence concerning the project from this point onwards was kept on an electronic and telephonic basis. Constant contact was maintained with the subjects for the duration of the study. These included regular reminders and checkups (E-mail and telephonic). These reminders were performed to ensure that subjects were keeping up to date with the completion of the day to day questionnaire, the weekly profile of mood states (POMS) analysis, the monthly 8km submaximal running time trial and the next hormonal sampling date. Those athletes completing the electronic data file submitted it on a monthly basis, where as those who opted to use a hard copy data file handed it in at the registration for the Ironman 2009. Separate data sheets were handed out for the remaining 2 weeks of the data collection; these were collected as soon as possible after the final sample date.

#### DATA COLLECTION

Data collection for the duration of the project was performed by the subjects themselves, athletes completed the day-to-day training log and POMS score sheet on a daily and weekly basis respectively. The control group completed the daily log (excluding training details) and POMS questionnaire on a weekly basis for the duration of their data collection period. While the time trial was performed on a monthly basis, the hormones were collected on the allocated days provided (Table XIV). Although constant contact was maintained and reminders were sent to the subjects, the physical monitoring of the subject's adherence to the procedure lay in the hands of the subjects themselves. Once monthly, when allocated electronic data files were completed and submitted, these were readily analysed to ensure their accurate completion. Any outlying data were brought up with the subject/s concerned. This, constant 'check up' aided in keeping track of subject participation and accurate completion of all relevant tests.

**Table XIV: Data collection requirements and frequency of these requirements**

FREQUENCY OF COLLECTION				
REQUIREMENTS	Daily	Weekly	Monthly	Allocated samples
	Day-to-day Training Log	POMS Analysis	8km Sub-maximal Running Time-Trial	Salivary Hormone Sampling

At the registration and exhibition of the Ironman 2009, a meet was held and the 7<sup>th</sup> hormone sample (performed on that morning) was handed in to an informed team of Rhodes University Human Kinetics and Ergonomics (HKE) postgraduate students. These students acted as a testing team aiding in sample collection over the days surrounding and including the event itself. The subjects were further thanked for their co-operation throughout the study so far, and the final race phase sample collection was explained. The testing team was situated at the finish of the race, where subjects were to give drool samples immediately after finishing the race (Medical tent) and another sample one and a half hours post race which was relative to each subject finish time. The second samples required 90min post race, could either be performed at the finish line medical tent or at a private venue then delivered later that day or at a specified meeting point the next day. Prior to these samples, subjects had to rinse out their mouth with water, wait one minute and produce a drool sample. Due to the nature of the race, oral intake prior to these samples was not restricted and therefore would have influenced the validity and reliability of these results.

Race numbers and estimated finish time were recorded prior to the start of the race and athlete progress was followed throughout the race to gain an approximate finish time. This allowed the research assistants in the medical tent at the finish to prepare for the athletes' arrival making it easier to identify

subjects. Additionally, subjects were requested to inform the officials in the medical tent that they were a part of the study, to ensure that a post-race sample was taken.

## **STATISTICAL ANALYSIS**

Repeated measure analyses of variance (ANOVA) were used to determine statistically significant changes in the physiological, psychological, biochemical, immunological and performance related markers associated with training completed in the current study. Statistical measures were run for the experimental sample (n=18), over a six month data collection phase, and control sample (n=15) during the competition month. The comparison of the two sample groups (control and athlete) was made using a repeated measures analysis of variance. Data from the control sample was only collected for a one month period. Tukey post-hoc multiple comparison tests were performed where significant group effects were found. One month of control data was compared to all of the athlete samples months. One way ANOVAs were run on the control sample data, these confirmed similar results for the three weeks of data collection and therefore illustrated that there would be limited changes in the responses of this sample. Therefore it was logistically, financially and statistically more feasible to limit this data collection to only one month, financial limitations prevented this postulation being justified using a pilot study. Independent T-Tests were run to determine significance between the experimental groups and the predicted versus actual race finish times. Correlations were calculated using  $y = mx+c$ , finding the regression with  $R^2$ . All of the statistical tests run were performed using Statistica version 8.

## CHAPTER IV

### RESULTS

#### INTRODUCTION

The purpose of this investigation was to holistically monitor a group of athletes (n=18) training for an ultraendurance triathlon event. As it was expected that training loads would likely be high, a second aim was to establish how such intensive training affects the body's response measures. Measurements obtained over a six month period were divided into physiological, psychological, immunological, biochemical and performance responses. The effects of ultraendurance training of this nature were compared to 15 sedentary age and sex matched controls.

#### TIME COURSE OF RESPONSE CHANGES

##### ANTHROPOMETRIC MEASURES

**Table XV: Changes in body mass over the period of training.**

**Data are expressed as means for each month**

Month	Body Mass (kg)		Standard Deviation (kg)		Coefficient of Variation (%)	
November	82.0		10.0		12.2	
December	82.1		9.8		12.0	
January	82.5		10.8		13.1	
February	80.5		9.9		12.3	
March	79.9 *		9.6		12.0	
April Competition Month	79.8 * #	90.5 #	9.6	17.8	12.1	19.7

(\* Denotes significant differences to athlete sample in November to February)

(# Denotes significant differences to athlete sample; November to April)

(Highlighted cells indicate control sample results)

Body mass remained stable in the first four months of measurement and then decreased significantly ( $p < 0.05$ ) the month prior to competition and the month of competition (Table XV). Mean starting body mass was  $82.0(\pm 10.0)$  kg while immediately prior to the competition it was  $79.8(\pm 9.7)$  kg. The control sample, which was analysed alongside the athlete sample during the competition month, showed a significantly greater ( $p < 0.05$ ) mean body mass ( $90.5(\pm 17.8)$  kg). However this mass was no different to the athletes for November and January

## TRAINING MEASURES

**Table XVI: Mean ( $\pm$ SD) total, swimming, cycling and running training loads**

Month	Total Training	Swim Training	Bike Training	Run Training
November	1947.2 (901.7) 46.3%	246.7 (179.9) 72.9%	1039.9 (603.3) 58.1%	660.5 (411.1) 62.3%
December	2206.3 (767.1) 34.8%	266.0 (189.4) 70.9%	1132.5 (415.1) 36.7%	806.8 (450.7) 55.9%
January	2460.6 (1088.9) 44.3%	324.4 (269.0) 82.9%	1302.9 (642.8) 49.3%	833.4 (396.1) 47.5%
February	3197.3 (1454.1) 45.5%	303.6 (176.1) 58.1%	1856.9(1070.9) <sup>#</sup> 57.7%	1041.9 (500.9) 48.1%
March	2946.7 (1425.4) 48.4%	500.6 (558.2) 111.5%	1543.9 (949.6) 61.5%	987.1 (410.9) 41.6%
April Competition Month	427.4 (485.4) * 113.6%	28.5 (19.6) * 68.9%	141.5 (124.3) * 87.8%	257.3 (405.3) * 157.5%

(\* Denotes significant differences within discipline to November, December, January, February and March)

(<sup>#</sup> Denotes significant differences within discipline to December)

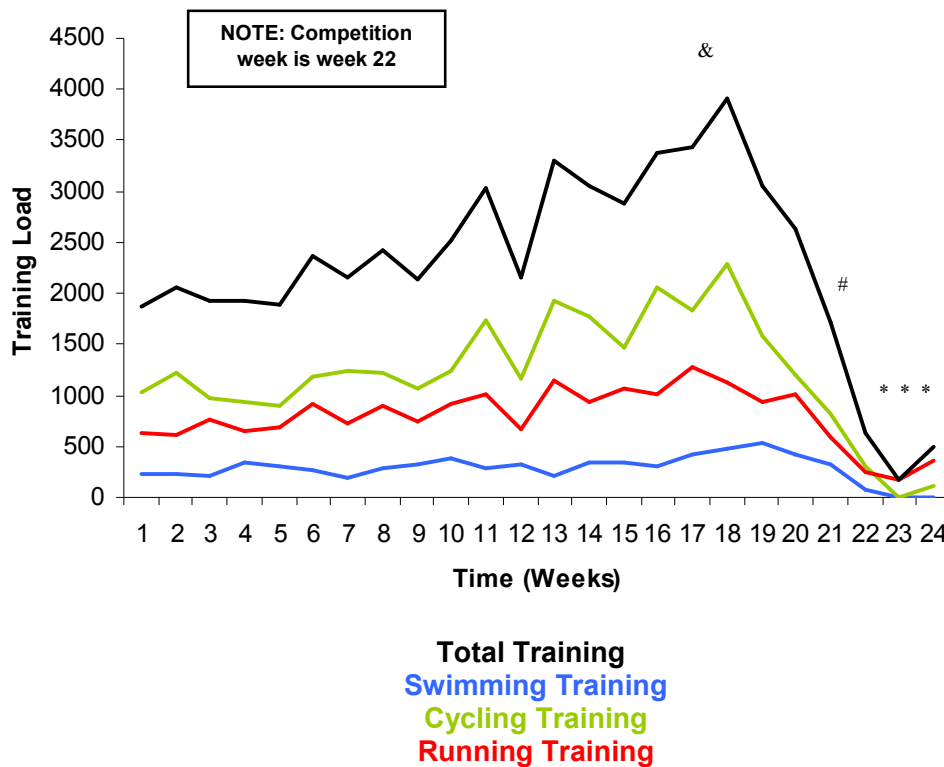
(% = coefficient of variation)

### Mean total, swimming, cycling and running training loads

Mean total training load did not change with the exception of the typical taper period at the end of March and April. While training load was not significantly lower in March (taper begun during March) there were significant ( $p < 0.05$ )

decreases in total training load to 427.4( $\pm$ 485.4) in April from the highest load of 3497.27( $\pm$ 1454.1) which was during the month of February. As the athletes represented a broad range of performance abilities, the high variability in total training load was to be expected and may explain the non-significant increases during training load in the months of training particularly in February. November, December, January and February all included four full weeks of data collection, while March and April contained five and three weeks respectively. In the majority of athlete's cases, although March contains a significantly higher total training load than April (possibly due to it containing more weeks of training), this month is considered a taper month.

Average monthly swim training load ranged from 246.7 $\pm$ 179.9 to 500.6 $\pm$ 558.2. The peak training load (500.6 $\pm$ 558.2) was evident in the month prior to competition. There was a significant ( $p < 0.05$ ) decrease in swim training load in the competition month with no athletes undertaking any swim training for the last two weeks of April, these two weeks were however post event and this was to be expected. Cycling training increased significantly ( $p < 0.05$ ) in February (1856.9 $\pm$ 1070.9) with more cycling training performed than there was in December (1132.5 $\pm$ 415.1). There was significantly ( $p < 0.01$ ) less training performed during the competition month (141.5 $\pm$ 124.3) than all of the other months. This peak in training load during the month of February has been extensively advocated by a number of coaches and Ironman professionals. Training load for running did not significantly increase ( $p < 0.05$ ) during the data collection period, except in the last month where there was a significant decrease ( $p < 0.01$ ). Training peaked in February at 1041.9( $\pm$ 500.9) decreased to the lowest load of 257.3( $\pm$ 405.3) in April. The high variability in training performed in all three disciplines is shown by the high standard deviations and coefficient of variations.



**Figure 13: Mean swimming, cycling, running and total training load**

(\* denotes significant differences to weeks 22, 23 and 24 (total training load))  
 (# denotes significant differences to weeks 13, 17 and 18 (total training load))  
 (& denotes significant differences to week 5 (total training load))

Training load during the last three weeks (competition period; weeks 22, 23 and 24) was significantly lower ( $p < 0.05$ ) than all weeks in the previous five months. Further, week 21's training load was also significantly lower than weeks 13, 17 and 18. The peak in training at week 18 (3623.5) was significantly higher ( $p < 0.05$ ) than training performed in the early stages of training (week five). The mean total training load was comprised of 11.8% swim training, 49.5% bike training and 38.8% run training. Significant ( $p < 0.05$ ) increases in training were evident between week 19 compared to weeks three, seven and 13. There were significant decreases ( $p < 0.05$ ) found between the final preparation week (week 22) and weeks ten, 17, 18, 19 and 20. Bike training was increased significantly ( $p < 0.05$ ) in the 13<sup>th</sup> and 18<sup>th</sup> weeks in comparison to the 5<sup>th</sup> week of training and reduced significantly ( $p < 0.05$ ) in weeks 21, 22, 23 and 24. Like the cycling

training, running training was cyclical in nature, with alternating ‘on-off’ weeks (Figure 13). However, it did decrease significantly ( $p<0.05$ ) in the week leading up to (week 22) and the two weeks following (week 23 and 24) the event.

## PHYSIOLOGICAL MEASURES

**Table XVII: Mean ( $\pm$ SD) waking heart rate ( $\text{bt}\cdot\text{min}^{-1}$ ), postural dizziness (1-4), sleep rating (1-4) and sleep duration (hr)**

Month	Waking Heart Rate ( $\text{bt}\cdot\text{min}^{-1}$ )		Postural Dizziness Rating (1-4)		Sleep Rating (1-4)		Hours of Sleep (hrs)	
November	53 (5.0)	9.5%	1.3 (0.5)	38.0%	2.0 (0.4)	22.0%	7.0 (0.5)	7.5%
December	52 (5.4)	10.4%	1.3 (0.5)	22.7%	1.9 (0.4)	22.7%	7.1 (0.4)	5.6%
January	52 (5.1)	9.8%	1.4 (0.4)	29.54%	2.0 (0.4)	20.4%	7.4 (0.5)	7.4%
February	52 (5.9)	11.3%	1.4 (0.4)	31.8%	1.9 (0.5)	24.4%	7.2 (0.6)	8.7%
March	52 (5.3)	10.1%	1.4 (0.4)	27.8%	1.9 (0.4)	23.2%	7.3 (0.6)	8.2%
April Competition Month	54 (4.9)	62 <sup>#</sup> (6.7) 10.7%	1.4 (0.4)	1 <sup>#</sup> (0) 0%	1.8 (0.4)	1.6 (0.4) 23.2%	7.6* (0.7) 8.5%	7.9 <sup>§</sup> (0.8) 10.3%

(\* denotes significant differences to November and December)

(<sup>#</sup> denotes significant differences to the experimental group scores November to April)

(<sup>§</sup> denotes significant differences to the experimental group scores November to March)

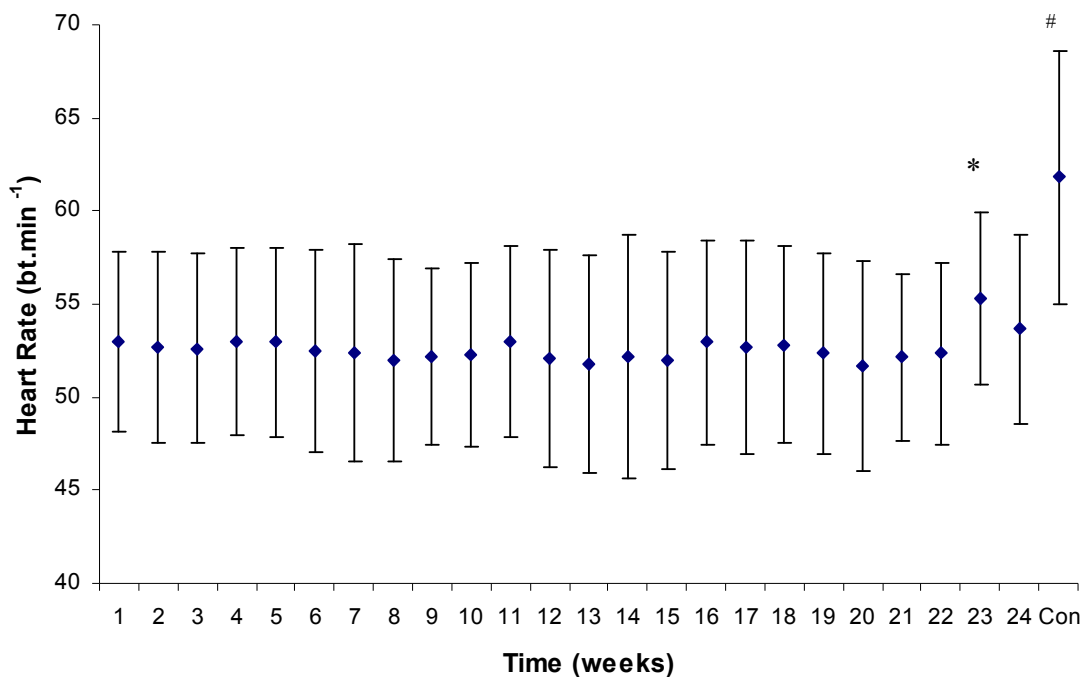
(% = coefficient of variation)

(Highlighted cells indicate the control sample)

### Waking heart rate

Mean waking heart rates ranged from  $52\pm 5.4\text{bt}\cdot\text{min}^{-1}$  to  $54\pm 4.9\text{bt}\cdot\text{min}^{-1}$  during the six month longitudinal analyses. There were no significant changes in monthly waking heart rate for the experimental sample, however the control sample’s mean monthly scores were significantly ( $p<0.05$ ) higher than all of the experimental group’s mean monthly waking heart rate scores (Table XVII).

Waking heart rate following the Ironman and more specifically in week 23, was significantly ( $p < 0.05$ ) higher than weeks 13, 14, 15, 20 and 21. Heart rate increased above  $54 \text{ bt. min}^{-1}$  for the first time during this week to a mean of  $55 \text{ bt. min}^{-1}$  (Figure 14). The control sample's mean week one ( $61.8 \pm 6.8 \text{ bt. min}^{-1}$ ) and week two ( $62.6 \pm 6.5 \text{ bt. min}^{-1}$ ) waking heart rates were significantly ( $p < 0.05$ ) higher than all 24 experimental sample weeks



**Figure 14: Mean waking heart rate ( $\text{bt. min}^{-1}$ )**

(\* Denotes significant difference in week 23 compared to weeks 13, 14, 15, 20 and 21)

(# Denotes significant difference to experimental group weeks 1 to 24)

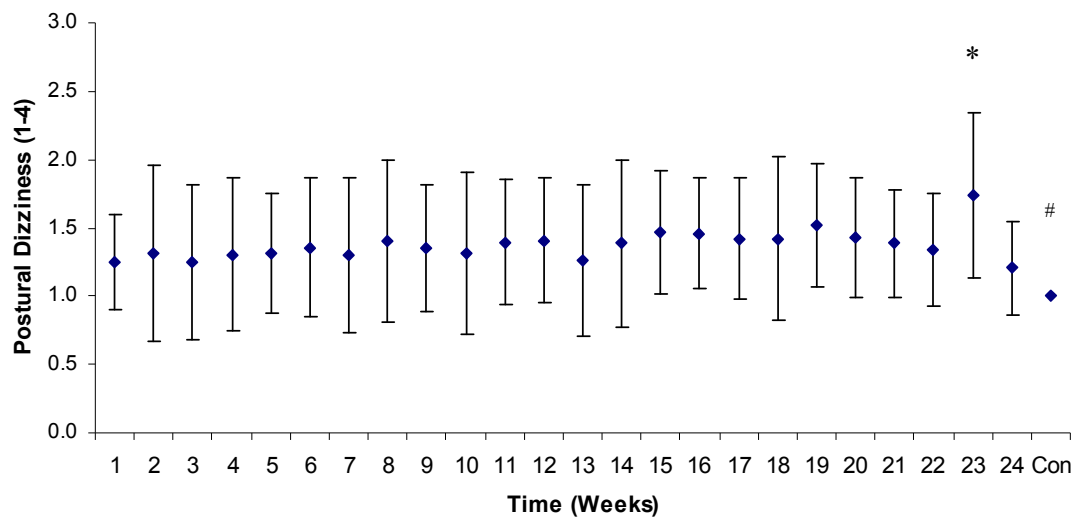
(Error bars indicate standard deviations)

(Con indicates control sample)

### Postural dizziness

Mean monthly postural dizziness (Table XVII) shows that although there was an increased prevalence and severity of postural dizziness from month one to month five, these changes were not significant. The scores, out of 4 (1=No dizziness; 2=Slight dizziness, disappears with 2 seconds; 3=Marked dizziness, time taken

to orient one's self; 4=Marked dizziness, requiring sitting or lying down again), increased from 1.28 to 1.44, before dropping down to 1.43 in the competition month, the control group's monthly scores were significantly ( $p<0.05$ ) lower than every month of the athlete sample (Table XVII). Like the waking heart rate response, there were significant ( $p<0.05$ ) increases in the rating of postural dizziness during week 23 (post Ironman event week) compared to weeks 7, 13 and 24 (Figure 15). The control group's mean weekly postural dizziness score was significantly ( $p<0.05$ ) lower than each of the experimental sample's mean weekly scores (week 1-24).



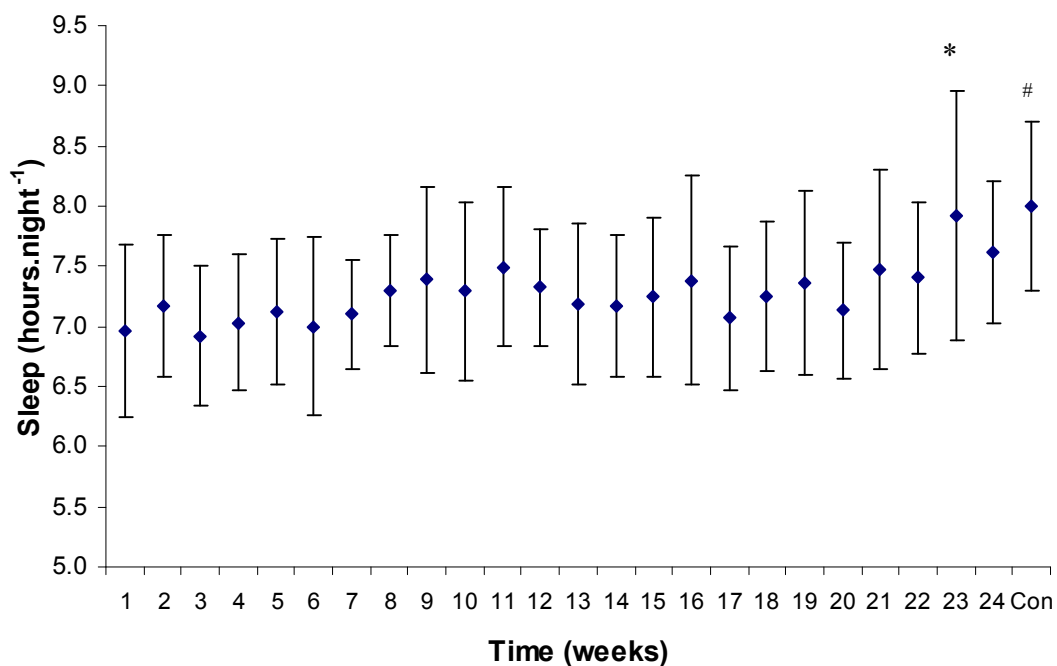
**Figure 15: Mean postural dizziness rating (1-4)**

(\* Denotes significant difference in week 23 compared to weeks 7, 13 and 24)  
 (# Denotes significant difference to experimental group weeks 1 to 24)  
 (Error bars indicate standard deviations)  
 (Con indicates control sample)

### Sleep ratings (quantity and quality of sleep)

The mean daily hours of sleep, averaged over each month, did not differ significantly. However there was a significant ( $p<0.05$ ) increase in the time slept in the month of competition (April) compared to November and December (Table XVII). While the control group's mean daily hours of sleep, averaged over each month, was significantly ( $p<0.05$ ) more than December, January, February and

March for the experimental group, it was, however, not significantly greater than April. Further, there was a significant ( $p < 0.05$ ) increase in hours slept during the post-competition week (week 23) compared to weeks 1, 3, 5, 6, 17 and 20 (Figure 16). Most athletes had interrupted sleep, but not sufficient to perceive a need for more sleep. The mean control group sleep rating was not significantly different to that of the experimental group.



**Figure 16: Mean hours of daily sleep (hrs)**

(\* Denotes significant difference in week 23 compared to weeks 1, 3, 5, 6, 17 and 20)

(# Denotes significant difference to experimental group weeks 1, 3, 6 and 17)

(Error bars indicate standard deviations)

(Con indicates control sample)

## PSYCHOLOGICAL RESPONSES

### General Mood State

The general mood ratings (Table XVII) ranged from 1.35 to 1.62 (1-4 scale; 1=Good mood; 2=Feeling sad or down only at times; 3=Feeling sad or down

most of the time; 4=Feeling sad or down all of the time). This suggests that the athletes perceived themselves to be in good moods and only felt sad, or down, at times. The mood rating in February ( $1.6 \pm 0.4$ ) was significantly ( $p < 0.05$ ) higher than that of December ( $1.4 \pm 0.2$ ) and January ( $1.4 \pm 0.3$ ). Mood ratings in March ( $1.6 \pm 0.3$ ) were significantly ( $p < 0.05$ ) greater than those in December ( $1.4 \pm 0.2$ ). The control group's general mood rating was not significantly ( $p < 0.05$ ) different to the athlete sample in any month, with the exception of February ( $1.6 \pm 0.4$ ).

**Table XVIII: Mean ( $\pm$ SD) general mood rating (1 - 4)**

Month	General Mood		Standard Deviation		Coefficient of Variation (%)	
November	1.4		0.3		17.9	
December	1.4		0.3		21.6	
January	1.4		0.2		15.3	
February	1.6 *		0.4		24.9	
March	1.6 <sup>&amp;</sup>		0.3		19.3	
April Competition Month	1.5	1.3 <sup>#</sup>	0.3	0.2	23.2	17.6

(<sup>#</sup> Denotes significant difference between the control measures and athlete measures obtained in February)

(\* Denotes significant difference to the athlete measures in December and January)

(& Denotes significant difference to the athlete measures in December)

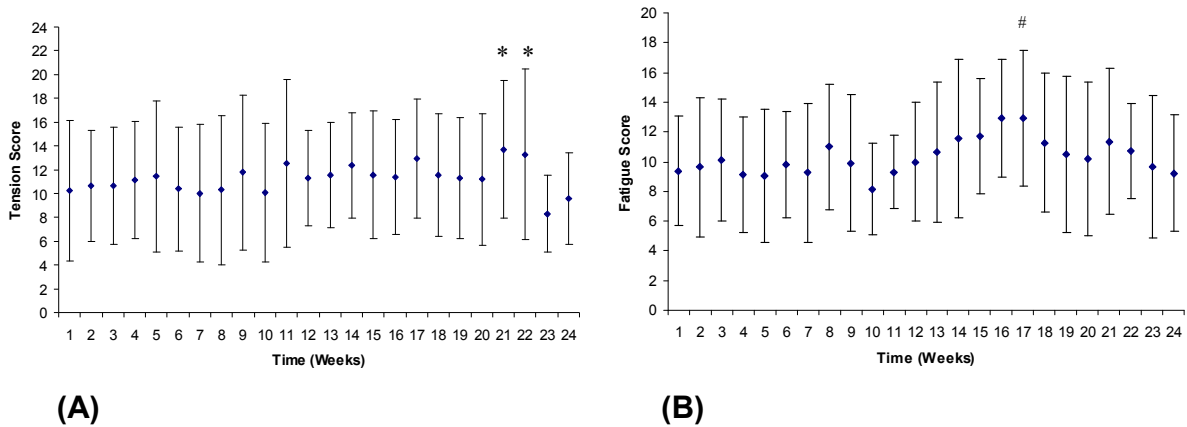
(Highlighted cells indicate the control sample)

(% = Coefficient of variation)

### **Profile of Mood States (POMS)**

There were no significant changes in all of the monthly POMS scores during training and competition. There was however a number of significant differences found between weekly scores. There was a significant ( $p < 0.05$ ) decrease in tension scores from the two weeks leading up to the event, week 21 ( $13.7 \pm 5.8$ ) and week 22 ( $13.3 \pm 7.2$ ), to the two weeks following the event, week 23 ( $8.3 \pm 3.2$ )

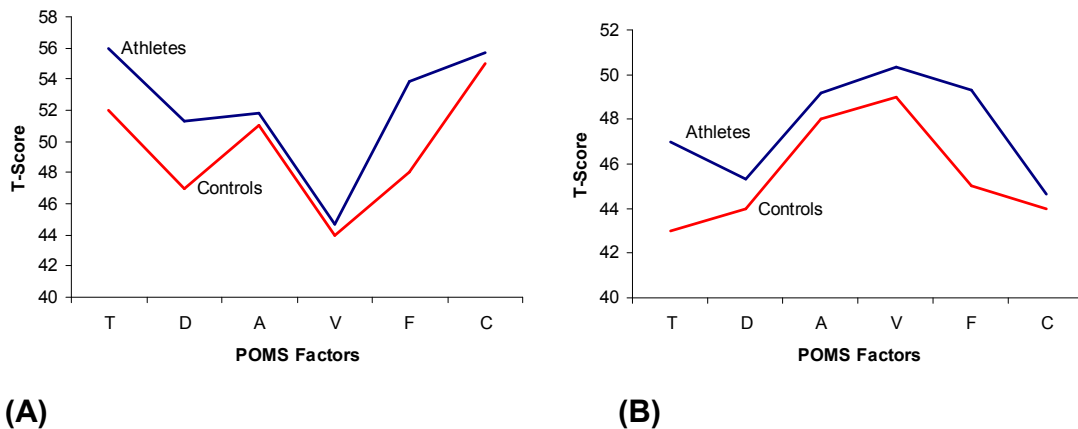
and 24 ( $9.6 \pm 3.8$ ) (Figure 17A). Likewise the fatigue score in week 17 was significantly higher ( $p < 0.05$ ) than that of week five and ten (Figure 17B).



**Figure 17: Mean POMS tension (A) and fatigue (B) scores**

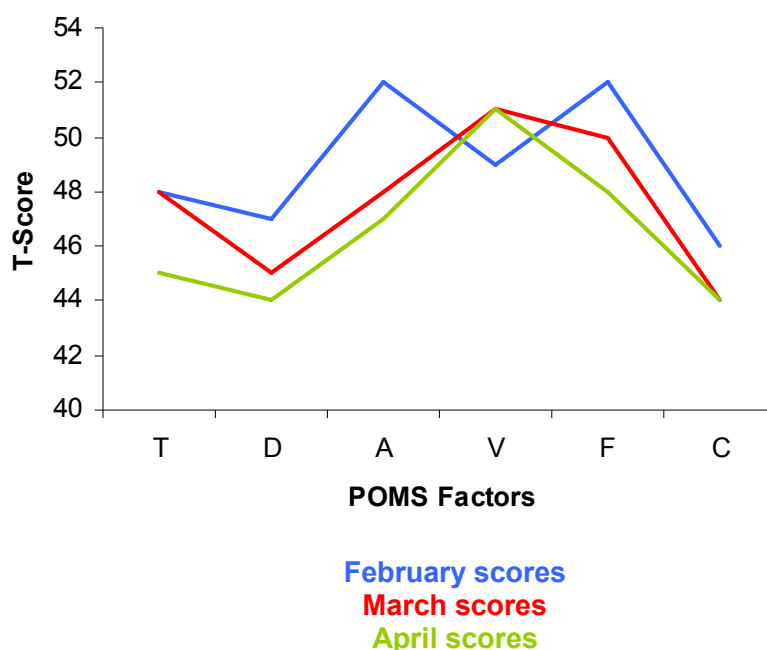
(\* Denotes significant difference to weeks 23 and 24)  
 (# Denotes significant difference to weeks 5 and 10)

The Adult profile (A) and the college student profile (B) illustrate no significant difference ( $p < 0.05$ ) between the control and athlete samples. From Figure 18 it is evident that depending on the profile used, the result differs.



**Figure 18: Mean monthly Profile of Mood State (POMS) scores using (A) the adult norms score sheet and (B) the college norms score sheet (T=tension; D=depression; A=anger; V=vigor; F=fatigue; C=confusion)**

The majority of research performed on training techniques using the POMS questionnaire has suggested the use of the college student norms score sheet (Figure 18B), in an attempt to observe changes to the 'Iceberg Profile' (vigor score above 50 as well as above the other mood states), an indicator of an athlete's mood state profile. This 'Iceberg Profile' is said to invert in cases where athletes become overtrained, making the identification of athletes approaching an overtrained state easier. The sample of athletes, however, more suitably falls into the adult population score sheet (Figure 18A). The adult score sheet shows a very different profile to that of the college student norms profile. The T-Scores for the adult norms and the college student norms were significantly ( $p < 0.05$ ) different.



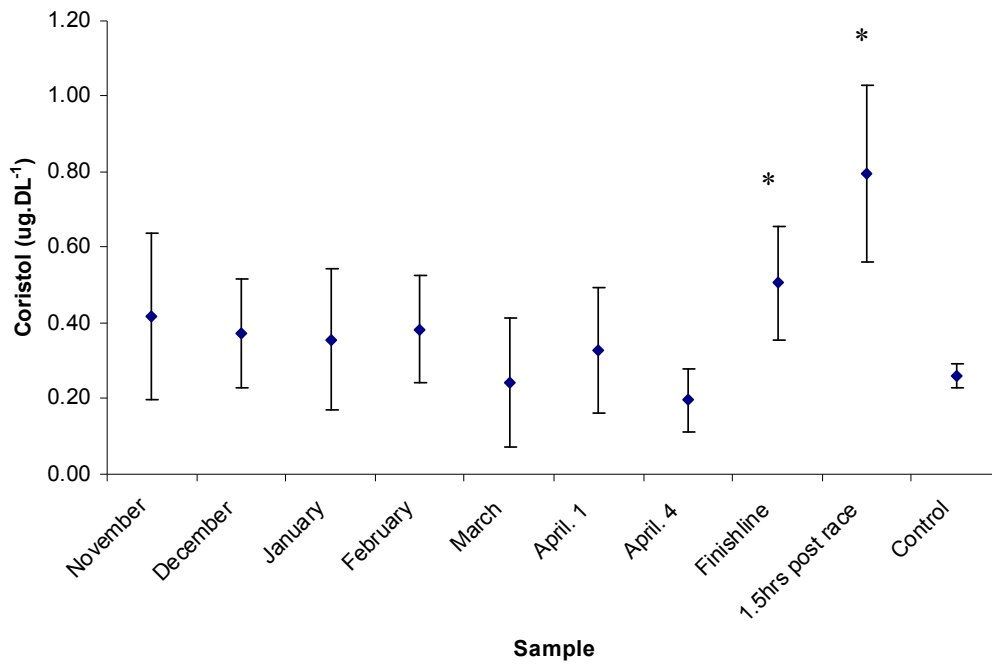
**Figure 19: Profile of Mood State (POMS) for February, March and April scores, for the athlete population using the college norms score sheet (T=tension; D=depression; A=anger; V=vigor; F=fatigue; C=confusion)**

The POMS profiles for the last three months of data collection show that there is an inversed iceberg profile during the peak training month of February, this includes a decreased vigor score as well as high anger and fatigue scores. The March profile shows development from the 'inversed iceberg' profile toward the 'Iceberg' profile. This profile develops further in April, suggesting that the athletes were approaching a psychological state indicative of high performance (Figure 19).

## BIOCHEMICAL RESPONSES

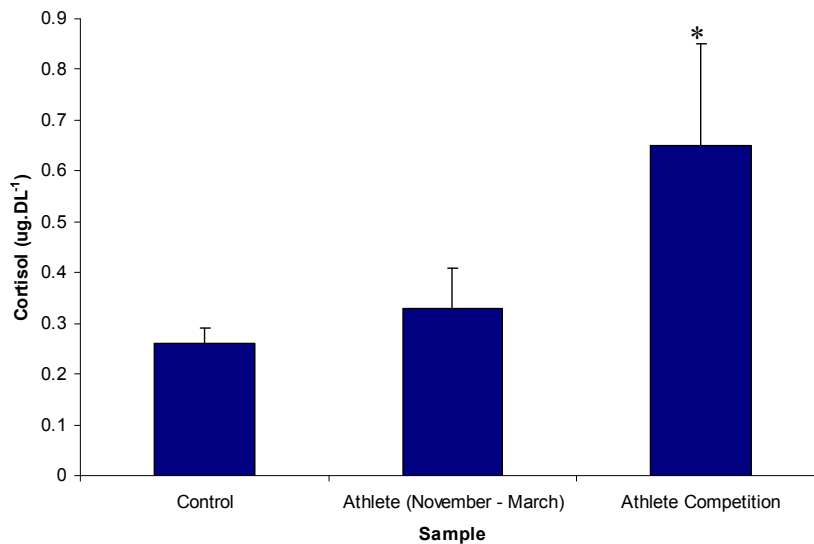
### **Cortisol**

Cortisol did not alter during the course of the training period. There was however, a significant ( $p < 0.05$ ) increase in cortisol immediately post race ( $0.507 \mu\text{g} \cdot \text{DL}^{-1}$ ) and 1.5 hours later ( $0.796 \mu\text{g} \cdot \text{DL}^{-1}$ ) (Figure 20). There were no significant differences ( $p > 0.05$ ) between the control group and the athlete samples taken from November to the one taken on the day prior to the race (April 4). The post competition samples  $*(0.65 \pm 0.2) \mu\text{g} \cdot \text{DL}^{-1}$  were significantly ( $p < 0.05$ ) higher than the control samples concentrations. The low standard deviations in Figure 20 and 21 illustrate that there is less variability in cortisol concentrations amongst the control sample. It can be noted that although not significant, there is an elevated training response associated with cortisol concentrations in the athletes. The cortisol levels were already mildly elevated in the athletes which was associated to most of these athlete's reported 'start' of started training.



**Figure 20: Mean (±SD) concentrations of cortisol (µg.DL<sup>-1</sup>) during training and post competition**

(\* Denotes significant difference to November, December, January, February, March, April 1 and April 4 and the control sample)



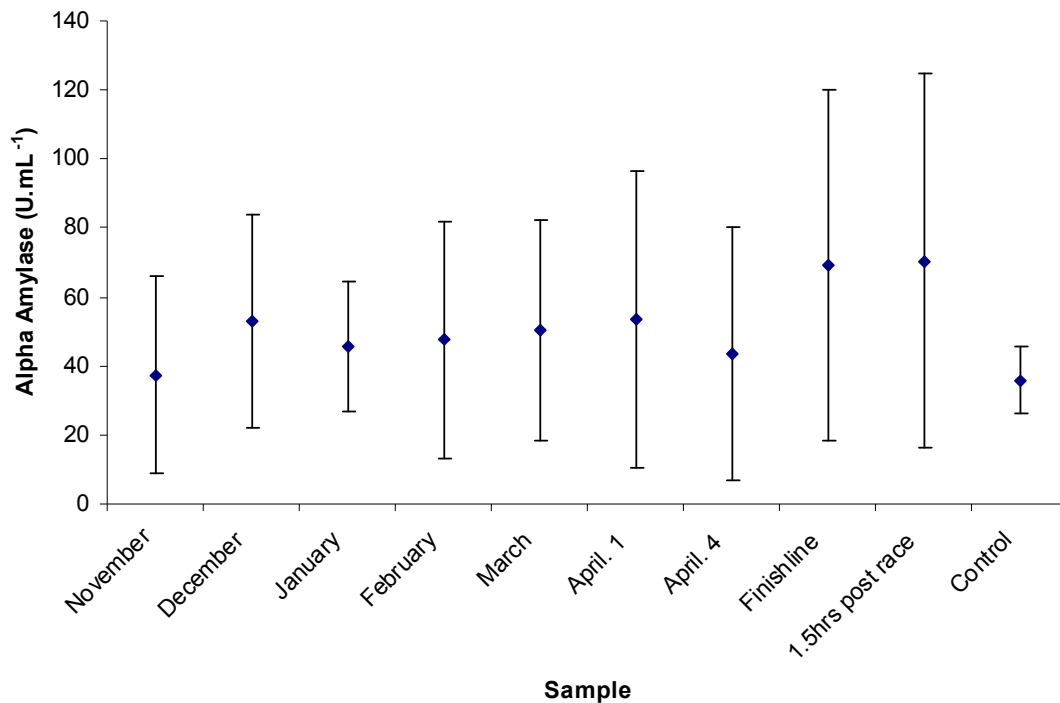
**Figure 21: Cortisol concentrations in the control and athlete sample**

(\* Denotes significant deviations to control and November - March athlete samples)

(Error bars indicate standard deviations)

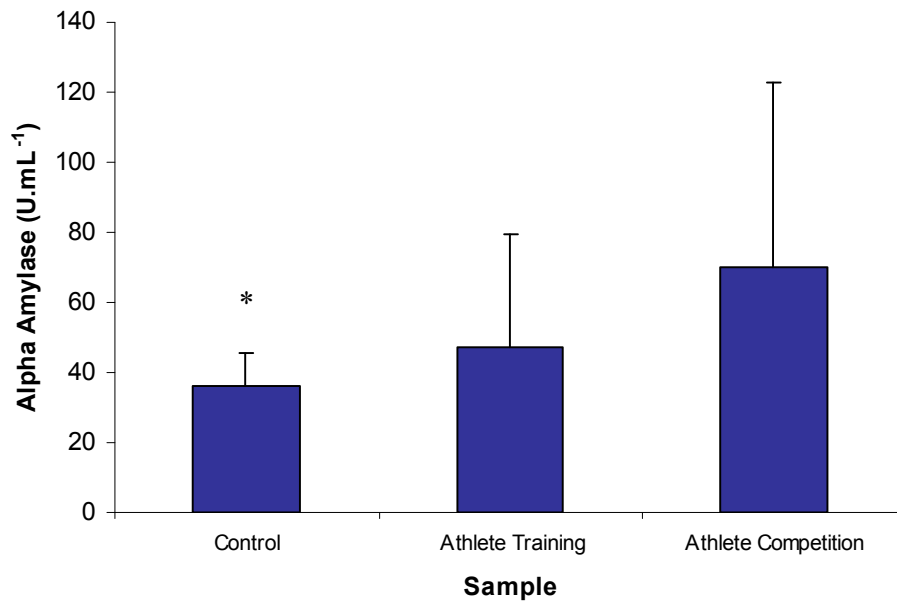
## Alpha-amylase

There were no significant ( $p>0.05$ ) changes in alpha amylase concentrations, this included differences between the experimental group and the control group measures. However, the trend was for alpha amylase to increase post race. It is possible that the huge inter-subject variability prevented a significant finding, although this is only speculation (Figure 22). The two samples after completion of the race show decidedly elevated alpha amylase concentrations ( $69.35\text{U}\cdot\text{mL}^{-1}$  for the sample taken at the finish line and  $70.48\text{U}\cdot\text{mL}^{-1}$  1.5hours after completion of the race).



**Figure 22: Mean ( $\pm$ SD) concentrations of Alpha Amylase ( $\text{U}\cdot\text{mL}^{-1}$ ) during training and post competition**

Mean alpha amylase concentration for the control sample ( $35.9\pm 9.5$ )  $\text{U}\cdot\text{mL}^{-1}$  was significantly less ( $p<0.05$ ) than the mean athlete competition phase sample ( $69.9\pm 52.6$ )  $\text{U}\cdot\text{mL}^{-1}$ . It was, however, not significantly different ( $p>0.05$ ) to the mean athlete training phase sample ( $47.3\pm 32$ )  $\text{U}\cdot\text{mL}^{-1}$  (Figure 22 and 23).



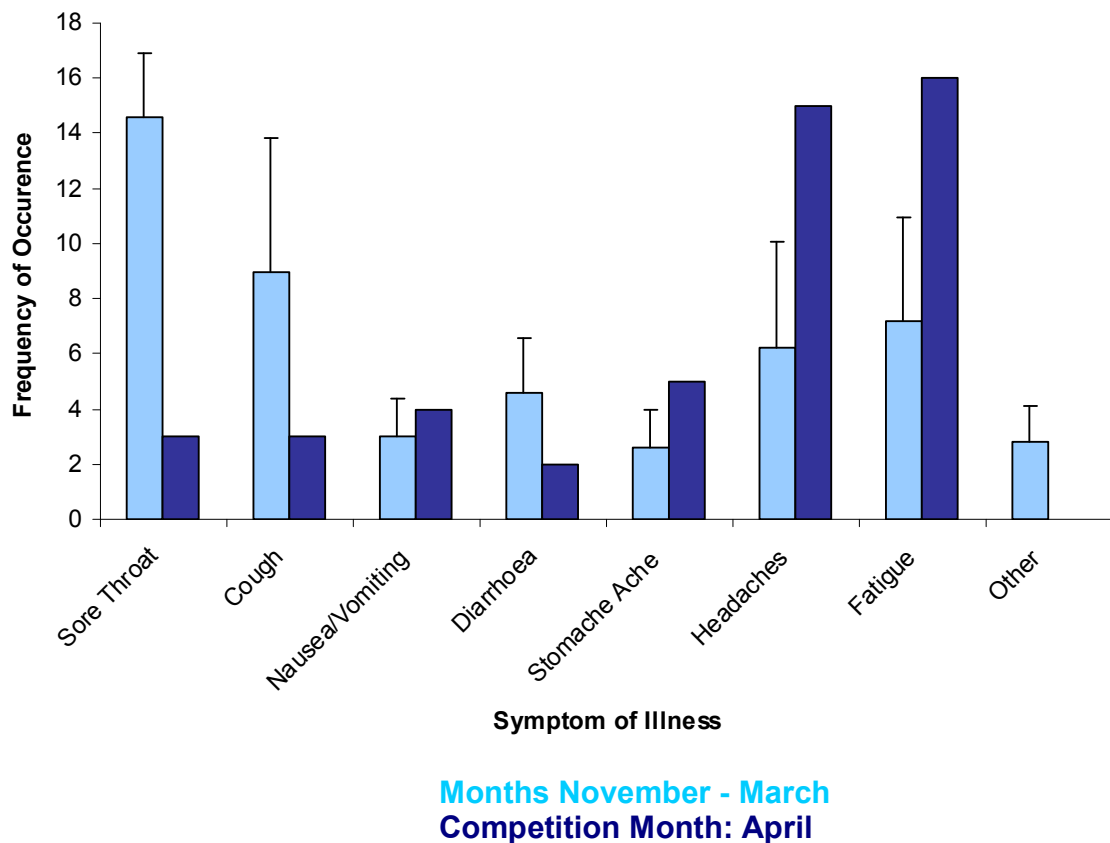
**Figure 23: Alpha amylase concentrations in the control and athlete samples**

(\* Denotes significant deviations to athlete competition samples)  
(Error bars indicate standard deviations)

## IMMUNOLOGICAL MEASURES

During the course of the investigation, there were two trends regarding the prevalence of illness; one during the training phase and one during the competition phase. The mean monthly frequency of illness was not significantly ( $p > 0.05$ ) different, with the same number, but not necessarily the same symptom, being reported. November to March illustrates that the most regularly occurring symptom was that of a sore throat, averaging  $14.6(\pm 2.3)$  times per month, with cough symptoms following in second at  $9(\pm 4.8)$  times per month (Figure 24). The prevalence of each of the other symptoms (Nausea/vomiting, Diarrhoea, Stomach aches, Headaches, Fatigue and Others) fell below an average of 7.2 occurrences per month. Symptoms reported in the week prior to the event included sore throats ( $n=4$ ) and stomach aches ( $n=2$ ). While during the week

post Ironman (week 23) there was a large increase in the occurrence of headaches, from 6.2( $\pm$ 3.83) to 15, and fatigue symptoms from 7.2( $\pm$ 3.77) to 16. Symptoms that were not included by the 1 – 7 symptoms of illness guideline were rated as an 8. These included symptoms such as sores in the athlete’s mouth and ear infections. During the data collection phase for the control group, there were only two reported symptoms of illness. These were one report of headache and another of coughing symptoms. Therefore these symptoms were not compared to the experimental group, but were simply used for discussion purposes.



**Figure 24: Monthly frequency of occurrence of symptoms of illness**  
 (Error bars indicate SD for the months, November - March)

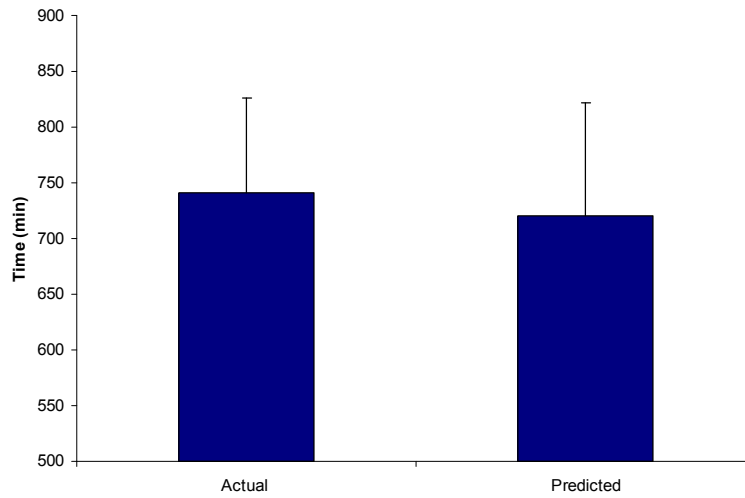
## PERFORMANCE MEASURES

There were positive improvements in performance with each subsequent time-trial (Table XIX). During November, the mean time-trial performance time was 42.1( $\pm$ 10.2) min which was reduced to 40.4( $\pm$ 9.1) min in the time-trial performed in the month prior to competition (March). As performance decrements have been considered as the 'gold standard' for the diagnosis of overtraining, it would appear that the current sample was not overtrained.

**Table XIX: Mean ( $\pm$ SD) performance times (min) for the monthly time-trial**

Month	Time-Trial (min)	Standard Deviation (min)	Coefficient of Variation (%)
November	42.06	10.2	24.3
December	41.63	7.8	18.8
January	41.61	8.5	20.4
February	40.79	7.1	17.5
March	40.43	9.1	22.6

Athletes were required to submit a predicted race performance time at the subject briefing the day prior to the event. The mean actual finish times (741.2 $\pm$ 85.2 min) and mean predicted finishing time (720.4 $\pm$ 101.4min) were compared and there was no significant ( $p < 0.05$ ) difference found between these two times (Figure 25). The trend shows that athletes marginally overestimated their performance (approximately 20min).

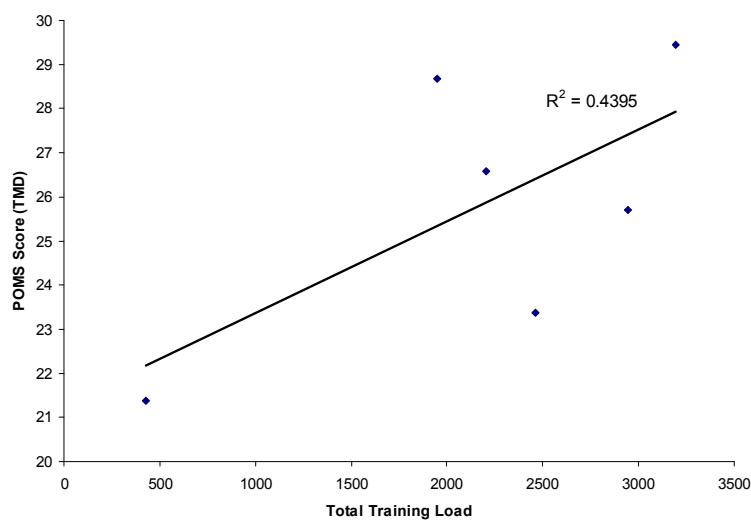


**Figure 25: Actual vs. predicted finishing times (min)**  
(Error bars indicate SD)

#### MULTIVARIATE ANALYSES

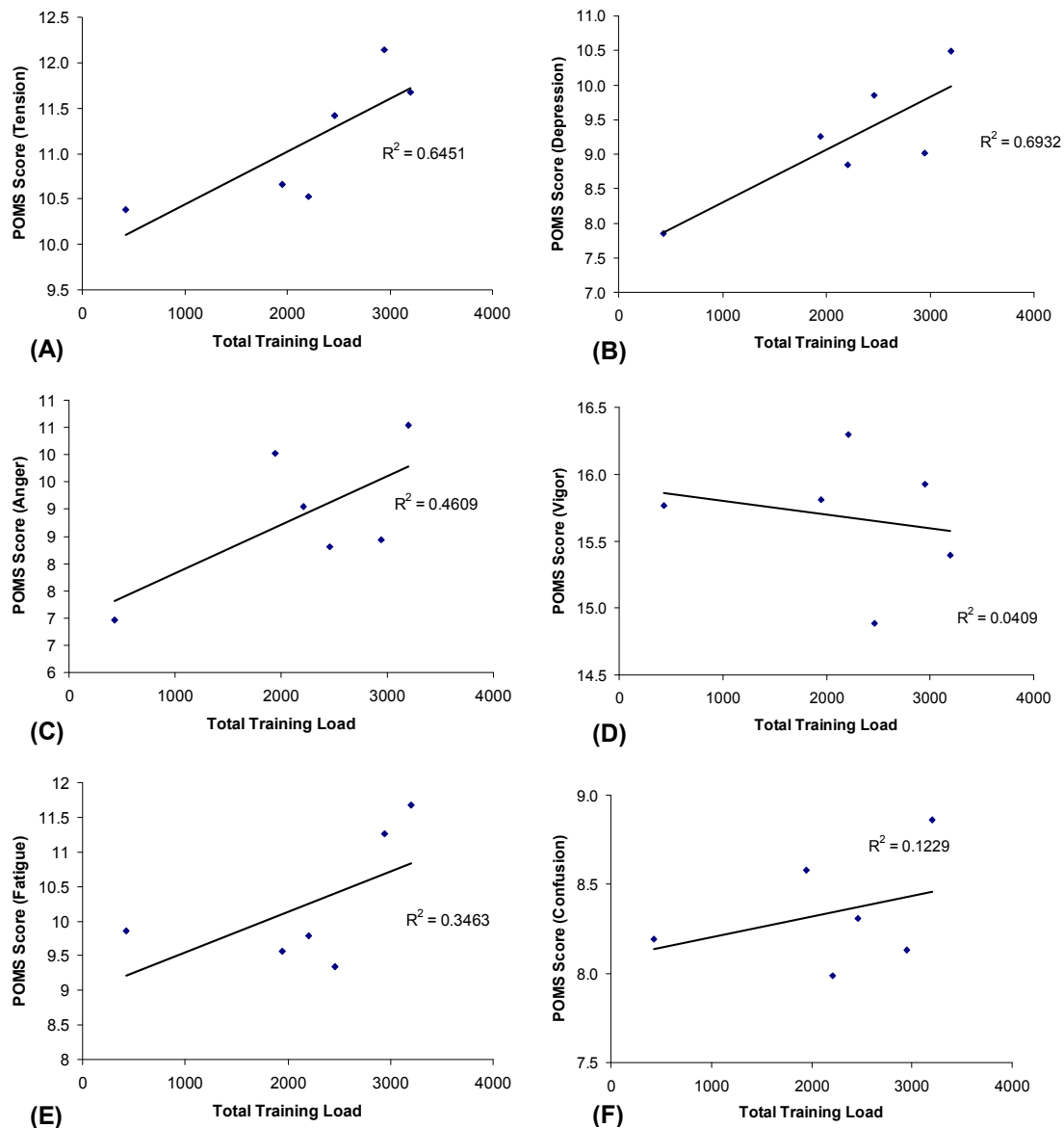
##### Training Load vs. Profile of Mood States (POMS)

The average monthly mean total mood disturbance (TMD) score was 25.9(±3.1). There was a positive relationship ( $R^2=0.4395$ ) between total training load and TMD (Figure 26). This relationship was non-significant ( $p>0.05$ ).



**Figure 26: Association between total training load and POMS total mood disturbance (TMD) (n=6)**

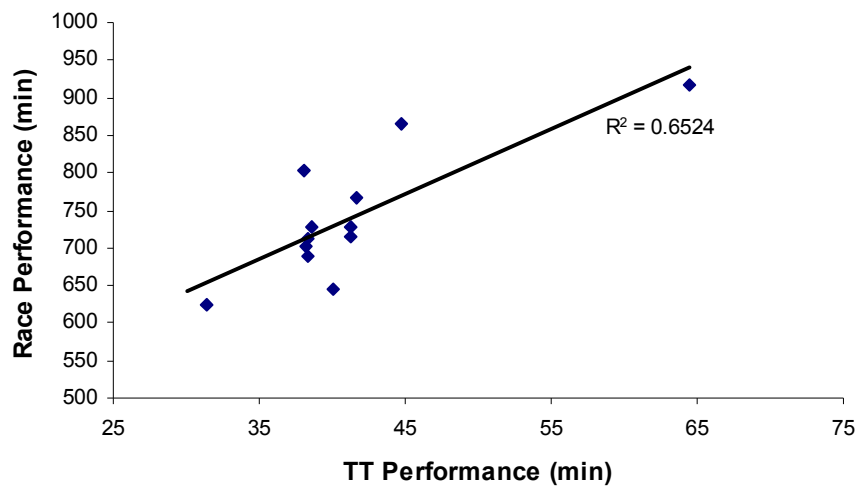
There were positive associations observed between training load and the POMS factors (Figure 27) Depression ( $R^2=0.6932$ ), Tension ( $R^2=0.6451$ ), Anger ( $R^2=0.4609$ ), Fatigue ( $R^2=0.3463$ ), Confusion ( $R^2=0.1229$ ). The strongest association was evident with training load and the POMS Depression score ( $R^2=0.6932$ ). Expectedly, there was a negative relationship between training load and the positive mood state Vigor ( $R^2= -0.0409$ ).



**Figure 27: Association between total training load and the mean Monthly (n=6) POMS factors tension (A), depression (B), anger (C), vigor (D), fatigue (E) and confusion (F)**

### Time-trial performance vs. race performance

The relative race performance of the athletes, measured in total time in minutes, was correlated to the mean time trial performance (5 separate time trial performances) of each athlete over the training period (Figure 28). The association was positive ( $R^2=0.6524$ ) and significant ( $p<0.05$ ) demonstrating athletes with faster time trial performances had faster finish times for the Ironman event. There were two athletes, from the sample, who did not finish the event, their mean time trial performances were 30 and 53 minutes.

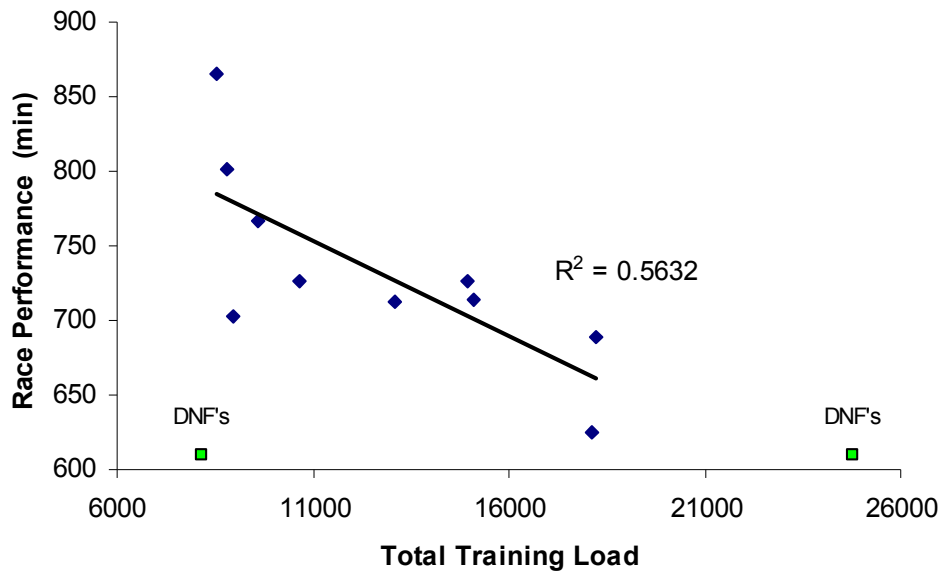


**Figure 28: Association between race performance and 8km submaximal running time trial performance**

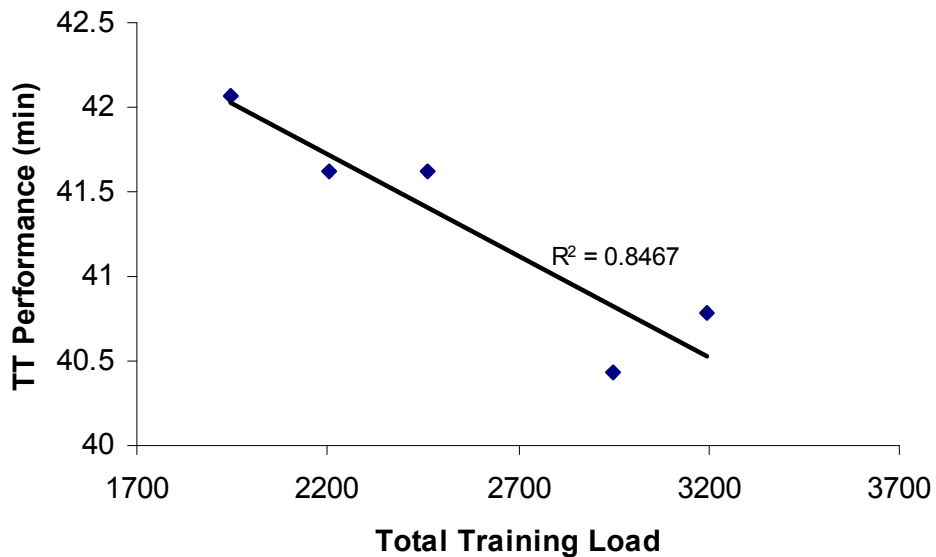
### Total training load vs. race performance

The total training load performed by the athletes for the six months of data collection (November to April) showed a negatively correlated association ( $R^2=0.5632$ ) with the overall race finishing time (min), the association was significant ( $p<0.05$ ). Two of the athletes did not finish (DNF) the race (Figure 29). These two athlete's training load is indicated in figure 29. These are absolute figures and therefore are not considered in the correlation. Athletes completing

more than 12645 TRIMPs of total overall training were significantly faster on race day ( $p < 0.05$ )



**Figure 29: Total training load vs. overall finishing times, DNF (did not finish) athletes included as absolute training loads (n=12)**



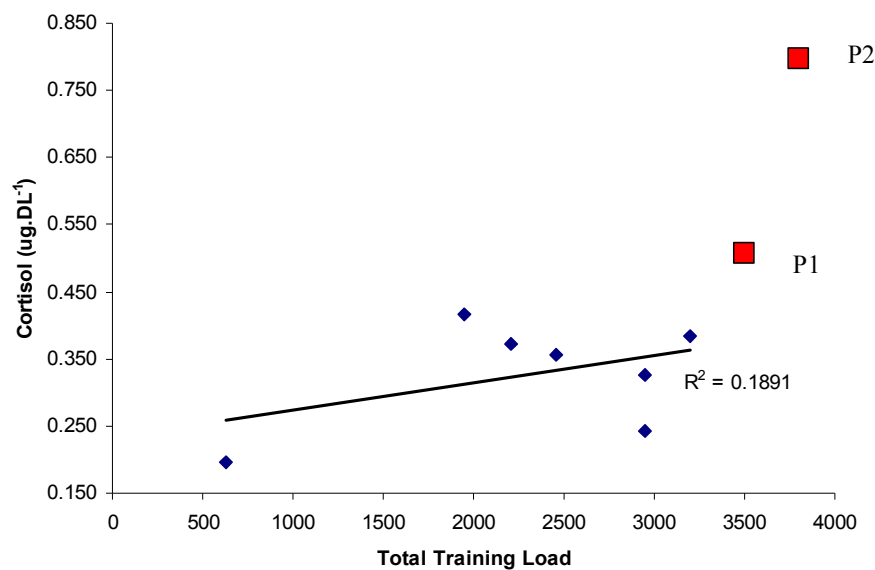
**Figure 30: Mean monthly total training load vs. TT performance (n=5)**

The mean of the five submaximal time trials (min) performed during the data collection period (November - April) were correlated to the mean total training load (Figure 30). There was a strong negative association ( $R^2=0.85$ ) between these two variables and the correlation was significant ( $p<0.05$ ).

### Total Training Load vs. Hormone concentrations

#### Cortisol

There was a positive but non-significant association ( $R^2=0.1891$ ) between the concentration of salivary cortisol and total training load, from sample 1 (November) to sample 7 (taken the day prior to the race). P1 and P2 indicate the mean race day hormone concentrations, for cortisol, taken at the finish line and 1.5 hours post finish. They are absolute figures and therefore were not considered in the correlation between training loads (Figure 31).

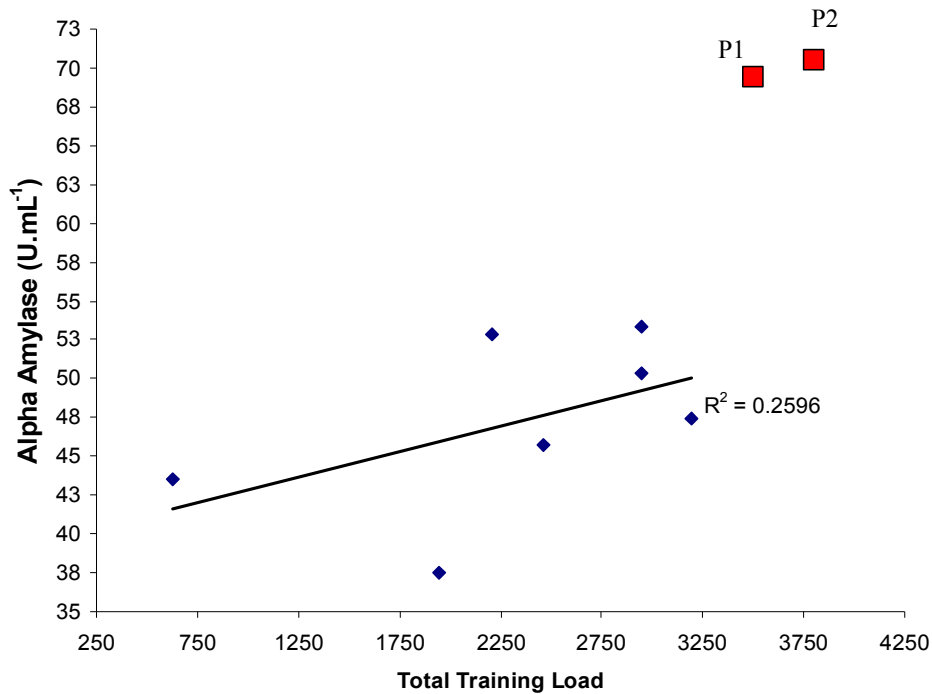


**Figure 31: Association between total training load and hormone concentrations of Cortisol ( $\mu\text{g.DL}^{-1}$ ) for the athlete group.**

(P1 indicates Finish line sample; P2 indicates 1.5 hours post finish sample)

### Alpha amylase

The concentration of salivary alpha amylase ( $\text{U.mL}^{-1}$ ) showed a positive but non-significant relationship ( $R^2=0.2596$ ) to total training load performed by the athletes, from sample 1 (November) to the sample 7 (taken the day prior to the race). While there is a positive association, it is not considered strong. P1 and P2 indicate the mean race day hormone concentrations for alpha amylase, taken at the finish line and 1.5hrs post finish. They are absolute figures and therefore are not considered in the correlation between training loads (Figure 32).



**Figure 32: Association between total training load and hormone concentrations of Alpha Amylase ( $\text{U.mL}^{-1}$ ) for the athlete group.**

(P1 indicates Finish line sample; P2 indicates 1.5 hours post finish sample)

## CHAPTER V

### DISCUSSION

#### ANTHROPOMETRY

##### BODY MASS

The control sample's mean body mass ( $90.5 \pm 17.8$ kg) was significantly greater than the athlete sample, which ranged from  $79.8 (\pm 9.6)$ kg to  $82.5 (\pm 10.8)$ kg. This was too be expected considering the control sample was largely sedentary (McArdle **et al.**, 2001). Further, the mean body mass index (BMI) of the control group was  $30 \text{ kg.m}^{-2}$ , placing them into an obese BMI category (McArdle **et al.**, 2001). The BMI of the athlete group ranged from  $24.9 (\pm 2.8)$  $\text{kg.m}^{-2}$  to  $25.6 (\pm 2.1)$  $\text{kg.m}^{-2}$ . While BMI readings greater than  $25 \text{ kg.m}^{-2}$  are considered overweight, individuals with a high lean body mass have distorted BMI readings, often placing lean individuals into higher categories (Eston and Reilly, 2001).

Recommendations for energy and macronutrient intake must be achieved in order to maintain body mass, recover muscle glycogen stores, regenerate tissue and supply essential fatty acids during ultraendurance training (Zalcman **et al.**, 2007). When training for the Ironman, meeting this intake can be challenging due to multiple training sessions performed per day. This investigation found no changes in body mass during the six month training period except for the month prior to the competition as well as the month of competition, where body mass dropped significantly. This shows that athletes were able to balance their energy expenditure and energy intake during training from November to February. This may be because triathletes seem to follow wiser nutrition habits than their single sport counterparts and avoid drastic reductions in body mass during training (Eichner, 1995).

The significant reduction in body mass in March signifies the start of the taper period and a reduction in training. This finding is unusual as meeting the energy intake demands should become easier during the taper period as activity energy expenditure has decreased significantly (Zalcman **et al.**, 2007). In fact athletes may even put on weight during this time as they are used to high energy intakes. However, a possible reason for this decrease in body mass may be due to athletes reducing energy intake particularly in the form of carbohydrate (CHO) to consciously decrease body mass prior to the race itself. While athletes are encouraged to continue high CHO intake during training and tapering, as is it beneficial to performance (Eichner, 1995; McArdle **et al.**, 2001; Bentley **et al.**, 2007; Zalcman **et al.**, 2007), this is often not the case as a reduction in body mass is also perceived as performance enhancing and can easily be achieved by reducing CHO intake and increasing protein intake (Eichner, 1995). This is due to the fact that for every gram of CHO stored, an additional 2.7 grams of water is stored (Maughan, 2005).

## **TRAINING MEASURES**

### **TRAINING LOAD AND PERFORMANCE**

While the wide range of performance capabilities (novice to professional), broadened the scope of the research, it did result in highly variable training regimes. A reflection of this was illustrated by the mean total training load of 12645( $\pm$ 4994) ranging from 8151 to 24806. There was a modest relationship ( $R^2=0.56$ ) between total training load and race performance (Figure 28). Thus, athletes with a greater training load elicited faster race times on the day. Top end 'elite' athletes tend to train significantly more than amateur athletes as their goal as professionals is not to merely complete the race but, to compete at a higher level of performance (Gublin and Gaffney, 1999). Half of the athletes fell either side of the mean total training load performed, and the race times for the athletes performing a training load above 12645 were significantly faster ( $p<0.05$ ). Those

performing less training (below 12645) showed a weak ( $R^2=0.32$ ) relationship with race performance while the higher training group was modestly correlated to race performance ( $R^2=0.54$ ). The question however remains as to whether these athletes were training optimally, particularly as of the eight athletes who took part in the 2008 Ironman South Africa, three improved their times, four had slower times and one athlete did not finish in 2009. To further investigate whether these athletes were training optimally it is important to note that two of them did not complete the event and interestingly these subjects were the two athletes at either extreme of the training load range. The athlete who did the least amount of training (8151) did not make the final bike cut-off (Neufer, 1989), whereas the athlete who performed the most training (24806) was unable to complete the run due to extreme fatigue symptoms including dizziness, confusion, vomiting, and collapse. While these symptoms may have been caused by the prevalence of non-functional overreaching or the overtraining syndrome, the prolonged, ultraendurance nature of a race such as the Ironman leaves many causes for failure on the day.

The percentage of total training load performed in each discipline aptly suits the total time spent completing each during the race, increasing progressively from swim (12.8%), to run (38.8%), to bike (51.6%). All of the training loads followed a cyclical 'hard-easy' pattern during training (Figure 13). This pattern shows that athletes have included recovery as a planned component to their training regime, aiding in reducing training stress (preventative role) and allowing for adaptation (permissive role) (Myburgh, 2003). During training, athletes promote an intended state of functional overreaching (Fry *et al.*, 1991) and then allow for super-adaptation to these training stimuli during a period of recovery, usually requiring a matter of days to a week (Lehmann *et al.*, 1993). While this method of training is generally accepted, if adequate recovery is not allowed for, over-reaching will persist to a state of non-functional over-reaching and could possibly further develop into the overtraining syndrome (Fry *et al.*, 1991).

## TAPERING AND THE RACE PERIOD

Tapering is defined by Myburgh (2003) as the recovery from hard training in preparation for major competition and is essential for optimal performance (Lac and Maso, 2004). While sport scientists are far from a complete understanding of the concept of optimal recovery, Noakes (2001) suggested that the length of the taper period is determined by the length of the race, as well as the intensity at which the athlete has trained. Therefore longer races, demanding more intensive training regimes, require more comprehensive taper periods.

Midway through training, there was a marked decrease in training load at approximately 11 - 12 weeks, this decrease in training correlated with the preparation (week prior to) and recovery weeks surrounding the 70.3 Half Ironman event in East London (South Africa), an event in which all but two of the athletes took part. This decreased training load prior to the 70.3 event indicates a short taper period (approximately 7-10 days) and while training load did decrease the other markers measured did not alter during this period. This race was not the major goal of these athletes, however, tapering before a competition enhances post event recovery enabling athletes to begin training for their goal race (Full Ironman) earlier than would be possible if the taper was not done (Child **et al.**, 2000).

### **Ironman taper period**

The cycling ( $1856.9 \pm 1070.9$ ), running ( $1041.9 \pm 500.9$ ) and total ( $3197.3 \pm 1454.1$ ) training loads all peaked in the month of February, while swim ( $500.6 \pm 558.2$ ) training peaked in March. A decrease in training load from this point allows athletes ample time to recover from a state of functional overreaching caused by heavy training, prior to the event (Coutts **et al.**, 2007a). The peak bike ( $2291 \pm 1859$ ) and run ( $1277 \pm 677$ ) training loads were completed at four and five weeks prior to the race. This taper in training coincides with anecdotal, yet proven successful evidence from Noakes' (2001) Lore of running 'learning from the experts' chapter (pages, 454-460), which covers Mark Allen's (six time

Ironman world champion) successful training regime and taper. The sample of athletes used in this investigation were questioned on their training regime and it was found that all were following either well developed anecdotal or scientifically produced training programs.

Peak swim training occurred later than the peak bike and run training weeks (three weeks prior). This was possibly due to the reduced muscle damage caused by low impact, non-gravitational stress sports such as swimming (Miller **et al.**, 2001). A minimum of ten days is required for competitive swimmers to adequately recover from training related stress (Lehmann **et al.**, 1994). In addition to this the physical demands of the swim leg of an Ironman triathlon is relatively small in comparison to the bike or run leg, therefore requiring a shorter taper period (Noakes, 2001). The significant fall in swimming, cycling and running training load in April was to be expected.

The two post race weeks (recovery weeks) were taken into account in the April measurement, and therefore reduced the total training performed in the competition month. However, the actual event was not taken into account when calculating this monthly load. The two weeks following the Ironman saw three athletes taking part in exercise training. One athlete continued run training in preparation for an ultraendurance marathon (56km, Two Oceans Ultra marathon) which was the week following the Ironman, and the other two athletes completed a short, low intensity bike training session. No swim training was performed by any of the athletes.

## **PHYSIOLOGICAL MEASURES**

### **WAKING HEART RATE**

Marked changes in waking and exercising heart rate have been found to occur as a result of endurance exercise and endurance training interventions. These changes have been put down to alterations to the autonomic nervous system (Hautala **et al.**, 2009). While this is the case, waking heart rates (WHR) of the

athletes did not change significantly during the study. However, the control group had a mean WHR of  $62(\pm 6.7)\text{bt}\cdot\text{min}^{-1}$ , which was significantly higher than that of the athlete's  $53(\pm 5.0)\text{bt}\cdot\text{min}^{-1}$ . Endurance exercise training results in an increased stroke volume and therefore an increased cardiac output but a decreased heart rate (McArdle **et al.**, 2001). These effects are also due to an increased left ventricular size, myocardial contractility and end diastolic volume (Spina, 1999). Endurance training also results in the sinus node of the heart being under a greater influence of acetylcholine, which is a parasympathetic hormone that slows heart rate (Seeley **et al.**, 1974). It is these endurance adaptations which partly explain the lower resting heart rates of the athlete sample (McArdle **et al.**, 2001). During the training period the athletic sample's heart rate did not decrease, this was possibly due to the already high level of training being performed prior to the start of data collection. Therefore, any decreases in heart rate due to physiological adaptation to endurance exercise may have already occurred. As an increase in resting/waking heart rate has been suggested as a symptom of overtraining (Noakes, 2001), the athletes were, according to this criterion, not overstressed or overtrained. Figure 14 illustrates that the week following the Ironman event (week 23) elicited significantly higher WHR values than a number of other weeks (13, 14, 15, 20 and 21). The event itself therefore placed considerable physiological stress on the athletes which persisted for a number of days post event, a result which corresponds with Gratze **et al.** (2005). These authors found a significant increase in resting heart rate values for two days after an Ironman event. While the use of changes in WHR as an indicator of overtraining has been reported, it has been questioned in prospective studies (Urhausen **et al.**, 2002).

#### POSTURAL DIZZINESS

Light-headedness or postural dizziness has been identified as a symptom of hypoglycaemia, hyponatremia and postural hypotension (Noakes, 2001). Chronic exercise reduces blood pressure, and heavy endurance training can therefore be the cause of light-headedness (Noakes, 2001). The control sample had a

significantly lower ( $p < 0.05$ ) postural dizziness score ( $1 \pm 0$ ) than the athlete sample for the duration of the data collection ( $1.3 \pm 0.5$  to  $1.4 \pm 0.4$ ). While the athlete sample was significantly higher than the control sample, the score correlated to a descriptive verbal interpretation of between 'no dizziness' and 'slight dizziness, disappears within 2 seconds'. These results would suggest that while the prevalence of postural dizziness was significantly greater than the control sample, it was not severe in nature.

## SLEEP

Regular endurance exercise training has been found to be therapeutic and have sleep promoting benefits (Driver and Taylor, 2000). In contrast, very long, strenuous exercise might disrupt sleep due to muscle damage and pain (Driver and Taylor, 2000). Further, insomnia symptoms such as trouble falling asleep, non-refreshing sleep, early awakening and others have been reported in individuals with high physical and emotional burnout questionnaire scores (Ekstedt *et al.*, 2009). The results show that there was no significant difference in the quality rating of sleep overtime within the athlete sample, or between the athletic and control samples. There was, however, a significant increase in quantity (hours of sleep) of sleep in the competition month ( $7.6 \pm 0.7$ hrs) in comparison to the five training months. The control group ( $7.9 \pm 0.8$ hrs) also slept significantly ( $p < 0.05$ ) more than the athlete group during the months the athletes were training, although, there was no significant difference during the competition month. Sleep disorders have been reported as a possible indicator of excessive stress and a symptom of suboptimal training and the overtraining syndrome (Urhausen *et al.*, 1998). While there is anecdotal evidence that proposes that overtraining results in sleep disorders, there is limited scientific evidence that confirms this fact (Driver and Taylor, 2000). Increase hours of sleep may also have been due to decreased training load and perhaps reduced early morning sessions.

The body restoration or compensatory theory of sleep proposes that the high catabolic activity caused by exercise will improve the conditions for anabolic activity during sleep (Driver and Taylor, 2000). According to this hypothesis, an increased need for restorative sleep after exercise is required (Oswald, 1983). This correlates with the significant increase in hours slept in the post competition week (week 23, Figure 16), allowing for the recuperation from the 'wear and tear' caused by the competition.

## **PSYCHOLOGICAL MEASURES**

Physical exercise has not only been found to have a positive influence on mental health and well-being, but substantial relationships have been found between exercise and the reduction of negative mood states (Morgan, 1997; Fox, 1999). Contrary to this, the results (Table VII, page 71) show that there was no difference in general mood state between the athletic and control samples, for all but one month (February), where the athletic sample had a significantly higher general mood state ( $1.6 \pm 0.4$ ). An increase in general mood state relates to an increased prevalence of negative moods. Further, negative mood ratings increased significantly in the athlete group two months prior to the race and elicited the only significant difference between the two groups. It was also at the same time that total training load peaked (month of February), further illustrating the sensitivity of the psychological markers (POMS and general mood states) to changes in training load performed. While it was expected that the athlete group would illustrate an 'iceberg profile' with lower negative, and higher positive profile of mood state (POMS) scores, results show that the mean POMS scores (positive and negative) for the athletes were all higher than the control group's scores (Figure 18). These findings may suggest that the athlete's were performing a training load beyond that of one which solely promotes physical well-being, and that as a result there may be higher negative mood states. However, the other variables of measurement, including, among others, waking heart rate (WHR), sleep ratings and postural dizziness suggest that the athletes

were not performing an excessive amount of training, placing them at risk for overtraining. It has been stated that there is a paradox concerning the effects of exercise and mood states (Morgan **et al.**, 1987). This was due to the confounding literature which shows positive mood states in athletes prior to becoming overtrained. There is a general agreement, however, that overtraining is characterised by a negative emotional state (Hooper **et al.**, 1997), therefore, the longitudinal assessment of mood states can be a valuable diagnostic tool for determining excessive training stress as well as symptoms of the overtraining syndrome (OTS) (Hooper and Mackinnon, 1995).

The POMS technical update (McNair and Heuchert, 2003), illustrates the concern that there is with past literature's inability to define which POMS score sheet has been utilised for analysis of results (Figure 18). The 'iceberg profile' (negative mood state T scores all fall below the population average of 50) which is said to be portrayed by athletes is only found when scored 'right now' on the college student norms score sheet but not when using the adult norms score sheet. This relationship is, however, not found when the same data is scored on the adult norms score sheet. While Figure 19 shows that there was a trend towards an 'iceberg profile', the changes to this profile over the months of training illustrated in Figure 19 alter this profile. The control showed a significantly similar trend to the athlete group. The mean scores on the adults norms sheet did not portray an 'iceberg profile' for the athletes but it did, however, also show no significant difference between the two groups.

Mood states have been found to fluctuate with significant changes in training volume and intensity (Morgan **et al.**, 1987; Hooper **et al.**, 1997). While there was a significant increase in bike training load in February, there were no significant increases in total training load from month one to month five. There was an expected, significant decrease in the training performed during the competition month (month six). Interestingly, fatigue scores in week 17 (month four) were significantly increased in comparison to weeks five and ten, which correlated to a

significant increase in bike training load ( $1856.9 \pm 1070.9$ ). Interestingly, although run training did not significantly increase in February, it did reach its peak ( $1041.9 \pm 500.9$ ) during this month, these two disciplines combined made up approximately 90% of total training during February. Figure 19 indicates that the period of peak training load (February) is correlated to an inversed iceberg profile (Morgan *et al.*, 1987), which is indicative of non-functional overreaching and the OTS. The inversed iceberg is characterised by increased fatigue, depression and anger scores as well as reduced vigor scores, which was scored on the college norms score sheet. Therefore, pointing out the sensitivity of the POMS questionnaire to increases in training load performed by the athletes (Eichner, 1995).

The progression from March to April showed that although an inversed iceberg profile was attained during February, the reduction in training (taper period) from the peak total training was adequate and of sufficient time to produce the iceberg profile at the time of competition, a profile indicative of positive mental health that is associated with successful athletic performance (Morgan *et al.*, 1987; Kellmann and Gunther, 2000). Tension scores for the athletes were significantly higher during the two weeks prior to the race during week 21 ( $13.7 \pm 5.8$ ) and week 22 ( $13.3 \pm 7.2$ ). This increase in tension is to be expected in the weeks or days prior to such an event. While the athletes elicited significantly increased tension scores surrounding the event, they also showed an absence of depressed mood states. Therefore the increased tension scores are proposed to be facilitative rather than debilitating, motivating that action is needed to achieve performance goals (Lane, 2001).

Total Mood Disturbance (TMD) has been reported to significantly increase during periods of overtraining (Kellmann and Gunther, 2000). The athlete's TMD was correlated to training load (Figure 26) and although not as strong as expected, there was a modest correlation ( $R^2=0.44$ ) between the two variables, this signifies that an increase in training load had some effect on mood disturbance of

the athletes (Lambert and Borresen, 2006). The negative POMS factor scores all showed positive relationships to total training load while vigor, the positive mood state showed a negative relationship. Depression ( $R^2=0.69$ ), tension ( $R^2=0.65$ ) and anger ( $R^2=0.46$ ), all showed modest correlations to training load, a finding which has previously been illustrated (Lane, 2001). A depressed mood has been suggested to create the inability to regulate the other dimensions of mood, thus leading to increases in anger, confusion, fatigue and tension, as well as a reduction in vigor (Terry and Lane, 2000). However, while tension and anger have moderate correlations with total training load, confusion, fatigue and vigor are weakly correlated to total training load.

## **BIOCHEMICAL MEASURES**

### **CORTISOL**

The continual exposure to stress may result in a prolonged activation of the hypothalamic-pituitary-adrenal (HPA) axis and therefore abnormal levels of glucocorticoid circulation (Grossi *et al.*, 2005). This response reduces adrenal responsiveness to adrenocorticotrophic hormone (ACTH), resulting in an increased cortisol concentration (Neary *et al.*, 2002; Hug *et al.*, 2003). There was however no change in cortisol concentration in this cohort. There are two likely explanations why the concentration of cortisol did not change over time with training. One is that the training load of the athletes was either optimal or not disproportionate to recovery (Fry *et al.*, 1991; Halson *et al.*, 2002). The other explanation is that the athletes could have demonstrated what is referred to as a non-responsive affect or response to ACTH, therefore blunting the cortisol response limiting the effects of training stress on the athletic group and reducing increases in cortisol concentrations. This response has also been found to occur during more pronounced stages of the OTS where the decreased adrenal responsiveness is no longer accounted for and therefore cortisol levels decrease. (Lehmann *et al.*, 1998; Hug *et al.*, 2002). As the athletes were already exposed

to relatively high training loads at the start of the data collection period, it cannot be negated that the cortisol blunting response may have been missed. It is, however, highly plausible that the response can be explained by the first theory as the other measures of fatigue indicated that the athletes were not overstressed, indicating that in general, training performed was balanced in nature (Rowbottom **et al.**, 1997).

Interestingly, the two athletes who did not complete the event, as well as one athlete whose actual finish time was significantly slower (2.5 hours) than predicted, illustrated negative relationships, although very weak ( $R^2=0.04$  to  $R^2=0.19$ ), between training load and cortisol concentration. All the other athletes showed a positive relationship between training load and cortisol concentration ranging from  $R^2=0.02$  to  $R^2=0.44$ . This suggests that these three athletes were not responding favourably to the training they were performing, and that they had possibly entered a state of reduced adrenal responsiveness synonymous with advanced overtraining symptoms (Lehmann **et al.**, 1998; Hug **et al.**, 2002).

It appears that while TMD scores are sensitive to small changes in training load, cortisol is not ( $R^2=0.13$ ). Rather, cortisol is affected by large increases in exercise volume as evidenced by the post competition measures. The cortisol concentrations for the athlete group post competition ( $0.65\pm 0.2 \mu\text{g}\cdot\text{DL}^{-1}$ ) were significantly greater than both the control and athlete training concentrations. As these samples were taken after the finish of the race, this was due to the exercise stress placed on these athletes due to the event (Rogers **et al.**, 1986; Rogers **et al.**, 1990). Cortisol has also been found to have a profound role in anti-inflammatory behaviour (Smith, 2000), and therefore is expected to be significantly increased post race due to the muscle damage caused by the race. It is widely accepted that psychological stress can produce similar effects to those produced by physical activity, therefore the psychological effects of completing such an event were additionally placed on the athletes (Takai **et al.**, 2004).

The control ( $0.26 \pm 0.032 \mu\text{g} \cdot \text{DL}^{-1}$ ) and athlete ( $0.33 \pm 0.08 \mu\text{g} \cdot \text{DL}^{-1}$ ) group's cortisol measurements did not differ during training (November to March). The expected 'normal' range for morning cortisol measures (including all necessary age categories) ranged from  $0.112 \mu\text{g} \cdot \text{DL}^{-1}$  to  $1.551 \mu\text{g} \cdot \text{DL}^{-1}$ , further indicating that the training load to which the athletes were exposed was possibly not excessive. The deviation amongst the control sample was substantially less than that of the athlete sample (Figure 21), which was likely due to the wide variability in training stress performed by the athletes (ranging from 8151 to 24806). This was in contrast to the control sample which did not have such a wide variation in concentrations. This lower variation could have possibly been due to the reduced amount of variables of stress placed on the control athletes in comparison to the athlete sample (more psychological stress than a combination of physical and psychological stressors (De Vente **et al.**, 2003).

#### ALPHA AMYLASE

The HPA axis and the sympathetic nervous system work together in coordination to produce changes associated with stress, however, alpha amylase and cortisol levels have been found not to correlate to one another (Chatterton **et al.**, 1996; Kivlighan and Granger, 2006; Nater **et al.**, 2006). Regardless of this, the alpha amylase results are similar to those of cortisol, with no changes in concentrations in response to training. While it cannot be concluded that this measure is a poor indicator of sympathetic activity, in contrast to the literature (Chatterton **et al.**, 1996; Walsh **et al.**, 1999; Skosnik **et al.**, 2000; Takai **et al.**, 2004; Kivlighan and Granger, 2006) it has not been found to be a sensitive measure of exercise stress during this study. The athletes did however seem to be following a balanced training regime (Rowbottom **et al.**, 1997) and therefore there were no significant fluctuations in the volume of training load performed. Daily alpha amylase levels have been found to be higher with chronic physiological and psychological stress (Nater **et al.**, 2007). Indicated by the average training concentrations of alpha amylase which were ( $47.2 \pm 31.9 \text{U} \cdot \text{mL}^{-1}$ ), and therefore at the lower end of the adult range ( $3.1 - 423.1 \text{U} \cdot \text{mL}^{-1}$ ) and below the mean of

92.4U.mL<sup>-1</sup>. There was a weak negative relationship ( $R^2=0.23$ ) found between Total Mood Disturbance (TMD) and alpha amylase concentrations.

While there was no distinct change in alpha amylase concentration in anticipation to competition, there was a trend for an increased alpha amylase concentration in response to competition although this change was not significant. This finding was in contrast to the literature which states that there is a significantly higher salivary alpha amylase concentration found in response to competition stress (Kivlighan and Granger, 2006; Nater and Rohleder, 2009) and during competition (Gilman **et al.**, 1979; Steerenberg **et al.**, 1997) as well as after competition (Chatterton **et al.**, 1996; Ljungberg **et al.**, 1997). Due to the fact that the athletic sample was so diverse in ability, the recorded competition stress placed on the athlete's during the event varied significantly due to the large deviations in competition duration, accounting for the large standard deviations and possibly, the non-significant result. Due to the nature of the event the carbohydrate ingestion during the event was not measured. Elevated alpha amylase concentrations due to the digestion of CHO found in sports nutrition products consumed during and post event have been set aside as research performed by Nater **et al.** (2007) found that there was no acute effect of eating and drinking on alpha amylase activity.

Competition-related changes (finish line and 1.5 hour post finish samples) in alpha amylase were significantly higher ( $p<0.05$ ) in the novice athletes ( $104.6\pm 69.6\text{U.mL}^{-1}$ ) in comparison to those athletes with experience in at least one Ironman event ( $50.7\pm 30.1\text{U.mL}^{-1}$ ). This result contradicts the findings of Kivlighan and Granger (2006) who found that more experienced athletes elicited higher alpha amylase concentrations in response to exercise. Due to the role of salivary alpha amylase in the digestion of carbohydrates, increased alpha amylase concentrations during and after intense exercise are expected. This is in order for energy stores to be adequately and effectively replenished (Lebanthal, 1987; Kivlighan and Granger, 2006). As exercise is a strong activator of the

sympathetic nervous system and previous studies have indicated that a high carbohydrate (CHO) diet, characteristic of an endurance athlete, is associated with an increased alpha amylase activity (Squires, 1953). There is consequently an inclination for athletes during training to have a greater alpha amylase concentration.

The athletic sample alpha amylase levels ( $47.2 \pm 31.9 \text{U.mL}^{-1}$ ) were not significantly higher than the control group's alpha amylase concentrations ( $35.9 \pm 9.5 \text{U.mL}^{-1}$ ). The control group did however have significantly lower alpha amylase concentrations than that of the athletes' competition levels ( $69.9 \pm 52.6 \text{U.mL}^{-1}$ ). While the diurnal rhythm of alpha amylase is influenced by age (Nater *et al.*, 2007), which varies significantly within the sample groups, the groups (athletic and control) were age matched and therefore invalidated any inconsistency which this could have caused.

Due to the once monthly sampling of the stress hormones, the time point of hormone sampling during the training cycles could have impacted the ability of the hormones to detect small changes in training load. More frequent testing could have enabled the identification of these small changes. This need for more frequent testing is illustrated by the weekly completion of the POMS questionnaire which, in week 17, detects a subtle increase in training load. An increase in hormonal response may have been overlooked if the testing performed was on a monthly data analysis rather than a weekly basis.

## **IMMUNOLOGICAL MEASURES**

A serious consequence of excessive training and non-functional overreaching may be the suppression of the immune system and a decrease in immune function which can lead to disruptions in training that can significantly affect performance (Fry *et al.*, 1991; Gleeson, 2002). However, while this is not universally accepted, anecdotal evidence suggests an increase in the prevalence of illness associated with overtraining (Smith, 2000). The trend in the results of

this study showed that the nature of the symptoms of illness changed between the training phase (November - March) and the competition phase (April). For example the signs and symptoms of upper respiratory tract infections (URTI) predominated in the training phase (sore throat ( $14.6 \pm 2.3$ ) and cough symptoms ( $9 \pm 4.8$ ) were the March reports per month) and fatigue related symptoms predominated in the competition phase, including headaches ( $6.2 \pm 3.83$ ) and fatigue ( $7.2 \pm 3.77$ ). The fatigue symptoms were probably induced by the muscle damage and inflammation caused by the event (Noakes, 2001). As athletes do not train the full race distance before competition, the unfamiliar effort required to complete the event results in the onset of delayed onset muscle soreness (DOMS) (Weerapong **et al.**, 2004).

The increased signs and symptoms of URTI are commonly observed during strenuous endurance exercise training (Peters and Bateman, 1983; Heath **et al.**, 1991; Nieman, 1994b). During the current study, there were only two reported cases of illness in the sedentary control group, there are a number of reported cases of illness (URTI predominating) amongst the athletic group during increased training loads. This relationship is expected as well as illustrated on the 'J' curve illustrated in Figure 8, where sedentary and overly active individuals are at greater risk of illness or URTI (Fry **et al.**, 1991).

The change in nature of the reported symptoms, around the competition period, shows that the decrease in training load, associated with the event taper, was sufficient to result in a decrease of reported symptoms of URTI (Nieman 1994a). During the two weeks post race there were fewer reports of URTI in response to a reduction in exercise load during this recovery period (Nieman, 1994a). In contrast, the symptoms which did prevail during the competition month (headaches and fatigue) were expected as this period is usually characterised by fatigue and headaches (Noakes, 2001).

The prevalence of headaches is more than likely due to a number of reasons, 'effort headaches' are not uncommon amongst ultraendurance athletes during or after such a gruelling event (Noakes, 2001). While these headaches are more commonly found in older less trained individuals, the high temperatures ( $>35^{\circ}\text{C}$ ) found on race day would have added to the environmental stress of such an event, this additional heat stress may have increased the stress of the event, adding to the prevalence of fatigue and headaches (Noakes, 2001).

## **PERFORMANCE MEASURES**

There was a trend for increased performance times in the monthly time-trial (Table XIX), from  $42.1(\pm 10.2)$  min in November to  $40.4(\pm 9.1)$  min in March, however this improvement was not statistically significant. Further, the mean total training load for each training month (November - March) was correlated to the mean time trial performance of the athletes during those months. It yielded a strong correlation ( $R^2=0.85$ ) which illustrates that an increase in training load results in an increase in submaximal time trial performance. These findings illustrate that there were no decrements in running performance, even with an increase in training load, a result which can define the athletes as 'not in an overtrained state' (Smith, 2000). The question remains whether these athletes were training optimally, or if they could have further increased their performance times. There was a modest positive correlation ( $R^2=0.65$ ) found between race performance and running time-trial performance amongst the athletes. This correlation indicates that time-trial performance is not only the 'gold standard' of predicting the onset of non-functional overtraining and suboptimal training, but is also a fair predictor of race performance (Noakes, 2001; Coutts *et al.*, 2007). This is possibly due to that fact that performance in the run leg of an Ironman is considered the most important factor in overall Ironman performance (Laursen *et al.*, 2002; Laursen *et al.*, 2005), and therefore is a better predictor of ability than swimming and cycling performance time-trials.

The amount of training performed by these athletes provides them with an excellent understanding of their personal performance capability and pacing strategy necessary for the race. The athletes' predicted finishing times ( $720.4 \pm 101.4$  min) and actual finishing times ( $741.2 \pm 85.2$  min) were similar, separated by only 20 minutes. This result was interesting as while the athletes may have performed 'brick sessions' (run training immediately after bike training) to prepare for the transition between cycling and running, none of the athletes from this study expectedly trained the full race distance, in sequence, prior to the race. However, they did take part in the half Ironman 70.3 to aid in the 'learning effect' of performing the disciplines sequentially. Therefore they would have never experienced the accumulative effects of fatigue encountered in the bike and run disciplines during training. Pacing therefore becomes a key determining factor of race performance.

One of the two athletes (aged 62 years), who did not finish the event, ran a mean time-trial of  $53(\pm 2.1)$  min and proposed that the excessive heat during the race resulted in the inability to finish. This has previously been shown to occur by Acevedo and Ekkekakis, (2001). The other athlete who did not finish, a professional athlete, had a mean time-trial performance of  $30(\pm 1.6)$  min. While this was the fastest mean time trial performed, this athlete collapsed during the run leg and did not finish the race. This athlete complained of severe fatigue symptoms and gastrointestinal discomfort prior to and during the event. This discomfort prevented the athlete from adequate nutritional intake and therefore inevitably the inability to finish (Jeukendrup, 2004; Bentley **et al.**, 2007).

With respect to the hormonal measures, there was no correlation between race performance and alpha amylase ( $R^2=0.003$ ) or cortisol ( $R^2=0.003$ ). This highlights the fact that regardless of performance time, the athletes' are under similar stress post event. While the same net energy cost value has been used to perform the event by each athlete, the variation in body mass between the athletes suggests that the heavier athletes would have been under greater stress

and elicited higher stress hormone responses (Steerenberg **et al.**, 1997; McArdle **et al.**, 2001). This was reflected by the large variability in hormonal responses within the group.

## **INTEGRATED DISCUSSION**

*NOTE: This section pertains only to the responses of the athletes.*

The most noteworthy finding from this investigation was that an increase in total training load accounted for approximately 60% of the performance variability, and that increases in training load were strongly correlated with time trial performance ( $R^2=0.85$ ). However, what these results do also highlight is that not all responses are influenced by training and competition in the same manner.

Table XX provides a crude overview of the responses during training only. What is most interesting is that training load was not significantly altered during the training period. This could be due to high training loads at the start of the study or that the high variability in responses, due to the wide range of athletic capabilities, masked any significant findings. This may hold true for many of the other findings. It was also apparent that the sample of athletes were not over trained as time trial performance was not negatively impacted and improved marginally although not significantly, over time. There was evidence that the immune system was compromised by the training as there was an increased prevalence of upper respiratory tract infections (URTI) in all of the months except for month four. This was despite the fact that in most of those months the other responses remained stable.

Changes in mood states were more apparent as race day approached (Table XX). For example, tension scores were significantly increased for the two weeks prior to the race, a mood change that is expected during the final preparation for such an event. Fatigue scores were also significantly increased and this elevated response coincided with the period of peak training performed (week 17). The

physiological and biochemical parameters were not altered during the training period suggesting that training load was not excessively stressful on these systems. However, cortisol concentration ( $0.33 \pm 0.08 \mu\text{g} \cdot \text{DL}^{-1}$ ) did fall on the lower end of the normative range for the general population ( $0.112 \mu\text{g} \cdot \text{DL}^{-1}$  to  $1.551 \mu\text{g} \cdot \text{DL}^{-1}$ ). While these results could suggest that there was a possible blunting effect due to training, the control sample's concentration was similar ( $0.26 \pm 0.032 \mu\text{g} \cdot \text{DL}^{-1}$ ) and therefore dismissed down-regulation of pituitary hormone release as a possible cause for these low concentrations. Alpha amylase followed a similar trend.

**Table XX: Comparative analysis of data during training (month 1 – month 5)**

Month	Training Load	Physiological	Psychological	Biochemical	Immunological	TT
1	↔	↔	↔	↔	↑ URTI	↔
2	↔	↔	↔	↔	↔ URTI	↔
3	↔	↔	↔	↔	↑ URTI	↔
4	↔	↔	↑ F Scores (week 17) ↓ General mood	↔	↔ URTI	↔
5	↔	↔	↔	↔	↑ URTI	↔

NOTE: Only Significant changes are indicated  
 (URTI = Upper Respiratory Tract Infections)  
 (Physiological = Waking heart rate, postural dizziness, sleep ratings)  
 (Psychological = General mood, POMS)  
 (Biochemical = Cortisol and Alpha amylase concentrations)  
 (Immunological = Symptoms of illness)  
 (TT = Time Trial performance times)

In contrast to the training data, the post-competition responses clearly indicate that the excessive competition load negatively affected most of the measures obtained (Table XXI). Cortisol spiked immediately after the race and waking heart

rate was elevated in the two weeks post competition. Athletes also reported increased postural dizziness in the weeks following competition and while the incidence of URTI decreased, there was an increased prevalence of headaches and fatigue still indicating compromised immune function.

**Table XXI: Comparative analysis of data during competition (month 6)**

Month	Training Load	Physiological	Psychological	Biochemical	Immunological	TT
6	↓	↑WHR ↑Postural Dizziness	↑ T Scores (weeks 21, 22) ↓ T Scores (weeks 23, 24)	↑Cortisol (Post race)	↓URT ↑ Fatigue/ Headaches	↔

There was possibly one athlete who was over trained and this was one of the elite, male athletes in the sample who had a very high training volume and much higher than the rest of the sample. The difference in training load between this athlete and the athlete with the next highest training load was 2527.7 units of training. In fact, the mean training load of the sample (2262.9) was even below this difference. This athlete showed signs of increased prevalence of illness during training, recording an average of ten symptoms per month. These symptoms were predominated by URTI and gastrointestinal symptoms. This athlete also illustrated a higher negative general mood rating. While these markers pointed to excessive overload, the trend in performance of the submaximal running time trial decreased, although this was not significant.

Overall the study emphasizes the importance of using multiple measures to assess demands placed on athletes as some measures are more sensitive than others at different times surrounding training and competition. Further, individual monitoring is important as all athletes respond differently depending on the demands placed on them by training and other lifestyle factors not accounted for in this investigation.

## **CHAPTER VI**

### **SUMMARY, CONCLUSIONS AND RECOMMENDATIONS**

#### **INTRODUCTION**

During the ultraendurance training required in preparation for an event such as the Ironman, an athlete's body is placed under numerous types of stress. It is therefore important to monitor the balance of training, competition and recovery to assist in optimising performance. Very few studies have longitudinally assessed what types of demands are being placed on all levels of athletes independent of research manipulation. This study therefore sought to better understand how athletes trained in preparation for such an event and then how their training load impacted physiological, biochemical, immunological and psychological parameters.

#### **SUMMARY OF PROCEDURES**

The study took a longitudinal, field based approach assessing ultraendurance triathletes training for the Ironman triathlon in South Africa. Consultation with a number of researchers, with relevant domain experience, aided in the authors knowledge and provided invaluable guidance. These domains included: physiological (waking heart rate; postural dizziness; sleep ratings), psychological (general mood; profile of mood states), biochemical (salivary alpha amylase and cortisol), immunological (symptoms of illness) and performance (submaximal running time-trial and race) markers, as well as training details (duration, time, perceived effort and muscle discomfort). Athlete recruitment took place at the exhibition and registration of the 2008 Ironman South Africa as well as at a

number of multisport races prior to the start of data collection. Two triathlon stores (one in Johannesburg and one in Port Elizabeth) were approached for their assistance in hormone sample collection. Triathletes handed in their samples to these stores before collection by the author or a logistics company. Hormones were stored at the Rhodes University Biochemistry department (Eastern Cape samples) and the Tshwane University of Technology (Gauteng samples) at  $-68.4^{\circ}\text{C}$  prior to analysis.

Eighteen ultraendurance triathletes ( $82.0\pm 10.0\text{kg}$  body mass;  $1796\pm 62\text{mm}$  stature) from around South Africa and 15 control subjects ( $90.5\pm 17.8\text{kg}$  body mass;  $1738\pm 73\text{mm}$  stature) from Grahamstown, Eastern Cape, were recruited for the study. Prior to the start of data collection, the athletes attended a habituation and orientation session. During this session, the requirements and procedures of the study were explained, information documents, recording books and salivary sampling kits were handed out and any questions pertaining to the project were answered. All subjects signed an informed consent to a protocol approved by the Rhodes University Ethics Committee. Data collection began in November 2008, six months prior to the Ironman in South Africa in 2009, where athletes began completing the daily questionnaire, weekly profile of mood states questionnaire, monthly submaximal time-trial and date-specified hormone sampling. Data was submitted after each month and was analysed to ensure that it was being accurately completed. Any issues of concern surrounding data collection were raised with the relevant athletes.

Over the race week additional hormonal samples were taken, one sample the day prior to the event and two post event samples were collected (immediately after finish and 1.5 hours after finish). Data collection continued for two weeks post Ironman South Africa, where follow-up measures were taken. All data, apart from hormonal samples, were collected during this two week period. The control subjects were analysed for a three week period, this collection period fell over the

competition month. The control group submitted the same data as the athletic group for this period.

## **SUMMARY OF RESULTS**

The large standard deviations found between the training loads performed by the athletes were a result of the wide range of performance capabilities. During the course of data collection these training loads ranged from  $1947.2 \pm 901.7$  (November) to  $3197.3 \pm 1454.1$  (February) and dropped to  $427.4 \pm 485.4$  in the competition month. The cycling ( $1856.9 \pm 1070.9$ ), running ( $1041.9 \pm 500.9$ ) and total ( $3197.3 \pm 1454.1$ ) training loads all peaked in the month of February, while swim ( $500.6 \pm 558.2$ ) training peaked in March. There were two taper periods illustrated in the results, a short period surrounding the 70.3 Half Ironman event in East London (South Africa) and a longer period starting from the peak training periods in February (cycling and running) and March (swimming). These taper periods were allowed to assist in recovery for optimising performance in the relevant races (Noakes, 2001).

Physiological data showed that there were no decreases in mean waking heart rate amongst the athletes during training. There was however an increase in WHR the week post event (week 23), reflective of competition load and stress. The sedentary control group had significantly greater WHR values, a finding which is expected due to the cardiovascular adaptations of endurance exercise (McArdle *et al.*, 2001). Postural dizziness scores amongst the control sample were lower ( $1 \pm 0$ ) than the athlete sample for the duration of the data collection phase ( $1.3 \pm 0.5$  to  $1.4 \pm 0.4$ ). There were no differences in the quality ratings of sleep within the athlete sample, or between the athletic and control samples. There was, however, a significant increase in quantity (hours of sleep) in the competition month ( $7.6 \pm 0.7$  hrs). The control group ( $7.9 \pm 0.8$  hrs) slept more than the athlete group during training, although, there was no difference during the competition month.

The profile of mood states (POMS) fatigue (F) scores were significantly increased during a period of significantly increased bike training load (week 17, month four), illustrating the sensitivity of the POMS to changes in training stress. Run training did not significantly increase during training although it did reach its peak ( $1041.9 \pm 500.9$ ) during February (month four). These two disciplines combined made up approximately 90% of total training during February. The peak training load (February) was correlated to an inverse iceberg profile (Morgan **et al.**, 1987). This profile is symptomatic of overtraining, however, the reduction in training (taper period) from the peak total training was adequate, and of sufficient time to produce the iceberg profile at the time of competition, a profile indicative of positive mental health that is associated with successful athletic performance (Morgan **et al.**, 1987; Kellmann and Gunther, 2000). Tension scores were increased in the weeks surrounding the event (week 21:  $13.7 \pm 5.8$ , week 22:  $13.3 \pm 7.2$ ). They also showed an absence of depressed mood states, therefore these increased scores are proposed to be facilitative, and motivating that action is needed to achieve performance goals (Lane, 2001). The athletes' TMD was modestly correlated ( $R^2=0.44$ ) to training load. The negative POMS factor scores all showed positive relationships to total training load while vigor, the positive mood state, showed a negative relationship. Depression ( $R^2=0.69$ ), tension ( $R^2=0.65$ ) and anger ( $R^2=0.46$ ), all showed modest correlations to training load, a finding which has previously been illustrated (Lane, 2001). The mean POMS scores (positive and negative) for the athletes were all higher than the control group's scores.

Athletes who did not finish (DNF) or underperformed at the event showed a weak negative relationship ( $R^2=0 - 0.19$ ) between cortisol and training load. This is in contrast to the athletes whose predicted finishing times were not significantly different to their actual finishing times. Athletes who finished within predicted finish times all showed positive relationships between cortisol and training load. This suggests that the athletes with negative relationships may have been reacting unfavourably to training stress. The control ( $0.26 \pm 0.032 \mu\text{g} \cdot \text{DL}^{-1}$ ) and

athlete ( $0.33 \pm 0.08 \mu\text{g} \cdot \text{DL}^{-1}$ ) group's cortisol concentrations did not differ during training (November to March) and fell within the 'normal' range for morning cortisol measures ( $0.112 \mu\text{g} \cdot \text{DL}^{-1}$  to  $1.551 \mu\text{g} \cdot \text{DL}^{-1}$ ). Alpha amylase concentrations amongst the athletic sample ( $47.2 \pm 31.9 \text{U} \cdot \text{mL}^{-1}$ ) were not significantly higher than the control group's concentrations ( $35.9 \pm 9.5 \text{U} \cdot \text{mL}^{-1}$ ). The control group did however have significantly lower alpha amylase concentrations than that of the athletes' competition levels ( $69.9 \pm 52.6 \text{U} \cdot \text{mL}^{-1}$ ). As with cortisol, alpha amylase concentrations did not alter during training, and was not found to be sensitive to small changes in training volume during this study. The mean concentrations of alpha amylase during training was  $47.2 (\pm 31.9) \text{U} \cdot \text{mL}^{-1}$  which was at the lower end of the adult range ( $3.1 - 423.1 \text{U} \cdot \text{mL}^{-1}$ ) and below the mean of  $92.4 \text{U} \cdot \text{mL}^{-1}$ . Competition-related changes (finish line and 1.5 hour post finish samples) in alpha amylase were significantly higher ( $p < 0.05$ ) in the novice athletes ( $104.6 \pm 69.6 \text{U} \cdot \text{mL}^{-1}$ ) in comparison to those athletes with experience in at least one Ironman event ( $50.7 \pm 30.1 \text{U} \cdot \text{mL}^{-1}$ ). This result contradicts the findings of Kivlighan and Granger (2006) who found that more experienced athletes elicited higher alpha amylase concentrations in response to exercise.

The trend in the results was for time trial (TT) performance to increase during the progression through training. These increases in performance were, however, not significant. Nevertheless, as a decrease in performance has been established as the 'gold standard' of suboptimal training and therefore the absence of a decrease in TT performance is, in itself, a significant finding. The correlation between TT performance and training load was  $R^2 = 0.85$ , while its relationship with race performance was  $R^2 = 0.65$ . These results suggest that there was a strong association with running capability and Ironman triathlon race performance.

The prevalence of upper respiratory tract infection (URTI) symptoms increases with endurance type training (Gleeson, 2002). This was evident in this finding as during periods of high training load, URTI were common. During the reduced

period of training surrounding the taper period these symptoms are reduced. Competition, however, has its own stresses and shows symptoms of fatigue and headaches (Noakes, 2001).

## **HYPOTHESES**

1)

- a) With respect to the physiological measures, the null hypotheses should be tentatively accepted for waking heart rate values, postural dizziness scores, sleep quality ratings and sleep quantity throughout training, but rejected for waking heart rate and postural dizziness scores for the week (week 23) following the Ironman race. The null hypotheses for sleep quantity should be rejected for the competition month (week 22, 23 and 24).
- b) With respect to the Profile of Mood States (POMS) data, the null hypotheses must be tentatively accepted for months 1, 2, 3 and 6, but rejected for months 4 and 5. The null hypotheses for total mood disturbance (TMD), depression (D), anger (A), vigor (V) and confusion (C) should be tentatively accepted for the duration of data collection. The null hypotheses for the tension (T) scores are to be tentatively accepted except for weeks 21 and 22 which were significantly higher than weeks 23 and 24. In these instances the null hypothesis is rejected. The null hypotheses for the fatigue (F) scores are to be tentatively accepted except for week 17, where it should be rejected.
- c) With respect to cortisol, the null hypotheses for the training samples should be tentatively accepted, and for the competition samples, rejected.
- d) The null hypotheses for the alpha amylase samples should be tentatively accepted during both training and post-competition.

- e) The null hypothesis for the time trial (TT) results should be tentatively accepted.
- 2) The athlete and control group were compared for all data. The six months of data collection from the athletes was compared to one months data from the control sample.
- a) The null hypothesis for the comparison of physiological data between groups should be rejected for WHR and postural dizziness. The null hypothesis for the sleep ratings is to be tentatively accepted for all months except for April where it should be rejected as sleep quantity is significantly increased in the athlete sample and is therefore similar to the control sample's results.
  - b) With respect to general mood states data, the null hypothesis should be tentatively accepted for all months except February (month four), where it should be rejected as the athlete samples' mood states are greater than the control sample. POMS comparative data should be tentatively accepted.
  - c) The null hypothesis concerning the cortisol concentrations should be tentatively accepted for training values, but rejected in the case of the competition samples as the athlete's cortisol concentrations are greatly increased.
  - d) The null hypothesis must be tentatively accepted for the alpha amylase concentrations between the control and athlete training values, but rejected in the case of the competition samples.

## **CONCLUSIONS**

The results of the current study identify the importance of taking a holistic, longitudinal approach in the analysis of a group of ultraendurance triathletes. This approach allows athletes to identify any important changes in response to training. These athletes did not show signs that they were overtrained. However from the start there was a high prevalence of URTI indicative of a compromised immune function. The other responses show no indication of excessive overload except at week 17 when the POMS data showed high levels of perceived fatigue. During competition and post competition symptoms of fatigue and headaches predominated, indicating symptoms of high levels of competition stress.

## **RECOMMENDATIONS**

Future research into performance changes and fatigue markers surrounding the training for an ultraendurance triathlon should take into consideration the following recommendations:

1. The data collected for the longitudinal analysis of these athletes should begin earlier along the time course of the training regime and should include more frequent data collection points to enable a greater reliability and accuracy of data.
  - a. A longer time course would allow for investigating the period where training load increases from initial training up to the 'stable' level evident in the current study. This early increase in training could have a significant influence on the progression of training through to the event.
  - b. While a number of the variables were measured on a daily basis, the performance time trial was only measured monthly and

biochemical samples were only taken on nine occasions. More frequent measurements would increase reliability and validity.

2. Future studies should take a comparative stance when analysing the time course of effect of the measures from all domains:
  - a. The point where performance declines, the 'gold standard' of suboptimal training, should be related to the changes in other measures. This enables the identification of early diagnostic markers of suboptimal training.
  - b. Future studies should consider the use of immunological markers along with other responses. The sensitivity of immune system to training is evident in this research and requires more in-depth analysis.
3. While *in situ* analyses have practical applicability, less 'hands off' testing is recommended. An increased number of laboratory tests on a greater number of locally based athletes should be utilised to ensure a higher level of compliance to the testing protocol.
4. Investigating a group of athletes with a wide performance capability increases the scope of a study. However it is recommended that future research selectively groups ability, therefore analysing a more homogeneous sample.

The current study identified that the athletes assessed in this study who were training for the ultraendurance triathlon, Ironman, were following well developed training regimes. This is reflected in the limited changes to the fatigue and performance markers utilised in this study. The peak in training found in February was most notably indicated by the changes in the profile of mood states,

although there seemed to be sufficient taper allowed by athletes to obtain optimal performance on race day. The stress of the event placed the athletes under the greatest demands, this stress was illustrated by the significant changes to a number of markers, and these included waking heart rate values, immunological responses, postural dizziness and hormonal concentrations.

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## **APPENDICES**

**Appendix A: Flyer for Athlete Recruitment**

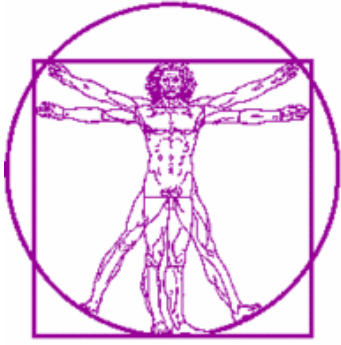
**Appendix B: Letter of Information**

**Appendix C: Data Collection Instructions (Athlete and Control Group)**

**Appendix D: Data Collection Sheet (Control Group)**

**Appendix E: Informed Consent**

**Appendix F: Statistical Table**

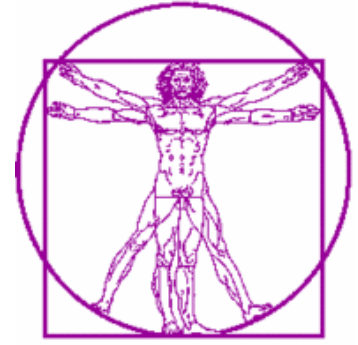


APPENDIX A



**RHODES UNIVERSITY**

*Grahamstown • 6140 • South Africa*

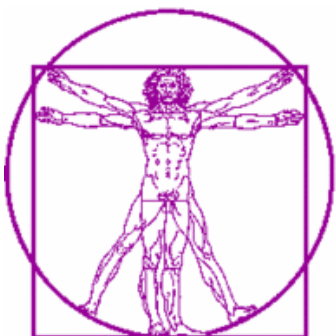


# IRONMAN RESEARCH

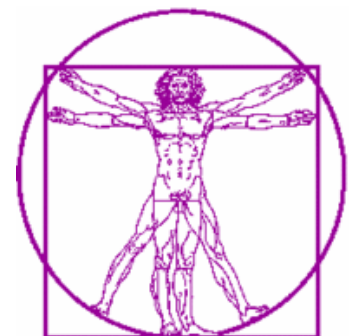
**During your training for the 2008 Ironman South Africa, did you, at times, feel tired, depressed and unmotivated... Did you consider that your training may have been suboptimal?**

**The Rhodes University Human Kinetics and Ergonomics department will be conducting research on training load in Ironman athletes. The study will be focused on athletes training for Ironman South Africa 2009 and will be run over the 8 month period prior to next years event. We need as many athletes as possible, country wide to take part in our study, so if you are keen to be involved in cutting edge research then contact Alex Joiner at [bokkiejoiner@gmail.com](mailto:bokkiejoiner@gmail.com). Once you indicate your intent to participate, I will give you more details – however, time commitments are minimal!!!**

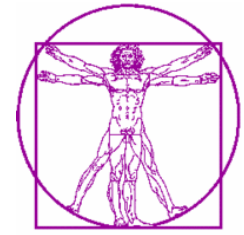
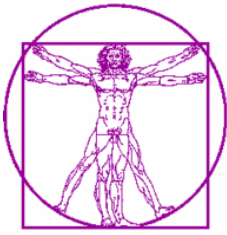
**Are you ready to enhance your performance?**



**For further information feel free  
to contact us!  
[bokkiejoiner@gmail.com](mailto:bokkiejoiner@gmail.com)**



## APPENDIX B



**RHODES UNIVERSITY**

*Grahamstown • 6140 • South Africa*

**HUMAN KINETICS & ERGONOMICS**

Cell: 072 116 3939 • Fax: (046) 603 8934 • E-mail: bokkijoiner@gmail.com

Dear Athlete

### **INVOLVEMENT IN MASTERS RESEARCH: IRONMAN SOUTH AFRICA**

Firstly, I must thank you for showing interest in my research, I trust that the data that are produced by the study will aid in your training and performance in the future, whether it be for the Ironman or for any other endurance event. The present project will be investigating performance changes and fatigue markers which are evident during the course of training for an Ironman triathlon. By correlating performance with fatigue markers the study aims to determine the most practical and efficient method of identifying suboptimal training, aiding in the development of optimized training techniques.

Due to the nature of the Ironman, the importance of the training component for such an event is highlighted by the extreme distances covered as well as the large commitment of time and money spent on the preparations prior to, and including, race day. It is therefore of utmost importance that training for the event is at optimal efficiency, and finding the balance between training/competition and recovery is imperative to performance. As I am sure most athletes understand, too little training will result in poor performance. However, too much training may also result in a condition that negatively impacts performance, the overtraining syndrome (OTS). This is a debilitating disorder that can take months of rest to make a full recovery. The importance of this intricate balance between too little and too much training is difficult to achieve, and an extensive review of the literature highlighted the need for further research into training and fatigue markers

likely to develop into the OTS. My primary objective is to establish whether some early onset chronic fatigue indicators can be detected and hence necessitate an adaptation to the training regime.

The study is of longitudinal nature and will therefore take place over a period of time, specifically 5 months leading up to the Ironman South Africa 2009. During this time you will be required to complete a number of activities including a day-to-day training log, a weekly mood state questionnaire (POMS), a monthly 8km submaximal running time-trial, and produce saliva samples at 10 stipulated times, mostly during the training period but also prior to and after the event next year. The instructions of how to complete these tasks are documented in this data file. Excluding the salivary sample, which literally requires you to 'drool' into a small plastic container (cryogenic vial), and the POMS questionnaire, all of the other requirements are likely things you would do normally (some may be slightly modified).

Please read through this file carefully before proceeding any further with the study. It is necessary, once you have read through the entire file and are happy that you have been fully informed of the nature of the study, that you complete the 'letter of informed consent' and send it back to me via the envelope enclosed in the file. Please note that you can withdraw from the study at any time and are under no obligation to me or the Department of Human Kinetics and Ergonomics. If you have any questions, please feel free to contact me (see above for contact details).

Yours sincerely

Alex Joiner  
*BSc Hons (HKE)*  
*MSc Student*

## APPENDIX C

### DATA COLLECTION INSTRUCTIONS

**\*Please read these instructions carefully before signing the letter of informed consent or commencing with the completion of the data forms.**

#### 1. DAY-TO-DAY TRAINING LOG

These instructions are to aid in your completion of the day-to-day training log. Please ensure that you have read these instructions, as well as gone through the log itself before commencing with recording data.

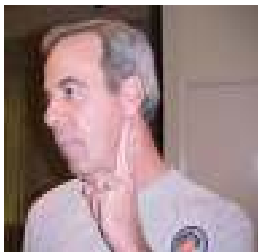
The training log (page 11/**Sheet 1 (day 2 day log)**) is to be filled out on a daily basis for the duration of the study. It is divided into ten categories which include waking heart rate, hours of sleep, sleep rating, body mass (to be measured in the morning on awakening), postural dizziness, symptoms of illness, general mood rating (measured in the evening), training details, perceived training effort and muscular discomfort during training (measured after completion of training sessions). The specific ratings for each of these is given on page 11.

**\* Please note that it is extremely important that the answers given here are as honest and accurate as possible.**

#### RECORDED ON AWAKENING

##### Waking Heart Rate:

This reading should be taken each morning while still in bed. After waking continue to lie relaxed for approximately 5 minutes. Heart rate can then either be taken by manual method, by palpation/touch at the neck/carotid artery (a) or at the wrist/radial artery (b) or, by the use of a heart rate monitor. To take a heart rate by manual method, place your index and middle fingers on the relevant artery and feel for a pulse. Once you can feel a pulse, count the number of beats for a one minute period. The use of a heart rate monitor to record waking heart rate will provide more accurate and consistent measurements. Please use the same method of measurement throughout the project.



(a)



(b)

**Manual heart rate palpation sites (a) and (b)**

**Hours of Sleep:**

Record the number of hours slept, rounding off to the nearest 30 minutes, e.g. 7h30, 8h00, etc...

**Sleep Rating:**

The quality of sleep can be just as important as the quantity, therefore it should be graded according to the following:

Gr. 1 = Uninterrupted sleep, refreshed

Gr. 2 = Interrupted sleep, however do not require more sleep

Gr. 3 = Interrupted sleep, do require more sleep

Gr. 4 = Interrupted sleep, feeling terrible

**Body Mass:**

Body mass should be recorded using the same scale every morning; this measurement should be taken before the consumption of anything per mouth (food or drink). Before weighing, go to the toilet (if you do not need to, that is fine). Remove all clothing and jewelry and stand upright in the middle and wait for the reading, then record.

**RECORDED IN THE EVENING**

\*At the end of every day, once you have completed your daily training, record the following data:

**Postural Dizziness:**

This measurement refers to any dizziness/light-headedness (feeling of ‘head rush’) felt when standing from a seated or lying position, this should be graded throughout the day as follows:

Gr. 1 = No dizziness

Gr. 2 = Slight dizziness, disappears within 2s

Gr. 3 = Marked dizziness, time taken to orient one’s self

Gr. 4 = Marked dizziness, requiring sitting or lying down again

**Symptoms of Illness:**

Record the relevant number(s) if you are suffering from any of the following symptoms:

1 = sore throat

2 = cough

3 = nausea and/or vomiting

4 = diarrhoea

5 = stomach ache

6 = headaches

7 = Fatigue

8 = other, specify: \_\_\_\_\_

\_\_\_\_\_  
\_\_\_\_\_

**General Mood Rating:**

General mood/feelings experienced throughout the day can be recorded in the evening by grading it as follows:

- Gr. 1 = Good mood
- Gr. 2 = Feeling sad or down at times
- Gr. 3 = Feeling sad or down most of the time
- Gr. 4 = Feeling sad and down all of the time

**RECORDED AFTER TRAINING**

\*On completion of each training session record the following data:

**Training details:**

All daily training details should be recorded for each discipline; this includes training distances (km) and durations (hh:mm:ss) as well as the average and maximum heart rates ( $\text{bt}\cdot\text{min}^{-1}$ ) for the session (divide brick sessions into appropriate disciplines).

**Perceived Training Effort:**

The perceived effort exerted throughout the training session should be graded and recorded as follows:

- Gr. 1 = Felt good throughout the entire session
- Gr. 2 = Felt good at times during the session
- Gr. 3 = Felt tired, somewhat difficult session
- Gr. 4 = Difficulty completing the session, very difficult

**Muscle Discomfort:**

Record any discomfort, stiffness and/or pain, experienced due to training sessions. Discomfort should be graded accordingly to the following:

- Gr. 1 = No stiffness, no pain
- Gr. 2 = Mild stiffness, mild pain – session unaffected
- Gr. 3 = Moderate stiffness, moderate pain – difficulty completing session
- Gr. 4 = Severe stiffness and pain – unable to complete session

## **2. POMS – PROFILE OF MOOD STATES QUESTIONNAIRE**

### **RECORDED EVERY SUNDAY**

\*Every Sunday of the month and throughout the duration of the study it is necessary for you to complete the POMS questionnaire

The POMS questionnaire was developed to determine the mood states of individuals and has been extensively used in sports research investigating suboptimal training techniques. This questionnaire is to be completed on a weekly basis, every Sunday at any time of the day.

The questionnaire (found on **Sheet 2 (POMS)**) has a list of 65 words that describe feelings that you may have had, each word/feeling needs to be rated from 'not at all', 'a little', 'moderate', 'quite a bit' through to 'extremely'. This relates to how you have been feeling over the past week, including the day of the rating. These 'feelings' are not only what you are experiencing during training sessions but also throughout the duration of the week. A change in mood during endurance training is not uncommon and a partner or spouse can be helpful when filling out this questionnaire. Once again it is important to be as accurate and honest as possible when rating your feelings to aid in producing reliable and valid results.

Completing the questionnaire takes approximately 5 minutes and involves filling in 65 rated feelings

### **3. SUBMAXIMAL 8KM TIME-TRIAL (RUNNING)**

#### **RECORDED ONCE EVERY MONTH**

\*Due to differences in individual training programs this time-trial can be performed when it suits you best, however it must be performed during the **first week of every month.**

Once monthly (during the first week of the month), you are required to perform an 8km running time-trial. The time trial is run at a submaximal, aerobic threshold pace which is determined by using the Maffetone Approach. This approach, also known as the '180 minus' formula is a guideline for athletes to maintain their training at an aerobic level. The formula will be used to ensure that all time-trials are performed at the same/similar intensity, throughout the training period. The submaximal time-trial is useful as there is minimal injury risk in comparison to that of a maximal time trial. The formula must be calculated each month before you proceed with your run; this is due to changes that may occur within the formula over the duration of the study.

For the purpose of the time-trial it is important to find a measured route (most accurately measured on your bike), which is as flat as possible. This exact route will be used for the duration of the testing to standardize results for comparison.

The Maffetone '180 minus' Heart rate can be calculated by:

Take 180 and subtract the following

- your age (in years)
- 10 if you are in recovery from a major illness or injury, or if you are on regular medication
- 5 if you have not been exercising regularly, or have not been exercising due to injury, illness or lack of interest
- 0 if you have been exercising regularly without any interruption

Finally if you have been exercising on a regular basis, without interruption, for approximately two years, and had a progressive increase in performance, then add 5.

E.g. if you are 35 years old and have been exercising regularly for over two years with an increased performance then:

$$180 - 35 + 5 = 150$$

Therefore heart rate should be maintained at  $150 \text{ bt} \cdot \text{min}^{-1}$  for that time-trial.

Results, including average and maximum heart rates, as well as time taken for the time-trial should be recorded on the TT data sheet (page 13 **Sheet 3 (TT)**).

#### 4. SALIVARY HORMONE MEASUREMENT

The measurement of salivary hormones has been found to be a useful indicator of physical, mental and emotional stress. During your training for the Ironman, you place your body under a great deal of stress, this stress, in appropriate quantity, is necessary for enhancing performance although in excessive quantities, is detrimental to your training and health.

Saliva sampling is a simple and risk free method of analyzing hormones, which involves the collection of 2ml of your saliva. The study will be using two salivary hormones, cortisol and alpha-amylase; which have been found to be the 'perfect couple' in hormonal analyses. These hormones will be sampled at 10 points during the course of the study. These sampling dates are illustrated in the table below:

October	December	January	February	March	April
T1 - Baseline Assessment during reduced training <b>31st Oct</b>	<b>Training for Ironman</b>			<b>Ironman</b>	
	Heavy Training			Taper	Event
	T2 - 1st December			T5 - 2nd March	T8 - Immediately after Ironman (medical tent)
	T3 - 5th January			T6 - 1st April	T9 - 1.5h post finish
	T4 - 2nd February			T7 - 4th April	

The samples are numbered T1-T10, and are collected as close as possible to the following times during the study:

- T1.** 31<sup>ST</sup> October– reference measurement; please try incorporating a week of reduced training, into your program, for the seven days prior to this measurement. If this is not possible then continue with the measurement on the date provided
- T2.** 1<sup>st</sup> of December – heavy training measurement
- T3.** 5<sup>th</sup> of January – heavy training measurement
- T4.** 2<sup>nd</sup> of February – heavy training measurement
- T5.** 2<sup>nd</sup> of March – pre-taper period measurement, approximately one month prior to the event a taper period should begin. This period is relative to individual athletes and therefore your taper may begin at a different time, please continue with the sampling on the date provided
- T6.** 1<sup>st</sup> of April – post-taper measurement
- T7.** 4<sup>th</sup> of April – pre-race measurement, this measurement will be taken on the Saturday prior to the event

**T8.** 5<sup>th</sup> April - Immediately after the finish in the medical/finish tent saliva samples will be taken, Rhodes University officials will aid in this sampling

**T9.** 1h30min after your respective finishing time, samples will be required; these samples can be performed by yourself at home/in your hotel and can be dropped off at a designated venue the following day

### **Method of sampling**

It is imperative that when the sampling is performed, it is done correctly and accurately, this is necessary in order to provide reliable results. Due to the nature of the study, and the fact that the sampling method relies on you rather than a Doctor/Sports Scientist, it is very important that you thoroughly read the next section regarding the method of sampling.

Contained within your sampling pack you should find ## cut drinking straws, and ## 2ml Cryovials marked with your initials and your personal reference number. These cryovials are also marked with the date at which the relevant sample should be collected, e.g.

<b>A.J. 001</b> dd/mm/yyyy
-------------------------------

-Cryovial for Alex Joiner (A.J.), personal reference number (001), for the sample taken on the dd/mm/yyyy.

**\*Please be careful when handling these cryovials as they are not easily attainable.**

**\*PLEASE DO NOT EAT, DRINK OR BRUSH YOUR TEETH PRIOR TO THE DROOL**

**!! N.B.!!**The instructions on how to perform the collection of your saliva sample are as follows:

On awakening, and after your waking heart rate measurement has been taken;

- (a) Rinse your mouth out with water,
- (b) **Wait 30 minutes** from the time that you wake,
- (c) Continue as follows:

1. Imagine eating your favorite food, and allow saliva to pool in your mouth
2. Tilt your head forward and drool down a cut straw and into a cryogenic vial (2ml)
3. Repeat this process as many times as necessary until a sufficient sample is collected, 1m (no less), excluding foam should be adequate
4. If your mouth is dry, chew on the end of the straw to stimulate saliva production
5. Try keeping samples cold after collection (4°C)

**\*These samples are susceptible to changing environmental conditions, especially temperature, once collected they begin to perish and are of no**

**use for analysis. Therefore, it is important that they are taken (on ice, but not frozen) to your respective collection point as soon as possible (On the day of, or one day prior to the sample date)**

**There are a number of things that should be avoided prior to sampling:**

1. Brushing teeth within 1 hour prior to collection.
2. Using salivary stimulants, which includes chewing gum, lemon drops, granulated sugar, or drink crystals.
3. Consuming a major meal within 1 hour prior to collection.
4. Consuming alcohol 12 hours prior to collection.
5. Consuming acidic or high sugar foods within 20 minutes prior to collection.

**\*If, for any reason, these factors could not be avoided, then please continue with sampling at the next possible opportunity (preferably the following morning) when you are able to avoid them.**

**\*The timing (30 minutes after waking up) that the samples are taken is imperative to the results due to the variation of your hormonal cycle! Please make sure that you are as vigilant as possible with this testing!**

The data sheet provided (page 14 **Sheet 4 (Hormone Sampling)**) should be used to record all additional information pertaining to the collection of the salivary samples. This information should include prescription and over the counter medication that you are taking, the exact time that your sample was taken as well as any information, which you feel, may have had an impact on the samples.

Day/Date		AM (On Awakening)				AM-PM (Throughout the Day)			During Training (Record after training session)																
		Waking Heart Rate	Hours of Sleep	Sleep Rating	Body Mass	Postural Dizziness	Symptoms of Illness	General Mood Rating	Swim				Bike				Run								
									Km/Duration	HR ave / max	Perceived Training Effort	Muscle Discomfort	Km/Duration	HR ave / max	Perceived Training Effort	Muscle Discomfort	Km/Duration	HR ave / max	Perceived Training Effort	Muscle Discomfort					
Mon								km/A		km/A		km/A		km/A		km/A		km/A		km/A		km/A		km/A	
Tue								M		M		M		M		M		M		M		M		M	
Wed								km/A		km/A		km/A		km/A		km/A		km/A		km/A		km/A		km/A	
Thur								M		M		M		M		M		M		M		M		M	
Fri								km/A		km/A		km/A		km/A		km/A		km/A		km/A		km/A		km/A	
Sat								M		M		M		M		M		M		M		M		M	
Sun								km/A		km/A		km/A		km/A		km/A		km/A		km/A		km/A		km/A	
Ave								M		M		M		M		M		M		M		M		M	

**Sleep Rating:**

- Gr. 1 = Uninterrupted sleep, refreshed
- Gr. 2 = Interrupted sleep, however do not require more sleep
- Gr. 3 = Interrupted sleep, do require more sleep
- Gr. 4 = Interrupted sleep, feeling terrible

**Postural Dizziness:**

- Gr. 1 = No dizziness
- Gr. 2 = Slight dizziness, disappears within 2s
- Gr. 3 = Marked dizziness, time taken to orientate one's self
- Gr. 4 = Marked dizziness, requiring sitting or lying down again

**Symptoms of Illness:**

- 1 = sore throat
- 2 = cough
- 3 = nausea and/or vomiting
- 4 = diarrhoea
- 5 = stomach ache
- 6 = headaches
- 7 = Fatigue
- 8 = other, specify:

**General Mood Rating:**

- Gr. 1 = Good mood
- Gr. 2 = Feeling sad or down only at times
- Gr. 3 = Feeling sad or down most of the time
- Gr. 4 = Feeling sad and down all of the time

**Perceived Training Effort:**

- Gr. 1 = Felt good throughout the entire session
- Gr. 2 = Felt good at times during the session
- Gr. 3 = Felt tired, somewhat difficult session
- Gr. 4 = Difficulty completing the session, very difficult

**Muscle Discomfort:**

- Gr. 1 = No stiffness, no pain
- Gr. 2 = Mild stiffness, mild pain – session unaffected
- Gr. 3 = Moderate stiffness, moderate pain - difficulty completing session
- Gr. 4 = Severe stiffness and pain – unable to complete session

# POMS™ Standard Form

BY DOUGLAS M. McNAIR, Ph.D., MAURICE LORR, Ph.D., JW P. HEUCHERT, Ph.D., & LEO F. DROPPLEMAN, Ph.D.

Client ID: \_\_\_\_\_ Age: \_\_\_\_\_ Gender: Male Female  
 (Circle one)

Birth Date: \_\_\_\_/\_\_\_\_/\_\_\_\_ Today's Date: \_\_\_\_/\_\_\_\_/\_\_\_\_  
Month Day Year Month Day Year

**To the Administrator:**

Place a checkmark ✓  
 in one box to specify the  
 time period of interest.

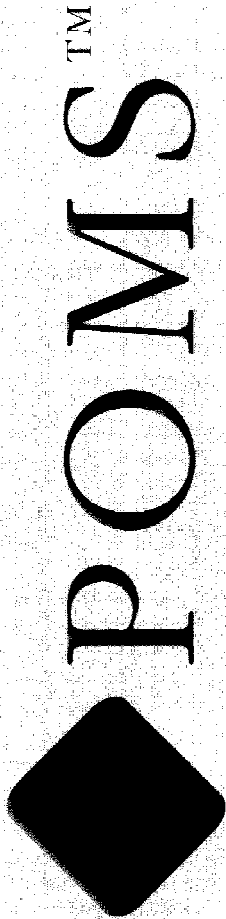


**To the Respondent:**

Below is a list of words that describe feelings that people have. Please read each word carefully. Then circle the number that best describes

- how you have been feeling during the PAST WEEK, INCLUDING TODAY.
- how you feel RIGHT NOW.
- other: \_\_\_\_\_

If no box is marked, please follow the instructions for the first box.



	Not at all	A little	Moderately	Quite a bit	Extremely
1. Friendly	0	1	2	3	4
2. Tense	0	1	2	3	4
3. Angry	0	1	2	3	4
4. Worn out	0	1	2	3	4
5. Unhappy	0	1	2	3	4
6. Clear-headed	0	1	2	3	4
7. Lively	0	1	2	3	4
8. Confused	0	1	2	3	4
9. Sorry for things done	0	1	2	3	4
10. Shaky	0	1	2	3	4
11. Listless	0	1	2	3	4
12. Peeved	0	1	2	3	4
13. Considerate	0	1	2	3	4
14. Sad	0	1	2	3	4
15. Active	0	1	2	3	4
16. On edge	0	1	2	3	4
17. Grouchy	0	1	2	3	4
18. Blue	0	1	2	3	4
19. Energetic	0	1	2	3	4
20. Panicky	0	1	2	3	4
21. Hopeless	0	1	2	3	4
22. Relaxed	0	1	2	3	4
23. Unworthy	0	1	2	3	4
24. Spiteful	0	1	2	3	4
25. Sympathetic	0	1	2	3	4
26. Uneasy	0	1	2	3	4
27. Restless	0	1	2	3	4
28. Unable to concentrate	0	1	2	3	4
29. Fatigued	0	1	2	3	4
30. Helpful	0	1	2	3	4

*Please flip over.  
 Items continue on the back page...*

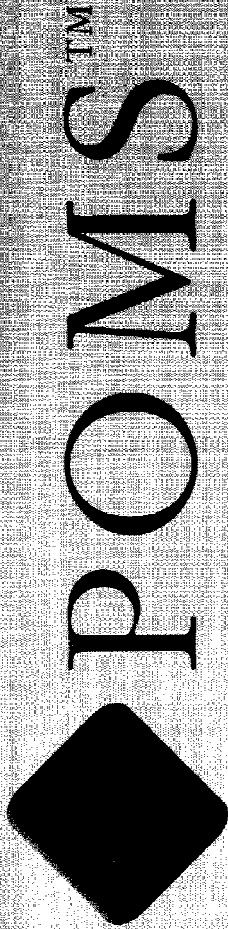


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Printed in Canada.

# POMS™ Standard Form

BY DOUGLAS M. McNAIR, Ph.D., MAURICE LORR, Ph.D., JW P. HEUCHERT, Ph.D., & LEO F. DROPPLEMAN, Ph.D.



	Not at all	A little	Moderately	Quite a bit	Extremely
31. Annoyed .....	0	1	2	3	4
32. Discouraged .....	0	1	2	3	4
33. Resentful .....	0	1	2	3	4
34. Nervous .....	0	1	2	3	4
35. Lonely .....	0	1	2	3	4
36. Miserable .....	0	1	2	3	4
37. Muddled .....	0	1	2	3	4
38. Cheerful .....	0	1	2	3	4
39. Bitter .....	0	1	2	3	4
40. Exhausted .....	0	1	2	3	4
41. Anxious .....	0	1	2	3	4
42. Ready to fight .....	0	1	2	3	4
43. Good natured .....	0	1	2	3	4
44. Gloomy .....	0	1	2	3	4
45. Desperate .....	0	1	2	3	4
46. Sluggish .....	0	1	2	3	4
47. Rebellious .....	0	1	2	3	4
48. Helpless .....	0	1	2	3	4
49. Weary .....	0	1	2	3	4
50. Bewildered .....	0	1	2	3	4
51. Alert .....	0	1	2	3	4
52. Deceived .....	0	1	2	3	4
53. Furious .....	0	1	2	3	4
54. Efficient .....	0	1	2	3	4
55. Trusting .....	0	1	2	3	4
56. Full of pep .....	0	1	2	3	4
57. Bad-tempered .....	0	1	2	3	4
58. Worthless .....	0	1	2	3	4
59. Forgetful .....	0	1	2	3	4
60. Carefree .....	0	1	2	3	4
61. Terrified .....	0	1	2	3	4
62. Guilty .....	0	1	2	3	4
63. Vigorous .....	0	1	2	3	4
64. Uncertain about things .....	0	1	2	3	4
65. Bushed .....	0	1	2	3	4

*Please ensure you have answered every item.  
Thank you for completing this questionnaire.*



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Printed in Canada

### **8 KM TT RESULTS TABLE**

**\*This monthly results table can be seen on Sheet 3 of the electronic data file (TT)**

\*Please record the results from your monthly TT below

<b>8Km TT Results Table</b>			
<b>Date (dd/mm/yy)</b>	<b>Time (hh:mm:ss)</b>	<b>Ave HR (Bt.min<sup>-1</sup>)</b>	<b>Max HR (Bt.min<sup>-1</sup>)</b>

**COMMENTS:**

**\* Please do not forget to follow the Maffetone formula for calculation of your submaximal heart rate!!**

## SALIVARY SAMPLING DATA SHEET

**\*Please record the relevant information pertaining to your sampling in the table provided below. Found on Sheet 4 of the electronic data file (Hormone Sampling)**

Please record the date and the time of sampling

Date (dd/mm/yy)	Time (hh:mm)	Medication (if any)

<b>Comments:</b>	
------------------	--

\* Just a reminder about the sampling days to come!!!

October	December	January	February	March	April	
<b>T1 - Baseline Assessment during reduced training</b> <b>31st Oct</b>	<b>Training for Ironman</b>			<b>Ironman</b>		
	<b>Heavy Training</b>			<b>Taper</b>	<b>Event</b>	
	<b>T2 - 1st December</b>	<b>T3 - 5th January</b>	<b>T4 - 2nd February</b>	<b>T5 - 2nd March</b>	<b>T6 - 1st April</b>	<b>T7 - 4th April</b>
				<b>T8 - Immediately after Ironman (medical tent)</b>	<b>T9 - 1.5h post finish</b>	

Please comment on anything that you feel may have had an impact on your hormonal results.

## APPENDIX D

### Data Collection sheet for control subjects

Day/Date		AM (On Awakening)				AM-PM (Throughout the Day)		
		Waking Heart Rate	Hours of Sleep	Sleep Rating	Body Mass	Postural Dizziness	Symptoms of Illness	General Mood Rating
Mon	30 Mar							
Tues	31 Mar							
Wed	1 Apr							
Thurs	2 Apr							
Fri	3 Apr							
Sat	4 Apr							
Sun	5 Apr							
Ave								

**TRAINING LOG GUIDELINES**

<p><b>Sleep Rating:</b></p> <p><b>Gr. 1</b> = Uninterrupted sleep, refreshed</p> <p><b>Gr. 2</b> = Interrupted sleep, however <u>do not</u> require more sleep</p> <p><b>Gr. 3</b> = Interrupted sleep, <u>do</u> require more sleep</p> <p><b>Gr. 4</b> = Interrupted sleep, feeling terrible</p> <p><b>Postural Dizziness:</b></p> <p><b>Gr. 1</b> = No dizziness</p> <p><b>Gr. 2</b> = Slight dizziness, disappears within 2s</p> <p><b>Gr. 3</b> = Marked dizziness, time taken to orientate one's self</p> <p><b>Gr. 4</b> = Marked dizziness, requiring sitting or lying down again</p>	<p><b>Symptoms of Illness:</b></p> <p><b>1</b> = sore throat</p> <p><b>2</b> = cough</p> <p><b>3</b> = nausea and/or vomiting</p> <p><b>4</b> = diarrhoea</p> <p><b>5</b> = stomach ache</p> <p><b>6</b> = headaches</p> <p><b>7</b> = Fatigue</p> <p><b>8</b> = other, specify:</p> <p><b>General Mood Rating:</b></p> <p><b>Gr. 1</b> = Good mood</p> <p><b>Gr. 2</b> = Feeling sad or down only at times</p> <p><b>Gr. 3</b> = Feeling sad or down most of the time</p> <p><b>Gr. 4</b> = Feeling sad and down all of the time</p>
---	---

## APPENDIX E



**RHODES UNIVERSITY**

*Grabamstown • 6140 • South Africa*

HUMAN KINETICS & ERGONOMICS

Tel: (046) 603 8468 • Fax: (046) 603 8934 • Cell: 072 116 3939 • e-mail:  
[m.goebel@ru.ac.za](mailto:m.goebel@ru.ac.za) (H.O.D.) [bokkiejoiner@gmail.com](mailto:bokkiejoiner@gmail.com) (Alex Joiner)

### CONSENT FORM

I, \_\_\_\_\_, do hereby consent to participate in the study entitled:

**“Time course of performance changes and fatigue markers during training for the Ironman triathlon”**

I agree that I have been fully informed, both verbally and in writing, of the procedures involved in this study. I have also been made aware of any potential risks associated with the project.

I realize that whilst my anonymity will be protected at all times, my results may be published or used for scientific and statistical purposes. I understand the conditions with which I am expected to comply for the duration of the testing procedure, and any queries I have with regards to this have been answered to my satisfaction.

By voluntarily consenting to participate in this research I accept joint responsibility together with the Rhodes University Human Kinetics and Ergonomics Department, whereby should any injury, caused by the project itself, be sustained the department will cover any fees incurred and take steps to rehabilitate the injury. I do however waive any legal recourse against the researcher, or against Rhodes University, and will take full responsibility in the event the injury is shown to be self inflicted. I will inform the researcher

immediately if at any point I experience distress or abnormality, and am fully aware that I may withdraw from participation in this study at any time.

I have read and understood the above information, as well as the information provided by the researcher. Signed at:

\_\_\_\_\_ on \_\_\_ / \_\_\_ / 2008/9.

**SUBJECT:** \_\_\_\_\_ (NAME) \_\_\_\_\_ (SIGN)

**WITNESS:** \_\_\_\_\_ (NAME) \_\_\_\_\_ (SIGN)

**RESEARCHER:** \_\_\_\_\_ (NAME) \_\_\_\_\_ (SIGN)

**APPENDIX F  
STATISTICAL TABLES**

**Repeated measures analysis of variance (ANOVA) of the responses of the athlete group during training and competition for the Ironman competition**

Measure	Variance Analysis Source	SS	DF	MS	F	P
Waking Heart Rate (bt.min <sup>-1</sup> )	Between:	213.6	23	9.3	1.94	0.007388
	Within:	1211.3	253	4.8		
Mass (kg)	Between:	53.1	5	10.6	6.8321	0.000051
	Within:	85.5	55	1.6		
Postural Dizziness (1-4 Rating)	Between:	4.4431	23	0.1932	1.4930	0.072548
	Within:	32.7350	253	0.1294		
Sleep Quantity	Between:	14.76	23	0.64	2.153	0.002209
	Within:	75.44	253	0.30		
Training Load (Swim)	Between:	2727529	21	129882	2.74884	0.000113
	Within:	10914709	231	47250		
Training Load (Bike)	Between:	71255351	22	3238880	5.56669	0.000000
	Within:	140803472	242	581833		
Training Load (Run)	Between:	22663836	23	985384	4.8039	0.000000
	Within:	51895572	253	205121		
POMS TMD (Weekly)	Between:	6425.7	23	279.4	0.90245	0.595619
	Within:	121044.8	391	309.6		
Time-Trial Performance (min)	Between:	45.6	4	11.4	2.0472	0.104180
	Within:	244.8	44	5.6		
Alpha Amylase (U.mL <sup>-1</sup> )	Between:	10038.0	8	1254.7	1.64661	0.123149
	Within:	67057.8	88	762.0		
Cortisol (µg.DL <sup>-1</sup> )	Between:	0.035137	8	0.004392	0.46473	0.877763
	Within:	0.831686	88	0.009451		

**One way ANOVAs were performed to determine similar results during control data collection**

Measure	Effect	SS	Degr. Of freedom	MS	F	p
General Mood	Intercept	72.20000	1	72.20000	780.9438	0.000000
	Sample	0.26395	2	0.13197	1.4275	0.251315
	Error	3.88299	42	0.09245		

Note: Significant differences found over the duration of the study were discussed in the Chapter V.