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THE EFFECT OF PARTIAL SLEEP DEPRIVATION ON SUBSEQUENT AEROBIC EXERCISE PERFORMANCE

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A dissertation submitted to the Faculty of Science, University of the Witwatersrand, Johannesburg, in fulfilment of the requirements of the degree of Master of Science

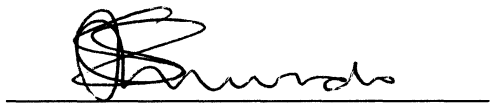
Johannesburg, 2013

DECLARATION

This dissertation is submitted in a format approved by the Faculty of Science, of unpublished research work.

I declare that the work contained in this dissertation is my own, unless otherwise acknowledged.

The work contained herein has not been submitted before for any degree or examination at this or any other university.



Signed on the __**10th**___ day of ____**October**____, **2013**.

ABSTRACT

This study examined the effects of partial sleep deprivation occurring for one or two nights before an endurance event compared to that of no sleep deprivation. We tested the hypothesis that sleep deprivation the night before an ultra-endurance event had no effect on performance provided no sleep deprivation occurred two nights before the event. Eight subjects with the following physical characteristics (mean ± SD) participated in the study: age 26.0 ± 0.4 years, height 1.76 ± 0.80 m, mass 75.0 ± 13.0 kg, VO_2max 54.0 ± 4.2 $ml.min^{-1}.kg^{-1}$. Each subject had their sleep reduced by an average of 3.8 ± 0.4 hours per night compared to a baseline condition (BL) on two occasions, either for one night (SD1) or for two consecutive nights (SD2). Exercise testing occurred after the required nights of sleep deprivation or following baseline sleep. Exercise performance was determined by measuring the cycling distance covered in each of four 40 minute sessions, every 140-160 minutes over an approximate period of 12 hours. Final blood lactate, mean heart rate, sleepiness, mood and psychomotor activity were also assessed. The Profile of Mood States (POMS) revealed that subjects experienced greater feelings of fatigue ($p = 0.032$) and lower levels of vigour ($p < 0.0008$) during both SD1 and SD2 compared to BL. Percentage of SWS was elevated during SD1 compared to both SD2 and BL ($p < 0.05$). Subjects exercised at a relative work intensity averaging 68.2 ± 15 % VO_2max under baseline conditions, 64.4 ± 16 % VO_2max under SD1 conditions and 64.7 ± 17 % VO_2max under SD2 conditions. These findings suggest that reduced sleep considerably alters

mood states but two nights of sleep loss do not influence the ability to perform exercise when compared to just one night of sleep deprivation before an endurance event.

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LIST OF ABBREVIATIONS

ANOVA	analysis of variance
ATP	adenosine triphosphate
BL	baseline
E	exercise session (1, 2, 3, 4)
EEG	electroencephalogram
EMG	electromyogram
EOG	electro-oculogram
EPOC	excess post-exercise oxygen consumption
F_EO_2	Fraction of expired oxygen
F_IO_2	Fraction of inspired oxygen
GH	growth hormone
MSLT	multiple sleep latency test
NREM	non-rapid eye movement sleep
OSA	obstructive sleep apnoea
POMS	profile of mood states
PRL	prolactin
REM	rapid eye movement sleep
RPE	rate of perceived exertion
ROL	REM onset latency
S	Questionnaires and tasks sessions (0, 1, 2, 3, 4)
SD	standard deviation
SD1	one night sleep deprivation

SD2	two nights sleep deprivation
SOL	sleep onset latency
SRT	simple reaction test
STPD	standard temperature, pressure dry correction factor
SWS	slow wave sleep
TMD	total mood disturbance
TSH	thyroid stimulating hormone or thyrotrophin
TST	total sleep time
V_E	volume expired rate
V_I	volume inspired rate
VO_2	oxygen consumption rate
VO_2max	maximal oxygen consumption rate

CHAPTER 1

LITERATURE REVIEW

1.1 General Introduction

Sleep, in humans, has been described as the behavioural state during which one is normally unaware of and unresponsive to one's surroundings (Bonnet 1995). It is a necessary physiological process whose function is widely studied but remains contentious. Although it is generally understood that sleep is essential for our health and wellbeing, many of us often suffer from a lack of sleep, either intermittently or cumulatively. This lack of adequate sleep has been shown to have a range of negative effects on an individual's reasoning ability, mood state, energy systems and physical performance. What follows herein is a thorough review of literature on exercise and sleep physiology, with a view of understanding the role that adequate sleep plays in ensuring optimal exercise performance.

All mammals sleep, although in some circumstances this sleep may be deferrable for long periods of time. However, sleep in other, less frequently studied species, may simply be unrecognizable, thereby, further perpetuating its misunderstood function (Carskadon and Dement 2005). Brain function restoration and energy conservation are amongst reasons cited to explain the role of sleep (Horne 1992; Carskadon and Dement

2005). However, Zhang (2004) later reported that considering the large random variability in daily mammalian sleep quotas, there is no correlation between brain functionality (learning ability) and amount of restorative sleep. Moreover, the significance of energy saved as a result of reduced metabolic activity during sleep remains unclear when taking into account the effects of body movements and arousals (Zhang 2004). This suggests that the function of sleep, though studied, remains unresolved and affords opportunity for further extensive research.

To further examine the function of sleep, sleep deprivation has also been used in research. Despite the focus of research on prolonged sleep loss, shorter bouts of sleep loss are a more common problem in daily reality. For instance, sleep deprivation resulting from disruption or restriction is commonly experienced by normal subjects engaged in rotating shift work, in conditions such as jet lag and by athletes involved in prolonged exercise. Although there is growing evidence and understanding of the complex function of sleep, the impact of its quality and quantity amongst endurance athletes remains elusive (Leeder *et al.* 2012). Anecdotes from athletes suggesting that adequate sleep prior to an event is essential for optimum endurance athletic performance can be credited to the herein described role of sleep in restoring brain function and conserving energy. However, there seems to be inconsistency among athletes as to when and how much they can be sleep deprived before an important event as this is individualistic. This is important when considering the time at which any

particular event commences. Whereas some athletes may display decreased performance, others are much less affected, and a few claim to perform better when they are sleep deprived. The basal sleep needs of athletes, as well as the impact of short naps and other ways of improving the quality of sleep, are still largely contested (Reilly and Edwards 2007). Nevertheless, it is clear that compromising the quality and duration of sleep can cause detrimental effects on one's physical performance.

Of particular importance to sleep prior to an important event, however, is the amount of time to be spent on the athletic activity. Previous studies have employed acute exercise measurements, extending only up to 1 hour in total, to determine both immediate and lasting effects of sleep loss (Koslowsky and Babkoff 1992). The failure of many of these studies partly rests upon the non-exhaustive and ineffective exercise assessments used to determine the effects of sleep loss on performance. Thus far, the effects of partial sleep deprivation on prolonged exercise, usually taking place throughout the day, have not been reported.

Considering the frequent occurrence of reduced sleep bouts amongst the general public (Reid 2002), one would expect extensive research into the impact of this kind of sleep loss on exercise performance. Yet, the effects of sleep loss on subsequent physical performance have been limited to perceived exertion rather than any significant physiological measurements. It is important to note that the effects of sleep deprivation

vary for anaerobic versus aerobic exercise performance. We tested the hypothesis gathered from anecdotal evidence supplied by competitive athletes that sleep deprivation the night before an event does not affect performance, provided no sleep deprivation occurs two nights before. This study investigated the effects of partial sleep deprivation on a subsequent prolonged intermittent exercise protocol.

1.2 Normal exercise physiology

Exercise is a normal physiological process that requires enhanced muscular activity. It is also associated with increases in the rate and depth of breathing to improve lung ventilation, as well as increases in heart rate and stroke volume to improve cardiac output. Exercise is generally grouped into two types depending on the energy supplying process. High intensity, short duration exercise is called anaerobic exercise because energy is derived from non-oxidative sources. Light to moderate intensity, long endurance exercise is normally referred to as aerobic exercise because the energy for the exercise depends on the body's ability to supply oxygen to the body's oxidative energy-generating processes (oxidative phosphorylation). This type of exercise involves large muscle groups for extended periods of time. Thus, the ability to perform exercise as well as the physiological responses to exercise is dependent on the type and severity of the work that is performed (Horne and Pettitt 1984). It is also determined by the fitness of the individual and gender differences

exist (Raven and Potts 1998). Apart from the muscles and cardiopulmonary factors, many other body systems influence or are influenced by exercise and may limit the extent of the exercise possible. These systems are discussed below.

1.2.1 Cardio-respiratory system

Several cardio-respiratory adjustments, including changes in the cardiac pump and oxygen delivery to the lung capillaries ensure that oxygen is adequately transported to the muscles during physical activity. The rate at which the muscles use oxygen while performing endurance exercise is determined by the rate of oxidative phosphorylation which is supported by the rate of blood flow (cardiac output) to the muscles and the amount of oxygen extracted per unit volume of circulating arterial blood (Hill 1994). At the onset of endurance exercise, increased sympathetic nervous system activity and the release of catecholamines results in the elevation of both heart rate (via the sino-atrial node) and stroke volume (via raised contractility) and ultimately leads to an increased cardiac output (Raven and Potts 1998).

Heart rate is determined by the number of ventricular contractions occurring in one minute. At rest, the activity of the parasympathetic and sympathetic nervous systems maintain a heart rate of approximately 70 beats.min^{-1} in normal fit human beings. During exercise, heart rate varies

in relation to the given workload and oxygen uptake (Martin and Haney 1982), and can reach over 200 beats.min^{-1} at maximum oxygen uptake. Stroke volume is the difference between the end-diastolic volume at the beginning of each heart beat and the end-systolic volume. With exercise, this volume can exceed 100 ml.beat^{-1} from a resting level of about 70 ml.beat^{-1}, thereby, also contributing to the increased cardiac output occurring during exercise.

A greater extraction of oxygen from arterial blood by the muscles during endurance exercise occurs because of increased diffusion rate resulting from an elevated membrane surface area, an increased partial pressure gradient and a reduction in the distance for oxygen to diffuse to the mitochondria (Raven and Potts1998). The rate of oxygen usage is usually termed the oxygen consumption/uptake and in the laboratory, is determined using indirect calorimetry, where oxygen uptake (VO_2) is equal to pulmonary minute ventilation (V_E) multiplied by the difference between the inspired (F_IO_2) and expired (F_EO_2) oxygen fractions at the mouth.

$$\text{i.e.} \quad VO_2 = V_E (F_IO_2 - F_EO_2)$$

Compared to the resting VO_2 of approximately 0.25 l.min^{-1}, the average maximum oxygen consumption (VO_2max) of a normal 70 kg man is about 3 l.min^{-1} (43 ml.min^{-1}.kg^{-1}). This maximum consumption is determined by measuring VO_2 during graded increases in exercise load. In most

instances a cycle ergometer or treadmill is used as the exercise mode and VO_2max is reached when additional workload produces no further increases in the VO_2 (Bruce 1963). Although its relevance has been questioned (Speedy 1999), the majority of exercise scientists use VO_2max as a measure of endurance exercise capacity. A person's VO_2max is genetically determined but it can be influenced by age, body mass, gender, training and altitude at which exercise is performed. The influence of body mass is standardised by dividing VO_2max by body mass (VO_2max in $ml.min^{-1}.kg^{-1}$). The greater this number, the more the maximal energy that can be produced by an individual per unit body mass and the better that individual's endurance performance, assuming that they are trained and mechanically efficient (Raven and Potts 1998).

1.2.2 Endocrinology and energy systems

Exercise is known to cause the release of a large number of hormones with various effects as shown in various exercise studies. Responses have been described in male and female untrained subjects (Horne and Pettitt 1984), amateur and professional athletes, in the laboratory and in the field. Among others, the catecholamines (CA), growth hormone (GH), cortisol, prolactin (PRL) and thyroid stimulating hormone (TSH) have been widely studied (Luger *et al.* 1992, McMurray and Hackney 2000). It is clear from these studies that the release of these hormones is dependent on training

as well as exercise intensity. Several other hormones are involved in the cellular processes responsible for energy generation.

Most relevant to the current study, an athlete's exercise training responses can be measured by changes in plasma lactate concentrations and heart rate, both of which are also indicators of exercise strain. During power exercises such as sprinting, when the rate of demand for energy is higher than can be provided by oxidative phosphorylation, lactate is produced faster than the ability of the tissues to remove it and lactate concentration begins to accumulate. The concentration of blood lactate is usually 1 to 2 mmol/L at rest, but can exceed 20 mmol/L during intense exertion. Since increased lactate production coincides with acidosis, lactate measurement is an excellent marker for the metabolic condition of the cell and will influence other physiological processes (Sahlin 1998). To this end, the lactate threshold and the corresponding level of exercise are both objective measures of an athlete's functional capacity and cardiovascular status, and will be utilised to measure the strain of the exercise regime employed in this study. All exercise physiological processes are dependent on the time of day at which the exercise is done or the time of day at which the processes are measured.

1.2.3 Circadian rhythms

Circadian rhythms are physical, mental, and behavioural changes that follow a 24-hour cycle, responding primarily to light and darkness in an organism's environment. These rhythms are found in most living things and are produced by natural factors within the body and affected by signals from the environment. Circadian rhythms can change sleep-wake cycles, the disruption of which has consequences for human behaviour and performance (Reilly and Edwards 2007). These rhythms also affect hormone release patterns, body temperature, and other important physiological functions. The physiological determinants of physical performance are also dependent on circadian rhythms (Akerstedt 1979).

The circadian curve in body rectal temperature shows the variability in rectal temperatures over a period of 24 hours. The implications of this rhythm are to be taken into consideration by sports practitioners who perform sub-maximal and maximal exercise as factors that may limit performance (like internal or external temperature) are dependent on time of day effects (Reilly 1998, Reilly and Waterhouse 2009). However, it is as important to consider individual preferences as some sports performances may be less influenced by circadian rhythms compared to others. Endurance exercise, during which one remains active for an extended period of time and builds the ability to resist fatigue, is most affected by circadian patterns. Notable examples of endurance exercise include long distance running, cycling and competitive swimming. During this type of

exercise, an athlete makes use of the aerobic as opposed to the anaerobic system. Often endurance is defined according to an athlete's cardiovascular and muscular strength, as well as the consistency and effectiveness with which he or she performs with the least effort.

Some instances of the influence of circadian rhythms on anaerobic performance have also been studied. For example, reports suggest that during indoor five-a-side games sustained for four days, there is a peak in the pace of play in a soccer match at about 18h00 and a drop between 05h00 and 06h00 (Reilly and Walsh 1981). Similarly, Souissi and colleagues (2004) reported circadian influence on physical performance following an anaerobic force-velocity test on a cycle ergometer, such that a significant time of day effect was observed for oral temperature, peak power, mean power and maximum power on the Wingate test. Thus, the ability of an athlete to perform to full capacity is affected by the time of day at which the exercise is performed. These exercise rhythms are, therefore, important to elite athletes for achieving optimum levels of performance at different times of the day.

Furthermore, advancing or delaying the phase of a good night's sleep by 3 hours differentially impairs subsequent performance. This phenomenon plays an important role in determining the time of night during which an endurance athlete should rather sleep in order to avoid performance deficits on the day of an event. The loss of sleep during the second half of

the night is more frequently observed in the case of endurance events that usually require an early start to the day. Given these factors, sleep in the current study was restricted to the first half of the night to mimic real circumstances.

Performance is also related to arousal levels and some athletes have indicated this as the reason to rather exercise at dawn, and this is perceived as an energising way to start the day. Other athletes report performing at their best in the evenings as the environmental temperature at this time is more favourable. Although performance may be improved in the early evening, the intensity and duration of prior exercise may affect subsequent nocturnal sleep (Bunnell 1982). Anecdotally, some athletes have proposed that following a bout of exercise, provided that it is not too vigorous, the degree of tiredness and sleepiness experienced allows for better sleep. However, if intense exercise continues for days, this may, in turn, lead to reduced sleep over time, the effects of which can be cumulative (Dinges 1997, Van Dongen 2003). Fitness of an individual to perform exercise can also be influenced by that individual's physical condition as well as alertness, as influenced by circadian rhythmicity. This is also determined by the quantity and quality of prior rest. Although usually neglected, sleep can be a determinant of exercise performance, as sleep is inextricably linked with temperature, circadian rhythms, hormonal production and other physiological determinants discussed above. This relationship is explored further below.

1.3. Normal sleep physiology

"Sleep research became vastly diverse and coherence was made possible with the development and validation of a common measurement axis in the early 1920's, namely electroencephalography (EEG)" (Carskadon and Dement 2005). The EEG was then expanded into the overnight polysomnogram with the addition of other measurements, namely, electromyography (EMG) and electrooculography (EOG). Changes that occur in all these measurements distinguish two sleep states from wakefulness; rapid eye movement (REM), which is associated with dreaming, and non-rapid eye movement (NREM) sleep.

Based on changes in EEG patterns (Figure 1.1), NREM sleep has been subdivided into four stages (1 - 4). Stage 1 is a drowsy state of transition when a subject changes from awake to asleep as well as when they move between sleep stages. Stage 1 takes up 2 to 5 % of the total sleep time. Stage 2 sleep occupies 45 to 55 % of the total sleep time and is characterised by K complex and sleep spindle waveforms. The combination of stage 3 and 4 is termed slow wave sleep (SWS). This slow, (high amplitude; low frequency) delta wave activity occupies approximately 13 to 23 % of total sleep time and has a higher arousal threshold (is more difficult to awake from) than other NREM stages. NREM sleep is said to be synchronous, whereas REM sleep is characterised by the presence of rapid eye movements in the EOG, as well as "saw tooth" waves in the EEG and takes up the other 15 to 20 % of the total sleep time.

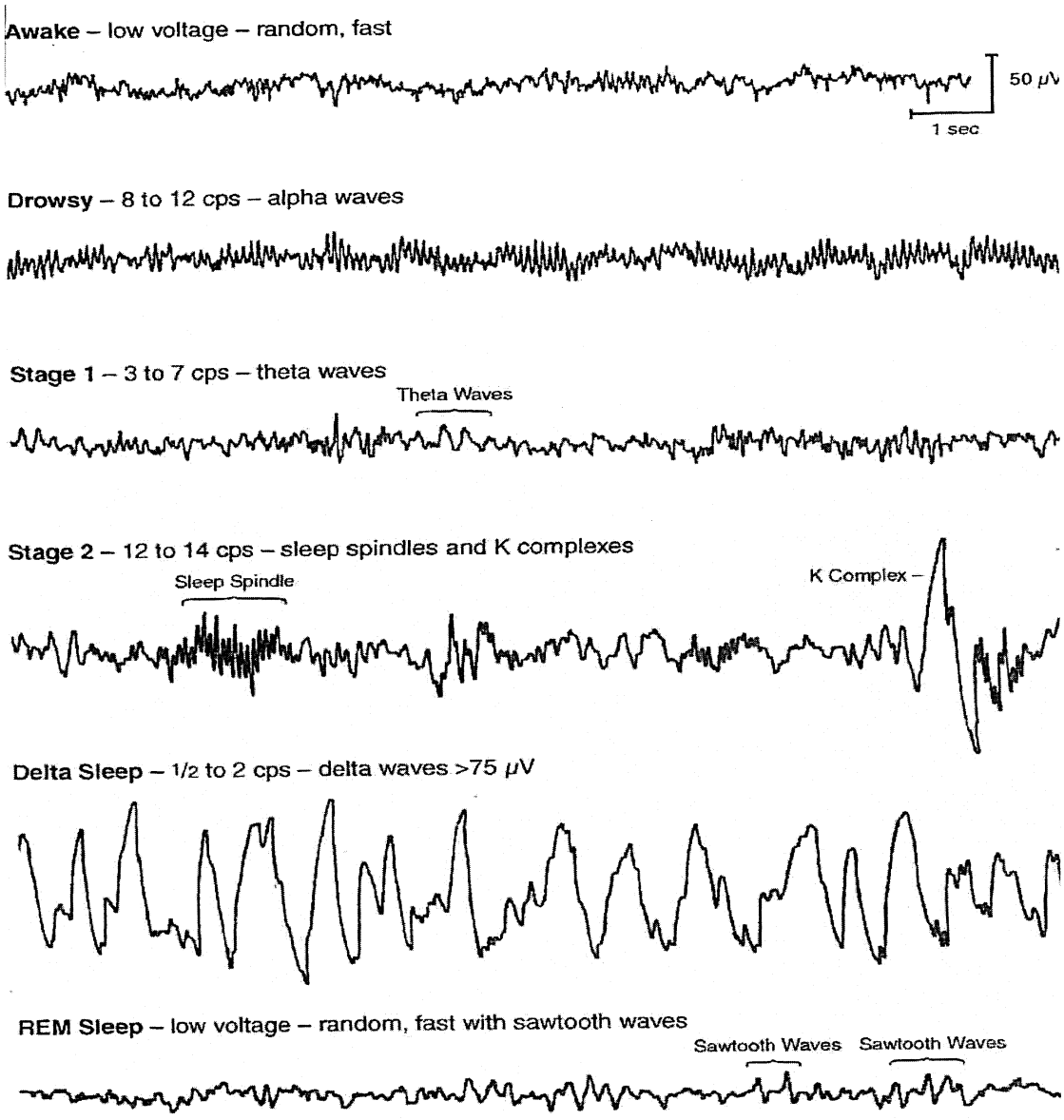


Figure 1.1 Diagram of a typical Electroencephalogram (EEG) depicting wakefulness and sleep stage patterns in humans. Delta waves refer to stage 3 and 4 (also known as slow wave sleep). Wave frequencies and amplitudes are presented as cps (cycles per second) and μV (micro volts) respectively (Best, 2006).

It is speculated that the body and brain restoration functions take place during NREM sleep (Bennington and Heller 1995). Historically, SWS has been found to be the most important component of the restorative function of sleep on metabolic activity (Bonnet 2005). An increase in SWS speeds up metabolic activity, and conversely, decreased levels of SWS accompany decreased metabolism (Shapiro 1981). The function of REM sleep, on the other hand, has been found to be more complex. Its role may include brain development, eye coordination and protein synthesis (Zhang 2004) as suggested by its possible relationship with neuron growth and development (Walker and Stickgold 2004).

The normal young adult, under ideal circumstances, enters an 80 to 90-minute cycle of sleep through NREM sleep, with REM sleep occurring thereafter. Normal human sleep is continuous, with stage 1 having the least arousal threshold and stage 4 the highest (Carskadon and Dement 2005). Table 1.1 shows the standard criteria used in defining the different sleep stages.

Table 1.1 Standardized scoring criteria for defining sleep stages (modified from Rechtschaffe

Sleep Stage	EEG	EOG
Stage W (awake)	Alpha (8-12 cps) activity and/or low voltage, mixed frequency EEG	Rapid eye movements and eye
Stage 1	Low voltage, mixed frequency Theta (3-7 cps) activity; vertex sharp waves	Slow eye movements, no rapid movements
Stage 2	Low voltage, mixed frequency activity, with spindles (12-14 cps), and K complexes (negative sharp wave followed immediately by a positive component)	Occasional slow eye movement
Stage 3	High amplitude, slow waves	None
Stage 4	High amplitude, slow waves	None
REM	Low voltage, mixed frequency, saw-tooth waves	Phasic rapid eye movements
Movement time	Movement in epoch, sleep stage obscured	Obscured

EEG = Electroencephalogram ; EOG = electro-oculogram ; EMG = electromyogram ; REM = rapid eye movement

The duration of a sleep cycle and the respective periods of the two main stages are dependent on factors such as circadian rhythms (daily periodicity in biological processes), genetic (heredity) and preferential determinants (staying up late or waking up early) (Carskadon and Dement 2005). Although individualistic and preferential, the time at which we sleep will generally determine the consequent alertness levels of an individual to perform bodily functions and daily activities (Horne 1992). The lack of sleep, or a shortened sleep period, can have significant effects on performance if sleep loss is significant and occurs for several days without recovery sleep.

1.4 Sleep deprivation

The restriction of sleep, voluntary or otherwise, is prevalent in both normal individuals and patients with sleep disorders (Bonnet 2003). Studies of sleep deprivation have been the topic of extensive research since the late nineteenth century, and in fact, have at times been used to try to understand the function of sleep. If sleep is considered to be a restorative process for daily activity, then the effect of a lack of any stage of sleep could be to the extent that daily activity is affected. The way in which an individual responds to sleep loss is dependent on the quantity, quality and distribution of stages of prior sleep (Endo *et al.* 1998; Sallinen 2004). A recent study has compared the sleep of elite athletes and non-athletic controls using wristwatch actigraphy and found significant differences in the quantity. This is despite proven proportional measures of time spent asleep or in bed, pointing to the

poor efficiency and other characteristics of sleep in athletes (Leeder *et al.* 2012).

Consequences of varying degrees of sleep deprivation can be distinguished. On the basis of the duration of sleep restriction, two types of sleep deprivation have been defined: total sleep deprivation and partial sleep deprivation (Bonnet 2005).Total sleep deprivation is the complete absence of sleep over a period of normal sleep and is a strong stimulus for subsequent sleep. Studies of this kind of sleep deprivation date back to the late 1800's, with particular focus on its physiological, mental, subjective, and later, EEG effects. Partial sleep deprivation is characterised by a short, non-sleep-stage-specific reduction in total sleep. It is usually experienced by rotating shift workers, long distance truck drivers and athletes competing in events after crossing several time zones or those engaged in prolonged exercise such as in the case of ultra-marathon events (VanHelder and Radomski 1989; Akerstedt 2003). Some studies have also distinguished between acute and chronic partial sleep deprivation. The effects of partial sleep deprivation are, therefore, exacerbated by an accumulating sleep deficit over several days, and sometimes as few as 2 nights of sleep loss.

Compared to other variables, physiological changes including cardiovascular and metabolic disruptions are usually minimal, while effects on subjective measurements and cognitive performance variables (alertness, mood and psychomotor activity) show significant changes and have been well studied. The most highly expected results of sleep deprivation of up to 5 days are

decreased alertness, increased subjective sleepiness, mood and cognitive performance. Using a treadmill-walking exercise regime, Angus and colleagues (1985) studied the effects of physical exercise and sleep deprivation, lasting up to 60 hours, on mood and cognitive performance in 12 male subjects. The outcome of self-report scales suggested that subjects experienced high perceived levels of fatigue in both the exercise and no exercise conditions. Conversely, oxygen consumption, respiratory volume and heart rate were unaffected by sleep deprivation in both conditions (Angus 1985). The overall results of this study revealed clear decrements in mood and cognitive performance as a function of sleep loss. In 2002, a further analysis of the interrelated effects of subjective, objective and EEG measures of alertness was performed. The results indicated that fatigue during a night of total sleep deprivation, is a multi-faceted phenomenon. It presents with the feeling of sleepiness, decreased brain glucose utilisation and decreased EEG activity as determined by "awake" sleep recordings (Leproult *et al.* 2002). There are more studies in the literature on the effects of prolonged sleep loss of up to 75 hours on vigilance and cognitive performance than there are on its effects on athletic performance. In reality, athletes would usually experience more moderate levels of sleep deprivation, the consequences of which will, for the purpose of this research, be examined in greater detail.

Although humans require sleep of about 8 hours per night, most young adults have reduced sleep of about 6.5 hours per night (Bonnet 1995), which has been shown to cause increased sleepiness, substantiated by results of questionnaires, sleep logs, multiple sleep latency tests (MSLT), as well as

slow reaction times (Dinges 1997). Beyond acute sleep deprivation lasting for 1 or 2 nights, a more chronic form of sleep loss may occur if this pattern is prolonged for 30 or more consecutive days, ultimately resulting in cumulative negative performance effects (Angus 1985; Takeuchi *et al.* 1985; Symons *et al.* 1988; Goodman *et al.* 1989).

A number of partial sleep deprivation studies (Webb 1975; Goodman *et al.* 1989; Brunner 1993; Pierce *et al.* 1993; Bach 1994; Dinges 1997; Meney *et al.* 1998; Williamson and Feyer 2000; Scott and McNaughton 2004) to date, have reported inconsistent results in terms of the effects of sleep loss, posing more questions than providing answers. While some revealed a weak correlation between subjective and objective measures of sleepiness and fatigue (Devoto 1999, Williamson and Feyer 2000) others agreed that REM sleep is delayed and NREM sleep compensation takes priority during recovery sleep (Brunner 1993, Bach 1994). However, other studies have shown that, in order to detect residual daytime performance or mood deficits, sleep must be reduced to at least 5 hours per night for 2 successive nights (Mougin *et al.* 1996; Dinges 1997).

Due to the nature of the sleep cycle, the duration of the entire sleep period may have an impact on the duration of some sleep stages obtained during one sleep cycle. For instance, sleep restriction of 4 hours or less per night will significantly reduce the amount of REM sleep obtained, thereby imposing a high level of REM deprivation (Tilley 1984). Similarly, one would expect a reduction in the amount of slow wave sleep should a sleep epoch be

shortened like it would in sleep disorders that cause frequent arousals and delayed sleep onset, such as insomnia and obstructive sleep apnoea. The restorative functions of sleep would be compromised as a result of the lack of progression into deeper stages of sleep. It is clear that the consequences of varying durations of sleep loss can also differ. The following section further addresses and compares the effects of both partial and total sleep deprivation.

1.5 Effects of sleep deprivation

The adverse effects of sleep deprivation, regardless of duration, are well documented (Martin 1981; Martin and Gaddis 1981; VanHelder and Radomski 1989; Scott *et al.* 2006) and will be examined further herein. For the purpose of this presentation, these will be divided into sleepiness, endocrine, cardio-respiratory and performance (both physical and cognitive) effects.

1.5.1 Sleepiness

Sleepiness, like hunger or thirst, is a basic physiological need state necessary for the survival of many organisms and is only reversed by sleep. The neural processes that govern the sleep-wake cycle would naturally increase the tendency for one to sleep following a period of wakefulness. The degree of our accumulated sleep debt can be measured by this inclination to fall asleep more rapidly or sleeping longer than usual (Bonnet 1995). Although the sleep needs of individuals vary, sleep loss of as little as 1 or 2 hours from one's

ideal sleep time can exaggerate sleepiness and the tendency to perform errors (Powell *et al.* 2001). Earlier research has previously focused on prolonged sleep deprivation effects, but the effects of partial and total sleep deprivation were later found to be quantitatively similar (Bonnet 2003). The loss of up to 4 hours of sleep per night decreases daytime sleep latencies (Carskadon and Dement 1981; Devoto 1999), increases subjective sleepiness and results in performance deficits (Mougin *et al.* 1991; Meney *et al.* 1998). The most noticeable of sleep loss effects are related to excessive daytime sleepiness. These are usually visible to the examiner's eye or the subject themselves (yawning, eye rubbing or nodding) or sometimes evidenced by objective sleepiness reports such as MSLTs. In a study to evaluate the effects of sleep loss on daytime sleepiness, sleep of 5 hours per night for 7 nights resulted in significantly reduced sleep latency scores on the MSLT by a difference of up to 10 minutes per day across all 7 days (Carskadon and Dement 1981).

Factors influencing daytime sleepiness following a night of sleep deprivation include the time of day of sleep deprivation, duration of prior sleep loss, as well as sleep structure (Dinges 1997; Devoto 1999). Within a conventional sleep and wake cycle, sleepiness naturally occurs in the late evening, with a peak in the middle of the night when an individual is sleeping. Maintaining wakefulness during this time may result in the loss of energy, fatigue, memory and lapses in concentration. Therefore, the maintenance of this wakefulness into the following day will further exacerbate sleepiness. When the effects of slow wave sleep loss on daytime sleepiness were assessed, an increase in

the tendency to fall asleep was related to the duration of sleep restriction (Devoto, 1999). In this study, subjective and objective performance effects were found to have a weak correlation with electrophysiological data. Therefore, persons at risk (drivers, etc) may develop sleepiness without adequate warning signs (Devoto 1999). The structure of prior sleep influences the composition of recovery sleep. An important feature in the regulation of sleep is to compensate for its loss in subsequent recovery sleep. For instance, selective sleep deprivation of either NREM or REM sleep increases the compensatory effects of recovery sleep, such that the restricted sleep stages are likely to be inflated in the recovery period. EEG slow wave activity is a reliable NREM sleep intensity marker that increases in recovery sleep, seen after prolonged waking (Endo *et al.* 1998). There is, however, no consensus on the loss or maintenance of NREM stages when sleep is only partially restricted. Following partial sleep deprivation of 4 hours of sleep on two consecutive nights, the sleep and awake EEG showed no differences in the amount of stage 3 and 4 sleep (Brunner 1993). This report further suggested that the preservation of SWS with sleep deprivation implies that it may have a more essential function.

1.5.2 Cardio-respiratory effects

Historically, studies have reported conflicting results in response to sleep deprivation; either increases or decreases in heart rate, blood pressure or ventilation, but some have historically reported no significant effects of sleep deprivation on these variables (Horne 1978). The effects of prolonged sleep deprivation of 30 hours have been studied both at rest and in exercise in a

group of 15 young male volunteers, using a combination of 1-min incremental exercise tests to exhaustion on a cycle ergometer and exercise endurance tests. This study concluded that 30 hours of sleep loss changes cardiorespiratory function and hampers the ability to perform maximal exercise, but does not affect exercise endurance, as indicated by a reduction in resting heart rate, plasma catecholamine levels and blood pH as well as an increase in minute ventilation and CO_2 production (Chen 1991).

Cardio-respiratory changes are reflective of performance in physical activities and have a significant effect on the ability of humans to perform normal levels of exercise. More studies have examined the extent to which sleep loss actually changes exercise responses (Cooper and Phillips 1982; Angus 1985; Myles 1985; Bond *et al.* 1986; Plyley *et al.* 1987; Canet *et al.* 1989) by examining sleep loss with and without intermittent exercise. They all found a lower heart rate in response to exercise after sleep loss. Later, a similar response was observed when sleep deprivation resulted in a decline in heart rate during sub-maximal exercise (Scott and McNaughton 2004). On the contrary, Spengler and Shea (2000) found that sleep deprivation does not change ventilation and metabolism in humans, provided that the exercise and environment are well-controlled. In addition, a difference was found in the response of normal humans and those with obstructive sleep apnoea (OSA) to sleep loss (Spengler and Shea 2000). These changes, however, were also reported to be reversed following recovery sleep, demonstrating that sleep loss of any magnitude is unlikely to result in human system failure.

A more recent review by Mullington and colleagues (2009), however, has outlined the effects of insufficient sleep on cardiovascular risk factors including blood pressure, glucose metabolism, hormonal regulation and inflammation. Although trivial, autonomic changes in humans during periods of sleep restriction have been demonstrated but it remains unknown whether these changes are short-lived and one is able to adapt over time (Mullington *et al.* 2009).

1.5.3 Endocrine activity

The normal daily variances in hormonal and metabolic indicators during the sleep and wake states are tightly regulated and related to circadian activity (Van Cauter 1997; Spiegel 1999). The concentrations of various hormones in the body at any given time can either be a function of prior levels of hormone, current levels of activity or time of day effects. Several early studies have demonstrated hormonal responses in normal subjects during varying periods of sleep loss. In the early 1990's, Van Cauter and her team (1991) evaluated the effects of a 12-hour sleep loss on plasma levels of GH, cortisol, prolactin (PRL) and thyrotrophin (TSH). This experimental design yielded significant differences in the profiles of all of these hormones. Cortisol showed the least shift in its profile, and TSH secretion was increased in the absence of sleep. On the contrary, later findings revealed elevated thyroid activity following total sleep deprivation thereby inhibiting the amount of TSH released under these conditions (Bonnet 2003). The GH and PRL levels were immediately elevated following this 12-hour period of no sleep (Van Cauter *et al.* 1991). Besides its

sleep onset-induced increase in secretion, GH concentration is generally highest during the late evening and early part of the night (Van Cauter *et al.* 1992). This profile is also indicative of a circadian rhythm regulation (Brandenberger 2000).

Although the total amount of hormone secretion may be consistent, the patterns of secretion may change, such as in the case of hormones which are dependent on sleep for their circadian rhythmicity (e.g. cortisol, GH and TSH) (Carskadon and Dement 2005). Considering the relationship between exercise performance and circadian rhythmicity, it can be expected that time of day effects of sleep loss on the endocrine system should also determine the ideal time at which one should exercise. Previous reports stated that in young adult males, GH release is related to exercise intensity in a linear dose-response manner (Pritzlaff *et al.* 1999). This fractional stimulation of GH secretion was later found to be greater in women than in men (Pritzlaff *et al.* 2002).

Blood and urinary levels of cortisol are physiological markers of stress. Under normal circumstances, cortisol decreases at sleep onset for 1 to 2 hours, increases during nocturnal awakenings, and again increases towards the end of the sleep period. Due to an increase in the amplitude of the secretory pulses at this time, cortisol displays a marked diurnal rhythm being highest in the early morning (around 04:00). Sleep is therefore initiated when activity of the corticotropic axis is limited. This sleep onset inhibition in cortisol secretion is less pronounced under conditions of sleep loss (Leproult 1997).

The control of plasma glucose concentrations is facilitated by the balance between glucose delivery and utilisation by the body. Insulin plays an important role in this regulatory process by inhibiting hepatic glucose production and stimulating glucose uptake by insulin-sensitive tissues like skeletal muscles (Peschke 2008). Conversely, the counter-regulatory hormone, glucagon, has the ability to increase plasma glucose levels by stimulating the breakdown of stored glycogen. It was also established that the characteristics of normal glucose regulation vary across the 24-hour cycle. Increased glucose in response to a standard oral glucose load occurs in the late afternoon and early evening, and actually persists during most of the night. Perhaps a detailed understanding of glucose regulation can provide strategies for dietary schedules that improve this regulation.

1.5.4 Mood, cognitive and psychomotor performance

Cognitive functions in human performance are likely to be easily impaired by a period of sleep loss. Although research has produced very conflicting results over time, several outcomes point to the deleterious effects of a lack of sleep on cognitive performance and vigilance, which can be extrapolated to exercise performance (Horne 1985; Dinges 1997; Scott 2006). These include cumulative and consistent changes in neurobehavioral measures of alertness as a function of sleep restriction, evidenced by results of subjective sleepiness scales, MSLTs and psychomotor vigilance tests. Generally, changes in these variables are greatest during the first 2 days of sleep restriction (Dinges

1997). Traditionally, sleepiness impairs normal functioning of the central nervous system (Horne 1985; 1992), thereby causing negative effects on psychological performance, indicated as cognitive slowing, memory loss, fatigue and decreased vigilance and alertness (Angus 1985; Scott 2006). The performance decrements caused by moderate sleep deprivation have even been equated to those induced by moderate alcohol intoxication in drunken-driving studies (Williamson and Feyer 2000).

Because of the challenges in measuring the physiological effects of sleepiness, researchers are more inclined to measure performance degradation using standardised performance tests. Long, monotonous, paced, newly-learnt psychomotor tasks with a short-term memory component and without incentive or feedback, are most affected by sleep deprivation because they unmask sleepiness (Pilcher and Huffcutt 1996). Vigilance and reaction times, such as in simulated driving tasks and simple reaction tests, are affected after sleep deprivation and are therefore effective measures of psychomotor performance (Dinges 1997; van den Berg and Neely 2006). In partial sleep deprivation studies, the decreased performance on vigilance motor tasks is the most obvious response to reduced sleep (Holland 1968). These effects on vigilance are intensified as one spends more time on the same task. Reaction times deteriorate when sleep is limited to two or fewer hours on two successive nights compared to increases in the subjective complaints of headaches, forgetfulness, decreased concentration, fatigue, irritability, and difficulty waking experienced after longer duration studies (Dinges 1997).

Two reviews of sleep deprivation were published in the 1990s, both reporting the influence of sleep deprivation on psychomotor performance. One analysis concluded that prolonged sleep loss of 45 hours per week or longer had increasingly greater impact on performance and that the speed, rather than accuracy, of performance was more affected (Koslowsky and Babkoff 1992). The other review suggested that mood was more affected by sleep loss than cognition, which, in turn, was more affected than motor tasks (Pilcher and Huffcutt 1996). Although sleep loss has been seen to affect mood states and cognitive ability, the effects on exercise performance may still require more research. A recent study has investigated the changes in psychological states during an ultra-endurance foot race, and found that an individuals' trait emotional intelligence may be a key factor in determining their response to repeated bouts of exercise (Lane and Wilson 2011). This difference means that some athletes have superior performance in hard exercise as compared to others with low trait emotional intelligence.

In an earlier study by Lucas and others (2009), using the Stroop test before, during and after an adventure race revealed that the cognitive performance of 9 athletes was modestly affected. Complex response times were 16% slower and were found to be dependent on the intensity of exercise. Therefore, it was concluded that moderate-intensity exercise may help complex decision-making during sustained stress (Lucas *et al.* 2009). Similar to the more deleterious effects of sleep loss on monotonous cognitive tasks, compared to complex tasks, exercise performance may be less affected by sleep loss if the

bout of exercise is longer and more challenging. It is, therefore, essential to quantify physiological sleep restriction effects separately from psychological effects.

1.5.5 Exercise performance

Research has investigated the effects of sleep restriction extending as long as 64 hours on subsequent exercise performance (Angus 1985; Takeuchi *et al.* 1985; Martin 1988; Symons *et al.* 1988; Goodman *et al.* 1989). For a long time these effects on physical performance were limited to perceived exertion rather than physiological measurements. In a study by Myles (1985), outcomes suggested that, where the exercise is of several minutes duration, rates of perceived exertion (RPE) are increased by sleep deprivation and by physical fatigue. Where the exercise period is as short as 30 seconds, sleep deprivation has no effect and physical fatigue causes only a small change in the perception of exercise intensity (Myles 1985). Further research then revealed that, in fact, acute sub-maximal exercise (at 80% VO_2max) is only affected by prolonged sleep loss if the exercise is exhaustive (Martin and Haney 1982; Bond *et al.* 1986; Goodman *et al.* 1989). These outcomes are based on the degree of contribution of the aerobic energy system to physical performance after sleep loss, by measuring oxygen consumption (VO_2) during a bout of exercise. This is important in determining the duration of the bout of exercise to be used in all sleep loss studies. In support, acute sleep loss, achieved through delaying bedtime until 3 am, was found to alter neither supra-maximal exercise evaluated from the 30-second Wingate test, nor the respiratory and metabolic responses to that test (Mougin *et al.* 1996).

Early efforts to determine cardiovascular, metabolic and respiratory responses to aerobic exercise, following a period of sleep restriction, yielded conflicting results. In a study conducted by McMurray and Brown (1984), exercise ventilation, heart rate and oxygen consumption were all not affected by sleep loss, during the sleep deprivation period. However, during the recovery period, ventilation and oxygen consumption increased (McMurray and Brown 1984). This could have been a compensatory reaction to the compromised oxygen usage during exercise while subjects were sleep deprived. In conflict with this report, following a partial sleep deprivation protocol of 3 hours of sleep loss in the middle of the night, there were statistically significant increases in heart rate and ventilation at sub-maximal exercise compared with results obtained after a baseline night (Mougin *et al.* 1991).

As demonstrated in many studies, the effects of sleep restriction on exercise performance are evidently dependent on three factors: 1) whether the exercise is aerobic or anaerobic; 2) whether the exercise protocol is exhaustive; and 3) which sleep stages are restricted, as a function of the half of the night in which sleep is restricted. In light of the conflicting findings of previous work concerning the effects of sleep restriction on intermittent sub-maximal exercise and cognitive performance, this study was specifically designed to take all three of these factors into consideration in order to yield consequential results.

1.6 Research aims

1. To test the effects of both one and two consecutive nights of partial sleep deprivation on various performance measures .
2. To compare the difference in the subsequent measures of aerobic exercise performance, mood and daytime sleepiness after one and two consecutive nights of partial sleep deprivation.
3. To test the correlation between measures of restricted sleep and aerobic exercise measurements.

CHAPTER 2

METHODS

2.1 Ethics

The study was approved by the Committee for Research on Human Subjects at the University of the Witwatersrand (protocol number M050220). Each subject provided written informed consent.

2.2 Subjects

Eight male volunteer cyclists between the ages of 18 and 30 years participated in the study. They were recruited from competitive cycling and spinning clubs in Gauteng, as well as from the University of Witwatersrand's cycling club, in response to disseminated marketing material and email requests for participation. In addition, marketing material was posted at various places including Little Company of Mary hospital in Pretoria and Wits Medical School in Johannesburg. Participants had all taken part in competitions such as the 94.7 cycle challenge and other known mountain bike and cycle races. Subjects were excluded if they had irregular sleep-wake schedules, and any sleep or medical disorders, as determined by the General Health Questionnaire (Appendix 1) used to screen for physical and physiological distress (Goldberg 1992). Subjects reported sleeping six to eight hours on average with little variation. They were instructed to abstain from caffeine and alcohol for 24 hours prior to each assessment and to maintain a

standard sleeping pattern for at least 48 hours before a scheduled study period.

2.3 Adaptation

At least one week prior to the first test period, subjects spent an adaptation night in the laboratory, during which they became familiar with the recording techniques and new environment. They had a VO_2max measurement, completed the questionnaires and reaction tests to be used on the experimental nights and slept with all electrodes to be used for the sleep recordings. Data was obtained but no values from these measurements were used for analysis purposes.

2.3.1 Maximal oxygen uptake (VO_2max)

A 3-min interval incremental protocol (Wyndham 1959) was used. Following a 10-minute warm-up period, subjects first cycled on a stationary cycle ergometer (Dynavit Meditronic 40, Keiper Dynavit GmbH & Co, Kaiserslautern, Germany) at a work rate of 150 Watts for 10 minutes to measure cycling efficiency, and then pedalled at a work rate close to their maximum ability for 3 minutes depending on their individual capacity. The subjects rested for a period of 10 minutes between each work load. The work load was increased by 20 $W.min^{-1}$ for each subsequent 3-minute exercise period until the VO_2 changed by no more than 1 $ml.kg^{-1}.min^{-1}$. Oxygen

consumption, carbon dioxide production and respiratory minute volumes were measured using a metabolic cart (Oxycon 4, Mijnhardt BV, Bunnik, Holland).

To assess the performance of the metabolic cart prior to the beginning of the study, the Douglas bag validation method was used (Consolazio1963). Before each use, the system was calibrated against a gas syringe to confirm gas volume measurements. Room air (20.93% O_2) and a certified gas mixture (Afrox, Johannesburg, South Africa) of known concentration (16% O_2, 4% CO_2, 80% N_2) were used to calibrate gas concentration assessments. Oxygen consumption (VO_2) and carbon dioxide production (VCO_2) were expressed as standard temperature (273° absolute), standard pressure (760 mm Hg), and dry (0 mm Hg vapour pressure) (STPD).

2.3.2 Questionnaires and tasks

Subjects were asked to complete two questionnaires: (i) Profile of Mood States (POMS) (McNair *et al.*1971) to assess mood. The POMS questionnaire (Appendix 2) is designed to assess the following six mood states: tension, depression, anger, vigour, fatigue, and confusion. It consists of 65 adjectives rated on a five-point scale of 0 to 4, where the numbers refer to these phrases: 0 = not at all, 1 = a little, 2 = moderately, 3 = quite a bit and 4 = extremely. Reliability, internal consistency and factorial validity are well established for the POMS (Weckowics 1978; Norcross *et al.* 1984). (ii) Wits Faces Sleepiness Scale (Maldonado *et al.* 2004) to assess subjective sleepiness. The Wits Faces Sleepiness Scale (Figure 2.1) uses cartoon faces

to measure perceived sleepiness and is validated against the Stanford Sleepiness Scale (SSS) and the Karolinska Sleepiness Scale (KSS) (Maldonado *et al.* 2004). Each face is assigned a number, with 1 representing the most awake face and 5 indicating perceived sleepiness.

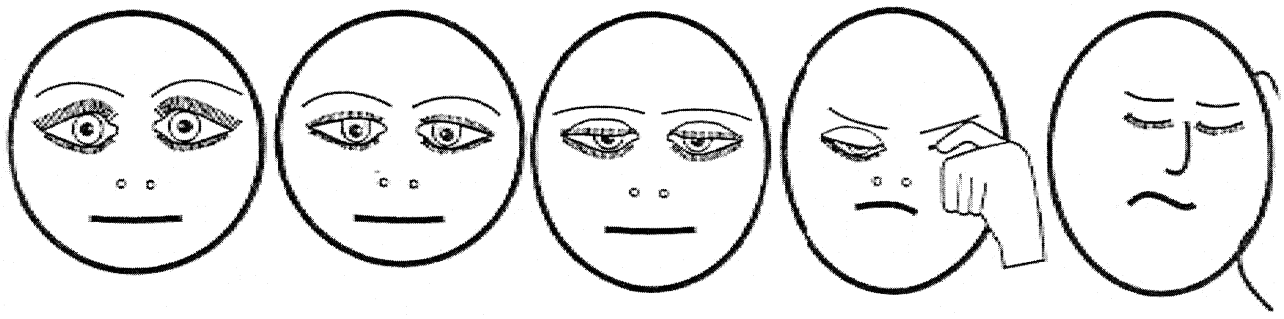


Figure 2.1 The Wits Faces Sleepiness Scale used to measure perceived sleepiness.

Subjects were also required to perform a Simple Reaction Test (SRT) (Reaction Tester, Version 2.0A, E.D. Cartwright, 1989), using a DOS-based computer time test to obtain an objective measure of alertness. During this task, subjects sat in a well-lit, quiet room facing a computer monitor from a distance of about 0.5 m. At random intervals, a white cross would appear somewhere on the surface area of the monitor. The positioning of the white cross was random. Each time a cross appeared, subjects were required to press the ENTER key on the keyboard with their right index finger as quickly and accurately as they could. Subjects performed one trial, featuring 50 crosses each.

2.3.3 Polysomnography

Gold-plated surface electrodes (Cadwell) were applied before bedtime, for the recording of EEG, EOG, and EMG on a digital electroencephalograph (Cadwell Easy EEG) at a recording speed of 15 $mm.s^{-1}$. Electrodes were placed according to the 10-20 international system of Electrode Placement (Jasper 1958), the most widely used method to describe the placement of electrodes at specific intervals along the head. Each electrode site has a letter to identify the lobe, along with a number or another letter to identify the hemispheric location. Once positions were measured and marked on the head, each site was cleaned with an alcohol solution. Electrode cups were filled with EEG paste and held in place using Collodian and tape. Subjects were then connected to a lead to the recording system, ensuring that an "earth" electrode is also attached before recording. The recordings were then scored at 30-second intervals for the following sleep stages: wake, 1, 2, 3, 4, and REM, using standardized criteria (Rechtschaffen and Kales 1968). The period of each sleep stage was taken as a fraction of the total sleep time (TST) and expressed as a percentage (%) of stage 1, stage 2, SWS (combination of stage 3 and 4) and REM. In addition, sleep onset (SOL) and REM sleep onset (ROL) latencies were calculated.

2.4 Protocol

The experimental nights were run in a randomised order, as a crossover protocol, with subjects acting as their own controls. All subjects were studied

on three different trial protocols; baseline (BL), one (SD1) and two (SD2) nights partial sleep deprivation (Table 2.1). Excluding adaptation, each subject spent a total of four nights (1 night each for BL and SD1, and both nights during SD2) and three days (for exercise testing) in the laboratory.

Subjects came into the laboratory at 20h00, at which time preparation and set-up for the next day's exercise sessions began. They completed the baseline (S0) POMS and Wits Faces Sleepiness Scale at 21h00, after which they were due to get ready for the first night of polysomnography. On the night of baseline testing, lights were turned off from 23:00 to 06:00, giving the subjects a total of 7 hours to sleep – this number was calculated based on the reported average sleep time of the subjects. On the other two occasions, subjects were partially sleep deprived for either one night (SD1) or two consecutive nights (SD2) and were allowed to sleep for a total of 4 hours from 23:00 to 03:00 on each of these nights. The first shortened night of the SD2 experimental protocol, was followed by a period of approximately 3 hours during which subjects were required to remain awake, engaged in sedentary activities in the sleep laboratory until 06:00. They were then released to attend to their normal daily activities and were required to report back to the sleep laboratory at 20:00 for the second night of recording. Although no monitoring occurred on this first day, subjects were given strict instruction not to nap, drink alcoholic and caffeine beverages or engage in strenuous activities. On the second night of SD2, subjects again slept for 4 hours, to be woken at 03:00 for the first experimental session the next morning. Upon waking, subjects were allowed 30 minutes prior to their scheduled start time, to prepare for the first exercise session. Due to the early start to exercising, subjects were given an energy

bar to increase available glucose levels (sugar = 10.8 g; energy = 723.6 kJ per 40 g bar) prior to the commencement of exercise, and only given breakfast at the end of the first session. They were also weighed (Mettler TE/J scale, Protea Laboratory, Johannesburg, South Africa) to detect any changes in weight as a result of possible dehydration. In addition, subjects performed the Simple Reaction Test (S1–S4) just before each exercise session of the day. Exercise performance determinations, the details of which are outlined below, occurred during the days following sleep on BL, SD1 and the second night of SD2; a total of three days.

2.5 Exercise

The chosen protocol allowed for subjects to engage in prolonged aerobic exercise taking place throughout the day, at a work rate that is self-selected, but exhaustive as subjects were asked to cover as much distance as possible within a given period of time. Subjects were requested to bring their own bicycles to the laboratory to exclude differences in performance resulting from the use of foreign equipment. Bicycles were mounted on a previously calibrated cycle ergometer (Cyclosimulator, Model CS-1000, Cateye Co LTD, Osaka, Japan). The metabolic cart used for this part of the experiment was the same as that used for the determination of VO_{2max} and was validated and calibrated in the same manner. Each exercise session (E1, E2, E3, E4) was preceded by a 5 to 10-minute warm-up bout and separated from the next by a period of 140 to 160 minutes. Subjects were asked to cycle for 40 minutes per session on a carefully calibrated cycle ergometer, covering as much distance

as possible, at a self-chosen work rate. However, all were encouraged to perform at their best in every exercise session, and to complete each bout having reached their personal target distance or greater.

Table 2.1 The protocol of all experimental procedures occurring over 24 or fewer hours. The protocol includes sleep periods, completion of tasks and questionnaires (S0-4), exercise (E1–E4) and rest periods; SRT = Simple Reaction Test; BL = Baseline; SD1 = one night of sleep deprivation; SD2 = two consecutive nights of sleep deprivation).

	BL	**SD1**	**SD2**
20h00	Arrival/set-up	Arrival/set-up	Arrival/set-up
21h00	Baseline scales	Baseline scales	Baseline scales
	S0	S0	S0
22h00	Sleep prep	Sleep prep	Sleep prep
23h00	Sleep	Sleep	Sleep
00h00			
01h00			
02h00			
03h00		Wake time	Wake time
		Scales and SRT	Scales and SRT
		S1	S1
04h00		E1	E1
05h00		Sedentary	Sedentary
06h00	Wake time	activities	activities
	Scales and SRT	Scales and SRT	Scales and SRT
	S1	S2	S2
07h00	E1	E2	E2
08h00	Sedentary	Sedentary	Sedentary
09h00	activities	activities	activities
10h00	Scales and SRT	Scales and SRT	Scales and SRT
	S2	S3	S3
11h00	E2	E3	E3
12h00	Sedentary	Sedentary	Sedentary
13h00	activities	activities	activities
	Scales and SRT	Scales and SRT	Scales and SRT
	S3	S4	S4
14h00	E3	E4	E4
15h00	Sedentary activities		
16h00			
17h00	Scales and SRT		
	S4		
18h00	E4		

Subjects were allowed to drink *ad libitum* during the sessions. They wore a nose clip and breathed through a mouthpiece for the last 2 minutes of every 10-minute interval. Minute ventilation (V_E), as well as oxygen consumption (VO_2) and carbon dioxide production (VCO_2), were determined at 30-second intervals using the metabolic cart (average of 4 measurements within 2 minutes of each 10-minute interval). The final VO_2 after 40 minutes of exercise was used to calculate the percentage of VO_{2max} at which each subject exercised for a given exercise session. Also recorded during each session were (i) Heart rate, every 30 seconds of the final 2 minutes of each 10-minute interval (Polar Edge Heart Rate Monitor, Polar electro Oy, Finland) and (ii) Distance covered in 10 minutes and the total distance at the end of each session. Capillary blood was taken from the tip of a pre-warmed index finger at rest and at the end of each exercise trial and analysed to determine plasma lactate concentration using a hand-held lactate analyser (Accutrend Lactate, Type 3012522, Roche Co, Johannesburg, South Africa).

2.6 Data analysis

Statistical analysis was carried out using *GraphPad Instat* Version 3 (El Camino Real, San Diego, USA). Parametric data analysis methods were employed following normal probability plotting to determine whether data came from a normal distribution. Analysis of TMD scores was done by repeated measures analysis of variance (ANOVA) to determine the difference between all 5 sessions (S0, S1, S2, S3 and S4) as well as the average scores during 3 conditions (BL, SD1 and SD2). To investigate the effects of partial

sleep deprivation on mood, repeated measures ANOVA were also performed amongst all mood subscales in 3 experimental conditions (BL, SD1 and SD2). Where significant differences in scores were found, a post hoc test (Tukey) was done. The average simple reaction times of subject 1 to 8 were also analysed using the one-way ANOVA to compare reaction times between experimental conditions. Repeated measures ANOVA were run on all outcome variables of polysomnographic data, comparing each subject to themselves on 3 occasions (BL, SD1 and SD2). In addition, total sleep time (TST), sleep (SOL) and REM onset (ROL) latencies, as well as total percentages of sleep stages obtained during the three experimental conditions were averaged for each condition to determine means and standard deviations of each parameter, and analysed using one-way ANOVA for averaged data. Pearson product moment correlations between percentage of sleep variables (including Stage 1, 2, SWS, REM, SOL and TST) and sleepiness, mood and reaction time during the BL, SD1 and SD2 conditions, as well as correlations between percentage of sleep variables and performance variables (heart rate, distance and % VO_{2max}) for exercise, were calculated. Furthermore, correlations amongst the different sleep variables and exercise variables were calculated. The averaged data for barometric pressure, lactate and weight, were analysed by one-way ANOVA to assess differences across conditions, while all other data between exercise sessions (E1–E4) for heart rate, distance, speed, V_E, and VO_2 were analysed by repeated measures ANOVA to assess differences between exercise sessions. A p value below 0.05 was considered significant. Data are expressed as mean and standard deviation (SD) unless otherwise stated.

CHAPTER 3

RESULTS

The demographics of the participants in the study (mean (SD)) are as follows: age, 26.0 (0.4) years; height, 1.76 (0.80) m; body mass, 75.0 (13.0) kg; VO_2max, 54.0 (4.2) ml/min/kg. Subjects maintained their baseline body weights between exercise sessions during all experimental days, i.e. body mass for BL = 74.2 (12.2) kg; SD1 = 75.2 (13.6) kg; SD2 = 74.7 (13.9) kg.

3.1 Questionnaires and tasks

The mean scores for the questionnaires (POMS and the Wits Faces Sleepiness Scale) that were completed in all five sessions (S0–S4) and in four sessions (S1–S4) for reaction time (SRT) during the three trial days (BL, SD1 and SD2) are indicated in table 3.1.

3.1.1 Mood

A repeated measures ANOVA showed that the total mood disturbance (TMD) was significantly higher ($p = 0.0004$) across conditions on the night prior to testing (S0) (average > 21) than the average of all sessions on the days of testing (S1–4). Differences in TMD were also found for SD1 and SD2 compared to BL conditions as well as SD1 compared to SD2 (Table 3.1).

Table 3.1 Mean (SD) scores of the POMS (Total Mood Disturbance).

	POMS Total Mood Disturbance scores		
	BL	**SD1**	**SD2**
S0	25.4 (1.8)	22.0 (2.8)	12.0 (2.9)*#
S1	6.5 (1.3)	9.2 (2.5)	7.3 (2.1)
S2	1.7 (1.5)	7.8 (3.0)*	3.4 (2.6)#
S3	1.4 (2.0)	3.6 (2.8)*	0.7 (2.8)#
S4	1.0 (2.0)	4.8 (3.4)	2.0 (3.3)*
Mean scores	7.2 (1.7)	9.5 (2.9)	5.1 (2.7)

* SD1 and SD2 ($p<0.05$) compared to BL and # SD1 ($p<0.05$) compared to SD2. All statistics are repeated measures ANOVA. BL = baseline condition; SD1 = one night of sleep deprivation; SD2 = two nights of sleep deprivation. S0–S4 = sessions during which tasks and questionnaires were completed.

Furthermore, the average score across all sessions for the subdivisions of the POMS scores, the subjective measures of "tension" ($p = 0.18$) and "confusion" ($p = 0.06$) were not significantly affected by sleep deprivation. However, changes were evident in the "depression", "anger", "fatigue", and "vigour" subscales, such that subjects felt less depressed during SD2 compared to BL

(p = 0.0041), less angry during both SD1 and SD2 compared to BL (p = 0.0266), more fatigued during SD1 and SD2 compared to BL (p = 0.032) and less vigorous during both SD1 and SD2 in comparison to the BL condition (p = 0.0008) (Figure 3.1). No significant differences were found when comparing the scores for any of the subscales between SD1 and SD2.

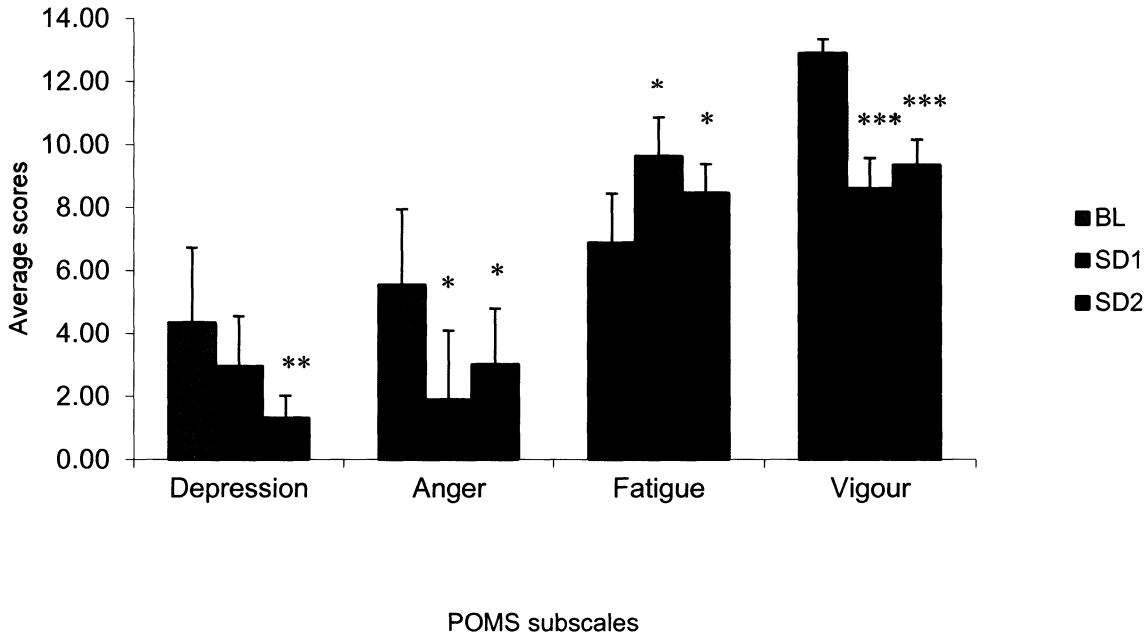


Figure 3.1 Mean scores of four significant POMS subscales (Depression, Anger, Fatigue and Vigour) over each day during baseline and two experimental conditions. BL = baseline condition, SD1 = one night of partial sleep deprivation. SD2 = two nights of partial sleep deprivation. Comparisons to BL (*p < 0.05; **p < 0.01; ***p < 0.001).

3.1.2 Sleepiness

A repeated measures ANOVA analysis revealed significant differences in the night before exercise (S0) versus session 2 (S2) scores of sleepiness faces in the baseline condition, such that subjects felt more awake during the day after a baseline night ($p < 0.01$). Subjects also felt more awake during S3 than the night before testing (S0) ($p < 0.01$) but sleepier in the very next session (S4) compared to S3 ($p < 0.05$) during the SD1 condition (Table 3.2)

Table 3.2 Mean (SD) scores of the Wits Faces Sleepiness Scale (Subjective sleepiness).

	Subjective sleepiness scores		
	BL	**SD1**	**SD2**
S0	1.9 (0.6)	1.8 (0.5)	2.1 (1.4)
S1	0.8 (0.6)	1.8 (1.1)	1.4 (1.0)
S2	0.5 (0.8)***	1.7 (1.1)	2.0 (1.0)
S3	0.9 (0.02)	0.6 (0.4)***	1.8 (0.2)*
S4	1.4 (0.08)	2.0 (0.3)**	1.6 (0.2)
Mean scores	1.1 (0.5)	1.6 (0.5)	1.7 (0.3)#

* SD1 compared to SD2 ($p<0.01$), ** SD1 S4 compared to S3 ($p<0.05$), *** values compared to S0 ($p<0.01$) in BL and SD1, # mean SD2 compared to mean BL ($p<0.05$). All statistics are repeated measures ANOVA. BL = baseline condition; SD1 = one night of sleep deprivation; SD2 = two nights of sleep deprivation. S0–S4 = sessions during which tasks and questionnaires were completed.

In addition, the mean sleepiness scores (an average of S0–S4) of SD2 were significantly higher compared to BL such that subjects felt sleepier throughout SD2 compared to BL ($p < 0.05$). However, average sleepiness scores in SD1

were not significantly different compared to BL and to SD2.

3.1.3 Reaction time

Statistical analysis revealed faster reaction times during S4 compared to S1 in the BL condition ($p = 0.01$) (Table 3.3). However, no significant differences were found for averaged data (S1–S4) between BL, SD1 and SD2.

Table 3.3 Mean (SD) scores of the Simple Reaction Test (Reaction time).

	Reaction time (ms)		
	BL	**SD1**	**SD2**
S0	-	-	-
S1	305 (16.8)	301 (8.8)	301 (14.2)
S2	289 (19.8)	283 (15.1)	293 (19.8)
S3	288 (21.9)	286 (11.4)	289 (23.2)
S4	282 (24.3)*	288 (16.7)	289 (18.6)
Mean scores	291 (20.7)	290 (13.0)	293 (18.9)

* BL S4 ($p<0.01$) compared to S1. All statistics are repeated measures ANOVA. BL = baseline condition; SD1 = one night of sleep deprivation; SD2 = two nights of sleep deprivation. S0–S4 = sessions during which tasks and questionnaires were completed.

3.2 Polysomnography

A repeated measures ANOVA revealed that each subject had their sleep significantly reduced by an average of 3.8 ± 0.4 hours per night on both SD1 and SD2, compared to BL ($p < 0.05$) (Table 3.4). The sleep and REM

latencies (min) and the percentage of sleep stages in relation to TST for all three conditions are also given in Table 3.4. During SD1, there was a significant increase in percentage of slow wave sleep and decrease in percentage in REM sleep compared to the BL condition. These changes reverted to normal percentages of sleep during SD2.

Table 3.4 Mean (SD) percentages of sleep stages and latencies (min) during polysomnographic recordings on baseline and sleep deprivation conditions.

Experimental condition	Percentage of TST Mean (SD)				TST in minutes Mean(SD)	SOL in minutes Mean (SD)	ROL in minutes Mean (SD)
	Stage 1	Stage 2	SWS	REM			
BL	2.5 (1.7)	49.9 (9.8)	22.7 (4.9)	22.3 (4.8)	353.3 (72.2)	9.2 (5.1)	65.1 (15.2)
SD1	3.5 (3.3)	45.3 (6.6)	32.0 (7.2)*	17.2 (6.7)*	218.9 (24.5)*	11.1 (8.4)	69.3 (18.5)
SD2	1.7 (1.5)#	54.2 (10.8)	22.7 (5.6)#	19.0 (7.4)	231.0 (25.6)*	6.6 (4.5)#	76.0 (39.6)

BL = baseline condition; SD1 = one night of sleep deprivation; SD2 = two nights of sleep deprivation; SWS = slow wave sleep; REM = rapid eye movement; TST = total sleep time; SOL = sleep onset latency; ROL = REM onset latency. Latencies are in minutes for SOL and ROL. *significantly different from BL ($p<0.05$) and # significantly different compared to SD1 ($p<0.05$) (one-way ANOVA).

3.3 Exercise

All subjects exercised in an environment that did not differ significantly across experimental days (temperature, humidity, air movement, barometric pressure). The average changes in plasma lactate concentrations at the end of exercise sessions across subjects and between conditions were not

significantly different from each other (Table 3.5). No significant differences were found when comparing SD1 and SD2 to BL minute ventilation (V_E).

Table 3.5 Mean (SD) blood lactate and minute ventilation scores across subjects and conditions.

Subject ID	Blood Lactate (mmol/l)			Minute Ventilation (l/min)		
	BL	**SD1**	**SD2**	**BL**	**SD1**	**SD2**
CL1	11.3(1.6)	4.6(0.4)	7.9(0.8)	86.7(7.7)	89.1(10.8)	89.3(7.4)
CL2	2.2 (0.1)	2.5 (0.5)	1.8 (0.3)	73.6(8.8)	78.5(7.2)	66.2(8.4)
CL4	5.7 (1.8)	3.3 (1.5)	5.9 (2.5)	93.7(5.3)	90.7(1.5)	87.6(5.7)
CL5	1.8 (0.3)	3.0 (0.3)	2.4 (0.2)	82.6(7.7)	103.7(9.7)	78.5(7.2)
CL7	5.8 (3.5)	4.2 (0.9)	6.6 (2.5)	62.1(20.8)	66.2(8.4)	60.9(11.8)
CL8	5.8 (2.7)	6.4 (2.3)	6.7 (3.2)	63.7(15.1)	63.9(11.6)	55.9(6.9)
CL9	3.1 (1.8)	2.5 (0.5)	3.7 (2.4)	54.7(1.1)	55.6(1.9)	52.2(8.2)
CL10	2,7 (2.0)	4.3 (1.8)	2.4 (0.1)	49.7(1.3)	47.3(4.1)	56.9(5.2)
Mean	4.8	3.9	4.7	70.9	74.4	68.4
SD	1.7	1.2	1.9	15.9	19.4	14.7

Average blood lactate after four 40-minute exercise sessions is expressed in mmol/l. Minute ventilation is expressed as l/min. BL = baseline condition; SD1 = one night of sleep deprivation; SD2 = two nights of sleep deprivation. ($p<0.05$) (one-way ANOVA).

Subjects were also able to maintain their normal body weight between exercise sessions during all experimental days: BL = 74.2 (12.2) kg; SD1 = 75.2 (13.6) kg; SD2 = 74.7 (13.9) kg ($p = 0.00034$). Although the distances cycled in each condition were significantly different ($p < 0.0001$) between subjects (minimum = 14.32 km; maximum = 29.71 km), the average total distances cycled throughout the exercise day during three conditions (BL =

93.2575; SD1 = 89.20375; SD2 = 89.24375) were not significantly different (p = 0.88) (see Figure 3.2). Table 3.6 shows the variation in the average distances covered by each subject in each condition.

Table 3.6 Mean and total distance covered by each subject, as well as the absolute difference in distance from baseline (BL).

	Average Distance per session (km)			Total Distance per day (km)			Change from BL (km)	
	BL	**SD1**	**SD2**	**BL**	**SD1**	**SD2**	**SD1**	**SD2**
CL1	15.02	16.15	16.64	60.08	64.58	66.56	1.13	1.62
CL3	26.1	23.93	26.15	104.4	95.73	104.6	-2.17	0.05
CL4	27.21	23.93	28.47	108.8	95.73	113.9	-3.28	1.26
CL5	29.71	26.35	25.55	118.9	105.4	102.2	-3.36	-4.16
CL7	24.26	26.15	22.53	97.04	104.6	90.12	1.89	-1.73
CL8	24.61	24.82	21.69	98.45	99.28	86.77	0.21	-2.92
CL9	22.95	22.76	22.98	91.8	91.04	91.93	-0.19	0.03
CL10	16.83	14.32	14.47	66.59	57.27	57.87	-2.51	-2.36
Mean	**23.34**	**22.30**	**22.31**	**93.26**	**89.20**	**89.24**	**-1.04**	**-1.03**

Average distance of four 40-minute exercise sessions is expressed in km travelled. Total distance is the sum of four 40-minute exercise session for each subject. Change from BL is calculated based on the total distance covered in each condition. BL = baseline condition; SD1 = one night of sleep deprivation; SD2 = two nights of sleep deprivation.

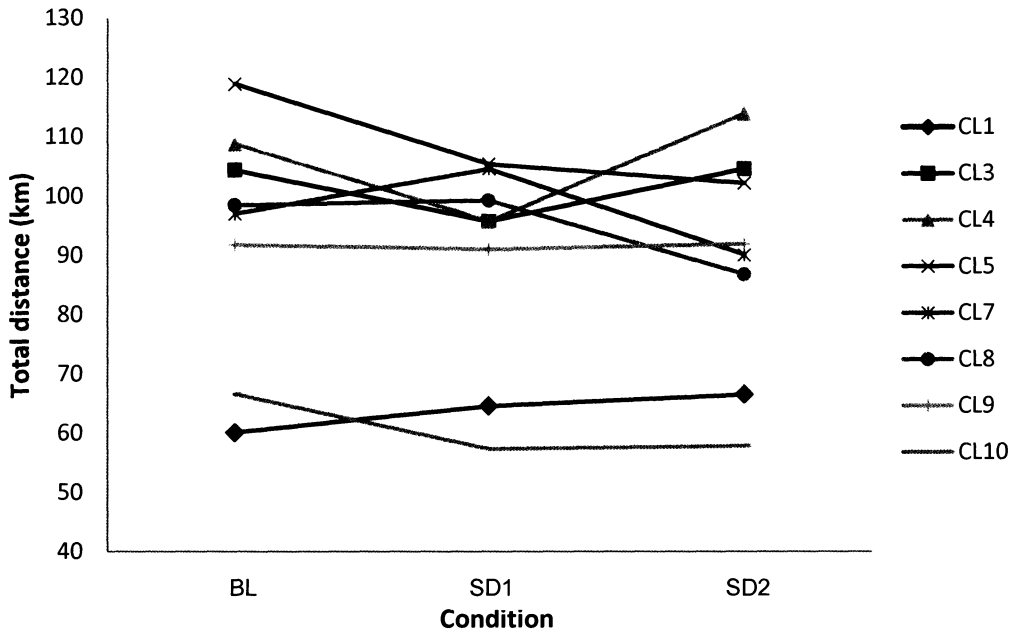


Figure 3.2 Distances covered by each subject in the baseline and two experimental conditions. BL = baseline condition, SD1 = one night of partial sleep deprivation, SD2 = two nights of partial sleep deprivation, CL – CL10 = subject codes.

Similarly, the average speed at which subjects cycled was not significantly different across conditions ($p = 0.36$ repeated measures ANOVA). The mean (SD) oxygen consumption during exercise, expressed as a percentage of each subject's VO_2max, did not differ significantly between conditions: BL = 68.2 (16.1) %, SD1 = 64.4 (18.8) % and SD2 = 64.7 (18.2) %) ($p = 0.89$). During exercise, the mean heart rate was found to be significantly lower in the first session of exercise compared to the third and the last session ($p < 0.01$) for all conditions (see Figure 3.3). However, the average heart rate between

conditions was not significantly different (BL = 146.1 (22.1) bpm, SD1 = 148.3 (24.0) bpm, SD2 = 142.4 (23.9) bpm) (p = 0.88).

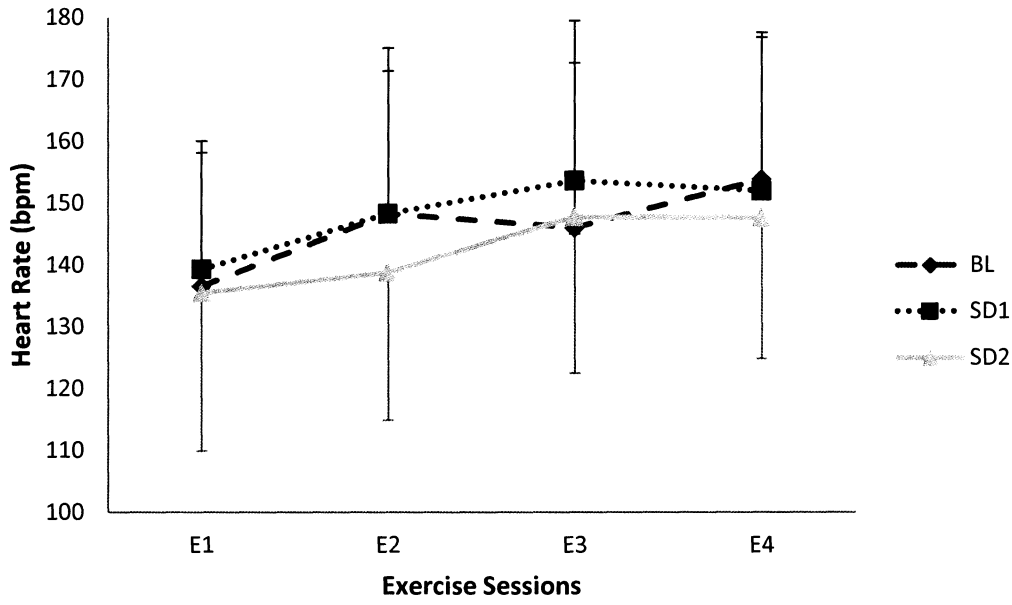


Figure 3.3 Mean heart rate over four exercise sessions (E1-E4) in the baseline and two experimental conditions. BL = baseline condition, SD1 = one night of partial sleep deprivation. SD2 = two nights of partial sleep deprivation. *p < 0.05 compared to E1 in all conditions (one-way ANOVA). Heart rate at the end of each exercise sessions did not differ significantly (p > 0.05) between conditions.

3.4 Correlations

Sleep, mood, sleepiness and reaction times were correlated to exercise variables and each other. None of the polysomnographic sleep variables correlated significantly to distance, speed and V_E, however, %REM sleep correlated significantly to $\%VO_2max$ (r = 0.99; p = 0.01). Of the four POMS

subscales that were evidently affected by sleep deprivation, only depression ($r = -0.55$; $p = 0.03$) was inversely correlated to subjective sleepiness.

Correlations of the other three subscales, anger ($r = -0.45$; $p = 0.09$), fatigue ($r = 0.40$; $p = 0.14$) and vigour ($r = -0.49$; $p = 0.06$) to subjective sleepiness were not found to be significant. Correlations between subjective mood ratings and polysomnographic sleep variables revealed an inverse relationship between fatigue on the POMS scale and stage 2 sleep ($r = -0.99$, $p = 0.05$). Furthermore, stage 1 was positively correlated to depression ($r = 0.99$; $p = 0.04$), stage 2 to anger ($r = 0.99$; $p = 0.02$) and REM to vigour ($r = 0.99$; $p = 0.05$). Mood ratings were not correlated to any exercise variables.

There were inverse correlations between sleepiness and stage 1 sleep ($r = -0.55$; $p = 0.03$), and SWS ($r = -0.53$; $p = 0.04$), as well as a positive correlation between ROL ($r = 0.53$; $p = 0.04$) and sleepiness but no other correlations to sleep variables were significant during BL, SD1 and SD2. Subjective sleepiness was inversely correlated to distance cycled ($r = -0.56$; $p = 0.03$), speed of cycling ($r = -0.56$; $p = 0.03$) and $\%VO_2max$ ($r = -0.51$; $p = 0.04$).

No correlations were found between perceived sleepiness, sleep and exercise variables or any of the POMS subscales with reaction time on BL, SD1 or SD2.

Table 3.7 Correlation matrix for mood, sleep and exercise parameters.

	Depression	Fatigue	Anger	Vigour	Sleepiness	ROL	Stage 1	Stage 2	SWS	REM	VO2max	Distance	Speed
Depression	1.0				-0.55		0.99						
Fatigue		1.0						-0.99					
Anger			1.0					0.99					
Vigour				1.0						0.99			
Sleepiness					1.0	0.53	-0.55		-0.53			-0.56	
ROL						1.0					-0.51		-0.56
Stage 1							1.0						
Stage 2								1.0					
SWS									1.0				
REM										1.0	0.99		
VO2max											1.0		
Distance												1.0	
Speed													1.0

Correlations in the matrix shown above are those which were significantly different at $p<0.05$.

CHAPTER 4

DISCUSSION

4.1 Summary

This study tested the anecdotally-derived hypothesis that sleep deprivation occurring the night immediately before an event does not negatively affect performance, provided that one sleeps well the night before the sleep deprivation night. Reducing the sleep of a group of eight cyclists to 4 hours per night for one or two nights prior to prolonged exercise had only a few quantifiable differences. Subjects experienced greater fatigue and reduced vigour after both one night and two nights of sleep deprivation as compared to the baseline condition. There was an increase in percentage SWS and a decrease in percentage REM during the first night of sleep deprivation (SD1) that were reversed after two nights of sleep deprivation (SD2). Exercise performance variables (lactate levels, oxygen consumption, heart rate, distance and speed) were not significantly different between the three conditions. Sleepiness was inversely related to depression, percentage stage1 and slow wave sleep, and positively correlated to REM onset latency.

4.2 Study limitations

The sleep protocol employed in this study did not include a recovery period and therefore, made it difficult to quantify the distribution of sleep stages and any possible rebound in sleep after each experimental condition. As seen

here, SWS was restored (SD2 levels were similar to BL) while REM measures were indicative of a slow return to basal levels after sleep deprivation. The current study also neglected to measure reaction times of subjects on the night prior to testing, as a true baseline measure (S0). This measurement is necessary in order to make comparisons with subsequent measurements taken during the experimental protocol. For exercise data, including distance, speed, oxygen consumption and heart rate, variability would have been reduced had the subjects been more comparable in age and level of fitness.

The exercise protocol in the current study was deemed exhaustive and the average cumulative distance covered by each subject is evidence of the importance of endurance capacity in completing the study. Measurements of power for the purpose of exploring the effects of sleep loss on anaerobic exercise was not part of the overall objectives of the study and was therefore excluded. However, it may be useful in a further study to draw comparisons between the effects of circadian rhythms and sleep loss on aerobic and anaerobic exercise performance. A further shortcoming is the lack of relation to a real life setting of endurance events because most 1-day events typically take place continually, covering on average, 50 to 70km per event. In the current study, the average cumulative distance covered is over 90km per day, spread over four sessions. It may be beneficial for future research to closely simulate a known endurance cycling or mountain biking event in order to allow for more direct applicability of findings.

In the current study, subjects were mostly detained within the boundaries of the experiment during each condition, and had no unsupervised access to their usual daily activities on the days of exercise recording (day 2 of SD1 and SD2). A possible limitation of the protocol was our reliance on subjects to 1) limit their own activity during the day after the first night of the SD2 condition prior to exercise, and 2) avoid taking a nap during that same day when their activity was not monitored.

4.3 Discussion of results

To test the subjective and objective performance effects in realistic terms, we used a model of partial sleep deprivation similar to that experienced by ultra-marathon athletes during events, as described herein as SD1 and SD2. The SD1 condition in this study is employed to examine the effects of sleep deprivation on the night prior to an event, provided that the athlete slept well (7-8 hours) two nights before the event. The hypothesis for this protocol was, as per anecdotal evidence, that exercise performance is not negatively affected by the partial sleep loss that is often the result of an early and anxious start to an ultra-marathon event.

On the contrary, SD2 is based on the assumption of losing up to 4 hours of sleep on both nights leading up to an important event. This, according to many athletes, is a cause for concern as performance is likely to dip as a result of the partial sleep restriction. Therefore, the SD2 condition should see

some performance deficits following the second night of deprivation in response to the accumulated sleep debt, compared to SD1.

4.3.1 Mood

The outcomes of the POMS included higher TMD scores during several sessions under sleep loss conditions compared to baseline. It is important to note the outlying true baseline (S0) score, taken the night before any intervention, and indicating anxiety. A meta-analysis (Pilcher and Huffcutt 1996) and other studies reported that losing at least 4 hours of sleep or more per night for 2 or more consecutive nights, results in mood deficits (Dinges 1997; Meney 1998). In one experiment, subjects were studied on two different occasions (control and sleep deprivation) of 2 nights each, with the results showing a significant negative effect of sleep deprivation on most mood states after night 1 but mood recovery on day 2 (Meney 1998). Another study (Dinges 1997) explored the effects of shorter but cumulative bouts of sleep deprivation over 7 consecutive days which resulted in longer-lasting mood deficits. This general alteration in mood states suggests that the ultimate damage in response to this sleep loss is a function of cumulative effects if the sleep loss is not interrupted by naps.

We also found that, more specifically, reducing sleep by 4 hours per night on either one or two consecutive nights resulted in considerably more subjective fatigue and less vigour. Fatigue after sleep loss tended to increase to levels higher than those after a baseline night, suggesting that subjects felt the tiring

effects of sleep loss prior to any physical activity. Vigour worsened to low levels as a result of sleep restriction thereby confirming the subjects' low levels of perceived physical and mental energy compared to baseline values. Deteriorating energy levels after extended periods of sleep loss have been reported before, where vigour and fatigue are worsened by the extent to which the subject is deprived of up to 30 hours of sleep (Scott 2006). In that study, it was evident that sleep deprivation was associated with significantly negative disturbances to depression as well, as assessed by the POMS. This is contrary to the current finding of reduced subjective depression in SD2 in comparison to baseline. However, both fatigue and vigour were influenced by partial sleep loss of up to four hours a night, whereas subjective depression may take a while to surface. Our results did not show a significant difference between the effects on subjects' mood by sleep loss between SD1 and SD2, contrary to our anecdotally-derived hypothesis; noteworthy differences were seen only in comparison of the two sleep deprivation conditions to the baseline condition. Furthermore, counter to findings by Dinges (1997), it can be seen here that mood deficits occur long before sleep loss reaches detrimental levels. Therefore, in line with the findings of Pilcher and Huffcutt's (1996) and Meney (1998) meta-analyses, it is clear that mood is more sensitive to sleep deprivation than any other variable, as seen with the effects on our subjects during SD1. It may even be that mood recovery occurs on the day after sleep deprivation (Meney 1998) thereby supporting the notion that mood improvements can be seen after a night of sleep deprivation where sleep loss is not cumulative.

4.3.2 Sleepiness

Subjects reported on their perceived levels of sleepiness on the preceding night and at the beginning of each session on the day of exercise testing in each condition. In general, subjective sleepiness levels varied throughout the period of testing from S0 to S4. During the baseline condition, subjects experienced expected levels of sleepiness, such that they felt sleepier on the preceding night than during the day of testing. This was likely as a function of circadian rhythms and possibly a result of the arousing effects of their physical exertion throughout the day, compared to the night before testing. During SD1, subjects reported being alert only until S3, but sleepiness surfaced towards the end of the day with subjects feeling sleepiest in the final session (S4). The faces scale also revealed that subjects felt the least alert during SD2 from S0. No proven recovery occurred during the day of testing (S1 – S4), and on average, the SD2 condition yielded the highest levels of sleepiness compared to baseline. This was, however, not significantly different from perceived levels of sleepiness in SD1.

Although the sleep needs of the subjects involved in this study may have been different under normal circumstances, it is clear that partial sleep loss of 3 hours per night for 2 consecutive nights exacerbated their sleepiness in comparison to baseline conditions. This is in agreement with a previous report on driving while sleepy (Powell *et al.* 2001) and is also comparable to much earlier reports of measured daytime sleepiness when one experiences prolonged (up to 7 consecutive days) partial sleep deprivation (Carskadon and Dement 1981; Devoto 1999). On the contrary, according to Horne (1985), the

effects of sleep deprivation on subjective sleepiness appear to stop accumulating beyond the second night of restriction, suggesting that after the initial changes experienced in daytime sleepiness, some adaptation to sleep loss may occur. This adaptation is said to be due to the changes in sleep architecture during sleep deprivation and it is important for further investigations to explore this phenomenon by measuring levels of sleepiness during a recovery period in addition to perceived sleepiness.

4.3.3 Reaction time

Cognitive and motor performance studies have previously used the simple reaction time test to investigate the deleterious effects of sleep loss on the speed of a subject's reaction to a given stimulus. This test is usually not used in isolation but rather as part of a range of performance tests including other coordination, accuracy and attention tasks. For the purposes of this research, we employed this speed test on the basis of its relevance to this protocol as well as its simplicity and objectivity. As far as vigilance is concerned, measures of psychomotor performance, as influenced by sleepiness and alertness levels, were hardly affected by the partial loss of sleep in the current study. These results were much like in a report that found no difference between deprived and non-deprived subjects in simple reaction time, vigilance, or selective attention tasks after one night of sleep loss (Forest and Godbout 2000). However, upon waking, an early start after sleep deprivation proved to diminish subjects' alertness. This is evidenced by slow reaction to stimuli during the first session of the baseline condition (S1) compared to

subsequent trials, although significantly so in comparison to only the last session (S4).

The subjects' reaction to stimuli gradually improved as a function of the number of times they attempted the simple reaction test, regardless of perceived alertness levels. This is evidenced by the improvement in the speed of reaction by the last session of the day, whereby, performance was better than in the first session, although this was not statistically significantly. This was clearly not as a result of learning as this phenomenon carried through all conditions in randomised order. It has been suggested that there are functional clocks in the human brain cortex that are associated with the possible expression of circadian rhythm changes in reaction time, and that these rhythms are lowest in the early hours of the day (Reilly 1998). Coupled with this is the influence of the sleep homeostatic process that controls sleep pressure between wakefulness and a sleep period (Van Dongen 2005). In this study, increased amounts of slow wave sleep determined from the subjects' polysomnographic records, was not seen to be correlated to reduced vigilance from the simple reaction test. Our results were therefore not able to show that there may be a relationship between the rate of improvement in neurobehavioral functioning and rate of decline of slow-wave sleep and/or slow-wave activity (Jewett 1999).

4.3.4 Polysomnography

There is still a lack of consensus on the loss or maintenance of NREM stages when sleep is partially restricted, but to this end, the current results point to the maintenance of these stages at least for the sleep deprivation period prescribed herein. The reduction in Stage 1 and slow wave sleep, as well as the faster onset to sleep during SD2 in comparison to SD1 was likely as a result of the first sleep deprived night during SD2, such that subjects felt the effects of accumulated sleepiness from the night before. The unchanged sleep onset and REM onset latencies in the SD1 condition suggest that there was a correlation between subjective (faces scale) and objective (polysomnography) data. On the contrary, subjects were as inclined to fall asleep as was perceived and measured in the SD2 condition. In agreement with Brunner's (1993) findings, the percentage of SWS did not change during the SD2 condition in comparison to baseline. The difference in the degree of build up of both stage 3 and 4 is still unclear. However, our findings are in agreement with previous research that points to the importance of the restoration of SWS (Vgontzas 2007). The current report, therefore, supports suggestions that the preservation of SWS with sleep deprivation implies that it may have a more essential function than REM sleep. Furthermore, there was an increase in SWS and a drop in REM on SD1 compared to a return to normal values of SWS on SD2 and a seemingly slow REM rebound. It may be more important to note that SWS was preserved, contrary to the reduction that would usually be seen in patients that experience frequent arousals and delayed sleep onset as a result of insomnia and obstructive sleep apnoea (Schneider 2004).

Athletes usually compromise their second half of the night when events require an early start to the day. In support of the anecdotes of many long distance athletes, the maintenance of SWS may be an important contributing factor to maintaining restfulness and one's physical performance following a night of reduced sleep. The current study therefore, suggests that with sleep loss taking place on the second half of the night, athletes are not likely to experience an exercise performance deficit. Moreover, REM sleep was significantly reduced following sleep loss compared to the baseline condition. Because REM sleep is concentrated in the second half of the night, restricting sleep to the first half of the night is expected to result in significant REM sleep loss. This phenomenon also plays an important role in determining the time of night during which an endurance athlete should rather sleep in order to avoid performance deficits on the day of an event.

The current study, in addition to showing the importance of preserving SWS after sleep loss, also found a correlation between %REM sleep and $\%VO_2$. This shows the importance of REM sleep in maintaining optimal physical performance. It, therefore, remains debatable 1) how circadian tendency, determined by which half of the night one is sleep deprived, affects subsequent athletic performance, and 2) which stage between SWS and REM is most likely to help preserve an athlete's physical performance following a considerable degree of sleep loss

4.3.5 Exercise

Subjects all cycled under controlled environmental conditions at above 60% of their individual aerobic capacities. Lactate levels remained unchanged and subjects were able to maintain their body weight thereby confirming 1) the fitness of individuals used for this research, 2) that they remained well hydrated during testing, and 3) that they avoided any lifestyle changes that may have brought about any loss of weight. Despite the differences in mood states between the two conditions, SD1 and SD2 oxygen consumption figures suggested that subjects were cycling with similar power. Generally, subjects exercised at the highest work intensity during BL compared to both SD1 and SD2, on the basis of the actual oxygen consumption results in a given 40-minute bout. However, these results were not statistically significant. Furthermore, although not significant, it is evident that subjects were able to cycle greater distances during BL compared to both SD1 and SD2. These findings imply that the effects of sleep deprivation of one night before an event, without prior sleep loss (SD1), are not significantly different to those of sleep loss of two consecutive nights (SD2). Even more importantly, the results also suggest that athletes are most likely to perform at their best when they have not lost any sleep in the build-up to an endurance event. These are cycling or mountain biking events lasting one day but can be applicable to staged races taking place over multiple days. Examples of such events may include the Ngwenya Glass mountain bike challenge in Swaziland (65km in 1 day), the 94.7 cycle challenge (over 90km in 1 day), the Sani2C (over 250km in 3 days) and even the 7-stage Cape Epic (averaging 108km at each stage).

Although mood displays sensitivity to the reduction of normal sleep, the degree of sleep loss that will significantly alter physical performance remains unclear. It is, therefore, important to establish the effects of one, two and more consecutive nights of sleep loss, with a recovery period in further investigations.

CHAPTER 5

CONCLUSION

The current findings suggest that exercise measurements are not as sensitive to partial sleep deprivation as subjective and cognitive indicators of performance. Many trainers and exercise experts advocate for training hard, eating well and getting enough sleep as true solutions to ensuring that an athlete performs at optimum levels during competitive events. However, anecdotes from athletes suggest that they do no place much emphasis on resting well the night before an event, but rather on earlier nights in order to avoid accumulated sleep loss. As has been the case in some studies, this accumulated sleep loss begins to have negative effects on exercise performance after more than 2 nights of sleep loss, especially where sleep has only been reduced by one or two hours a night from the norm on consecutive days.

This study was unable to prove that two nights of partial sleep deprivation impacts exercise performance more than one night of sleep deprivation. Although it seems that exercise performance a day or two after partial sleep deprivation is essentially unaffected, there could be a limit to how much sleep deprivation the body can take before the effects are evident. Although not significant, the decline in physical performance, as determined by oxygen consumption (higher BL $\%VO_2$ compared to SD1 and SD2) as well as cycling distances (greater BL distances compared to SD1 and SD2), implies that performance levels may be significantly affected by a higher degree of sleep

loss than what the present study allowed.

Furthermore, endurance training develops the energy production systems such that the demands of exercise activity can be met for the longest period of physical activity. In order to improve endurance, athletes usually strive to push their capacity to exercise longer and harder, an ability that can be limited by fatigue and exhaustion. However, sustained training has been shown to prolong the point at which this fatigue occurs, hence the decreased sensitivity to the currently experienced levels of fatigue and exhaustion. The question, in practice, remains: how fatigued do athletes have to be to have their performance significantly affected by sleep loss?

As per the known and outlined limitations of the current study, the following should be taken into account when designing further studies in an effort to obtain clarity on the effects of a similar partial sleep deprivation protocol. Firstly, in consideration of the distribution of REM sleep across a normal night and for the purposes of further research into the effects of partial sleep loss on exhaustive exercise performance, it may be valuable to test the differences in restricting sleep to either the first or the second half of the night. Pearson correlations revealed that the decrease in REM sleep duration during sleep deprivation was related to the slightly compromised (not significantly so) exercise performance as determined by $\%VO_2max$ seen during SD1 and SD2. Therefore, it is now also necessary to test the relationship between the REM sleep duration, as well as differences seen after REM recovery, and physical performance measures. Furthermore, recording sleep for a further day or two

under baseline conditions following the sleep deprivation protocol would be useful in determining the expected REM and SWS rebound. In terms of monitoring the daily activity of subjects, future studies should consider either confining subjects to the sleep laboratory for the complete duration of the study or making use of daily logs to monitor and record activity. Further studies attempting to determine how much sleep athletes should have to avoid performance decrements should take into account that under known circumstances, endurance athletes beginning an event during the early hours of the morning are not likely to miss more than 4 hours of sleep prior to the event. In this case, it would be beneficial to employ an exhaustive exercise protocol that simulates real life endurance events, as well as to include further measurements of performance such as perceived exertion.

Finally, it is also important to note that the results of this study may be restricted only to cycling and mountain biking, and other studies may find different results in other endurance sports such as running, swimming and rowing.

REFERENCES

Akerstedt, T. (1979). Altered sleep/wake patterns and circadian rhythms. Laboratory and field studies of sympathoadrenomedullary and related variables. *Acta Physiol Scand Suppl* **469:** 1-48.

Akerstedt, T. (2003). Shift work and disturbed sleep/wakefulness. *Occup Med* **53:** 89-94.

Angus, R. (1985). Effects of prolonged sleep deprivation, with and without chronic physical exercise, on mood and performance. *Psychophysiology* **22:** 276-282.

Bach, V. (1994). Effect of continuous heat exposure on sleep during partial sleep deprivation. *Sleep* **17:** 1-10.

Bennington, J.H. and Heller, H.C. (1995). Restoration of brain energy metabolism as the function of sleep. *Prog Neurobiol* **45:** 347–360.

Best, B. (2006). The nature of sleep and its impact on health. Available from internet: <http://www.benbest.com/health/sleep.html>. Accessed in July 2008.

Bond, V., Balkisson, B., Franks, B.D., Brownlow, R., Caprarola, M., Bartley, D. and Banks, M.A. (1986). Effects of sleep deprivation on performance during submaximal and maximal exercise. *Sports Med* **26:** 169-174.

Bonnet, M.H. (1995). We are chronically sleep deprived. *Sleep* **18:** 908-911.

Bonnet, M.H. (2003). Clinical effects of sleep fragmentation versus sleep deprivation. *Sleep Med Rev* **7:** 297-310.

Bonnet, M.H. (2005). Acute sleep deprivation. In: *Principle and Practice of Sleep Medicine* (4th ed). Kryger, M.H., Roth, T., Dement, W.C. (Eds), WB Saunders, Philadelphia, pp 51-66.

Brandenberger, G., Gronfier, C., Chapotot, F., Simon, C. and Piquard, F. (2000). Effect of sleep deprivation on overall 24h growth hormone secretion. *Lancet* **356:** 1408-1412.

Bruce, R.A., Blackmon, J.R., Jones, J.W. and Strait, G. (1963). Exercising testing in adult normal subjects and cardiac patients. *Pediatrics* **32:** 742-756.

Brunner, D.P. (1993). Repeated partial sleep deprivation progressively changes the EEG during sleep and wakefulness. *Sleep* **16:** 100-113.

Bunnell, D.E., Bevier, W. and Horvath, S.M. (1982). Effects of exhaustive exercise on the sleep of men and women. *Psychophysiology* **20:** 50-58.

Canet, E., Gaultier, C., D'Allest, A.M. and Dehan, M. (1989). Effects of sleep deprivation on respiratory events during sleep in healthy infants. *J Appl Physiol* **66:** 1158-1163.

Carskadon, M.D. and Dement, W.C. (1981). Cumulative effects of sleep restriction on daytime sleepiness. *Psychophysiology* **18:** 107-113.

Carskadon, M.D. and Dement, W.C. (2005). Normal human sleep: An overview. In: *Principles and Practice of Sleep Medicine*. (4th ed), Kryger, M.H., Roth, T. and Dement, W.C. (Eds), WB Saunders, Philadelphia, pp. 13-23.

Chen, H. (1991). Effect of 30-h sleep loss on cardiorespiratory functions at rest and in exercise. *Med Sci Sport Exerc* **23:** 193-198.

Consolazio, C.F., Johnson, R.E. and Pecora, L.J. (1963). *Physiological Measurements of Metabolic Function in Man.* New York: McGraw-Hill, pp.1-60.

Cooper, K.R. and Phillips, B.A. (1982). Effect of short-term sleep loss on breathing. *J Appl Physiol* **53:** 855-858.

Devoto, A. (1999). Effects of different sleep reductions on daytime sleepiness. *Sleep* **22:** 336-343.

Dinges, D.F., Pack, F., Williams, K., Gillen, K.A., Powell, J.W., Ott, G.E., Aptowicz, C. and Pack, A.I. (1997). Cumulative sleepiness, mood disturbance, psychomotor vigilance performance decrements during a week of sleep restricted to 4-5 hours per night. *Sleep* **20:** 267-277.

Endo, T., Roth, C., Landolt, H., Werth, E., Aeschbach, D., Achermann, P. and Borbély, A. (1998). Selective REM sleep deprivation in humans: effects on sleep and sleep EEG. *Am J Physiol Regul Integr Comp Physiol* **274:** R1186-R1194.

Forest, G. and Godbout, R. (2000). Effects of sleep deprivation on performance and EEG spectral analysis in young adults. *Brain Cogn* **43:** 195-200.

Goldberg, D. (1992). General Health Questionnaire (GHQ-12). Windsor, UK: NFER-Nelson.

Goodman, J., Radomski, M., Hart, L., Plyley, M. and Shephard, R.J. (1989). Maximal aerobic exercise following prolonged sleep deprivation. *Int J Sports Med* **10:** 419-423.

Hill, D.W., Borden, D.O., Darnaby, K.M. and Hendricks, D.N. (1994). Aerobic and anaerobic contributions to exhaustive high-intensity exercise after sleep deprivation. *J Sports Sci* **12:** 455-461.

Holland, G.J. (1968). Effects of limited sleep deprivation on performance of self-selected motor tasks. *Res Q* **39:** 285-294.

Horne, J.A. (1978). A review of the biological effects of total sleep deprivation in man. *Biol Psychol* **7:** 55-102.

Horne, J.A. and Pettitt, A.N. (1984). Sleep deprivation and the physiological response to exercise under steady-state conditions in untrained subjects. *Sleep* **7:** 168-179.

Horne, J.A. (1985). Chronic sleep reduction: daytime vigilance performance and EEG measures of sleepiness with particular reference to practice effects. *Psychophysiology.* **22:** 69-78.

Horne, J.A. (1992). *Why we sleep: the function of sleep in humans and other animals*. Oxford University press, London, New York.

Howlett, T.A. (1998). The endocrinology of exercise. In: *Oxford Textbook of Sport Medicine (2nd ed)*. Harries, M., Williams, C., Stanish, W.D. and Micheli, L.J. (Eds), Oxford University Press, USA, pp. 276-282.

Jasper, H.H. (1958). The ten-twenty electrode placement system of the International Federation. *Electroencephal Clin Neurophysiol*, **10:** 418-24.

Jewett, M.E., Dijk, D.J., Kronauer, R.E. and Dinges, D.F. (1999). Dose-response relationship between sleep duration and human psychomotor vigilance and subjective alertness. *Sleep* **22:** 171-179.

Koslowsky, M. and Babkoff H. (1992). Meta-analysis of the relationship between total sleep deprivation and performance. *Chronobiol Int* **9:** 132-136.

Lane, A.M. and Wilson, M. (2011). Emotions and trait emotional intelligence among ultra-endurance runners. *J Sci Med Sport* **14(4):** 358-362.

Leeder, J., Glaister, M., Pizzoferro, K.,Dawson, J. and Pedlar, C. (2012). Sleep duration and quality in elite athletes measured using wristwatch actigraphy. *J Sports Sci* **30:** 541-545.

Leproult, R. (1997). Sleep loss results in an elevation of cortisol levels the next evening. *Sleep* **20:** 865-870.

Leproult, R., Colecchia, E.F., Berardi, A.M., Stickgold, R., Kosslyn, S.M., and Van Cauter, E. (2002). Individual differences in subjective and objective alertness during sleep deprivation are stable and unrelated. *Am J Physiol Regul Integr Comp Physiol* **284:** R280-R290.

Lucas, S.J., Anson, J.G., Palmer, C., Hellemans, L.J. and Cotter, J.D. (2009). The impact of 100 hours of exercise and sleep deprivation on cognitive function and physical capacities. *J Sports Sci* **27(7):** 719-728.

Luger, A., Watschinger, B., Deuster, P., Svoboda, T., Clodi, M., and Chrousos, G.P. (1992). Plasma growth hormone and prolactin responses to graded levels of acute exercise and to a lactate infusion. *Neuroendocrinology* **56:** 112-117.

Maldonado, C.C., Bentley, A.J. and Mitchell, D (2004). A pictorial sleepiness scale based on cartoon faces. *Sleep* **27:** 541-548.

Martin, B. (1981). Effect of sleep deprivation on tolerance of prolonged exercise. *Eur J Appl Physiol Occup Physiol* **47:** 345-354.

Martin, B, and Gaddis, G.M. (1981). Exercise and sleep deprivation. *Med Sci Sports Exerc* **13:** 220-223.

Martin, B. and Haney, R. (1982). Workload choice after sleep loss. *Eur J Appl Physiol* **49:** 79-86.

Martin, B (1988). Sleep loss and subsequent exercise performance. *Acta Physiol Scand Suppl* **574:** 28-32.

McMurray, R. and Brown, C (1984). The effect of sleep loss on high intensity exercise and recovery. *Aviat Space Environ Med* **55:** 1031-1035.

McMurray, R. and Hackney, A. (2000). Endocrine responses to exercise and training. In: *Exercise and sport science.* Garrett, W.E. and Kirkendall, D.T. (Eds). Lippincott, Williams & Wilkins, Philadelphia, pp. 135-162.

McNair, D.M., Lorr, M. and Droppleman, L.F. (1971). *Manual* for the *Profile of Mood States*. Educational and Industrial Testing Service, San Diego, CA.

Meney, I., Waterhouse, J., Atkinson, G., Reilly, T. And Davenne, D. (1998). The effect of one night's sleep deprivation on temperature, mood, and physical performance in subjects with different amounts of habitual physical activity. *Chronobiol Int* **15:** 349-363.

Mougin, F., Simon-Rigaud, M., Davenne, D., Renaud, A., Garnier, A., Kantelip, J.P. and Magnin, P. (1991). Effects of sleep disturbances on subsequent physical performance. *Eur J Appl Physiol Occup Physiol* **63:** 77-82.

Mougin, F., Bourdin, H., Simon-Rigaud, M.L., Didier, J.M., Toubin, G. and Kantelip, J.P. (1996). Effects of a selective sleep deprivation on subsequent anaerobic performance. *Int J Sports Med* **17:** 115-119.

Mullington, J.M., Haack, M., Toth, M., Serrador, J.M. and Meier-Ewert, H.K. (2009). Cardiovascular, inflammatory and metabolic consequenses of sleep deprivation. *Prog Cardiovasc Dis* **51:** 294-302.

Myles, W.S. (1985). Sleep deprivation, physical fatigue, and the perception of exercise intensity. *Med Sci Sport Exer* **17:** 580-584.

Norcross, N.C., Guadagnoli, E., Prochaska, J.O. (1984). Structure of the profile of mood states (POMS): two partial replications. *J Clin Psychol* **40:** 1270-1277.

Peschke, E. (2008). Melatonin, endocrine pancreas and diabetes. *J Pineal Res* **44:** 26-40.

Pierce, E.F., McGowan, R.W., Barkett, E. and Fry, R.W. (1993). The effects of an acute bout of sleep on running economy and VO2 max. *J Sports Sci* **11:** 109-112.

Pilcher, J.J. and Huffcutt, A. (1996). Effects of sleep deprivation on performance: a meta-analysis. *Sleep* **19:** 318-326.

Plyley, M.J., Shephard, R.J., Davis, G.M. and Goode, R.C (1987). Sleep deprivation and cardio-respiratory function. Influence of intermittent sub-maximal exercise. *Eur J Appl Physiol Occup Physiol* **56:** 338-344.

Powell, N.B., Schechtman, K.B., Riley, R.W., Li, K., Troell, R. and Guilleminault, C. (2001). The road to danger: the comparative risks of driving while sleepy. *Laryngoscope* **111:** 887-893.

Pritzlaff, C.J., Wideman, L., Weltman, J.Y., Abbott, R.D., Gutgesell, M.E., Hartman, M.L., Veldhuis, J.D. and Weltman, A. (1999). Impact of acute exercise intensity on pulsatile growth hormone release in men. *J Appl Physiol* **87:** 498-504

Pritzlaff, C.J., Wideman, L., Weltman, J.Y., Abbott, R.D., Gutgesell, M.E., Hartman, M.L., Veldhuis, J.D. and Weltman, A. (2002). Gender governs the relationship between exercise intensity and growth hormone release in young adults. *J Appl Physiol* **92:** 2053-2060.

Raven, P.B. and Potts, J.T. (1998). Cardiovascular responses to exercise and training. In: *Oxford Textbook of Sport Medicine* (2nd ed). Harries, M., Williams, C., Stanish, W.D. and Micheli, L.J. (Eds), Oxford University Press, USA, pp. 32-44

Rechtschaffen, A. and Kales, A. (1968). A Manual of Standardized Terminology, Techniques and Scoring system for Sleep Stages of Human Subjects. Rechtschaffen, A. and Kales, A. (Eds), UCLA Brain Information Service/Brain Research Institute, Los Angeles, CA.

Reid, A., Maldonado, C.C. and Baker, F.C. (2002). Sleep behaviour of South African adolescents. *Sleep* **25**: 417-421.

Reilly, T. and Walsh, T.J. (1981). Physiological, psychological and performance measures during an endurance record for 5-a-side soccer play. *Brit J Sport Med* **15:** 122-128.

Reilly, T. (1998). Circadian rhythms. In: *Oxford Textbook of Sport Medicine* (2nd ed). Harries, M., Williams, C., Stanish, W.D. and Micheli, L.J. (Eds), Oxford University Press, USA, pp. 231-394.

Reilly, T. and Edwards, B. (2007). Altered sleep-wake cycles and physical performance in athletes. *Physiol Behav* **90:** 274-284.

Reilly, T. and Waterhouse, J. (2009). Sports performance: is there evidence that the body clock plays a role? *Euro J Appl Physiol* **106:** 321-332.

Rowell, L.B. and O'Leary, D.S. (1990). Reflex control of the circulation during exercise: chemoreflexes and mechanoreflexes. *J Appl Physiol* **69:** 407-418.

Sahlin, K. (1998). Acid-base balance during high intensity exercise. In: *Oxford Textbook of Sport Medicine* (2nd ed). Harries, M., Williams, C., Stanish, W.D. and Micheli, L.J. (Eds), Oxford University Press, USA, pp. 23-75.

Sallinen, J., Pakarinen A., Ahtiainen, J., Kraemer, W.J., Volek, J.S. and Hakkinen, K. (2004). Relationship between diet and serum anabolic hormone responses to heavy-resistance exercise in men. *Int J Sports Med* **25:** 627-633.

Scheen, A.J., Buxton, O.M., Jison, M., Van Reeth, O., Leproult, R., L'Hermite-Balériaux, M. and Van Cauter, E. (1998). Effects of exercise on neuroendocrine secretions and glucose regulation at different times of day. *Am J Physiol Endocrinol Metab* **274:** 1040-1049.

Schneider, C., Fulda, S. and Schultz, H. (2004). Daytime variation in performance and tiredness/sleepiness ratings in patients with insomnia, narcolepsy, sleep apnoea and normal controls. *J Sleep Res* **13:** 373-383.

Scott, J. and McNaughton, L. (2004). Sleep deprivation, energy expenditure and cardio-respiratory function. *Int J Sports Med* **25:** 421-426.

Scott, J.P., McNaughton, L.R. and Polman, R.C. (2006). Effects of sleep deprivation and exercise on cognitive, motor performance and mood. *Physiol Behav* **87:** 396-408.

Shapiro, C.M. (1981). Sleep and the athlete. *Brit J of Sport Med* **15:** 51-55.

Speedy, D.B., Noakes, T.D., Rogers, I.R., Thompsn, J.M., Campbell, R.G., Kuttner, J.A., Boswell, D.R., Wright, S. and Hamln, M. (1999). Hyponatremia in ultradistance triathletes. *Med Sci Sport Exer.* **31:** 809-815.

Spengler, C. and Shea, S. (2000). Sleep Deprivation Per Se Does Not Decrease the Hypercapnic Ventilatory Response in Humans. *Am J Resp Crit Care* **161:** 1124-1128.

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

Symons, J.D., VanHelder, T. and Myles, W.S. (1988). Physical performance and physiological responses following 60 hours of sleep deprivation. *Med Sci Sport Exer* **20:** 374-380.

Souissi, N., Gauthier, A., Sesboue, B., Larue, J. and Davenne, D. (2004). Circadian rhythms in two types of anaerobic cycle leg exercise: Force-velocity and 30-s Wingate tests. *Int J Sports Med* **25:** 14-19.

Takeuchi, L., Davis, G.M., Plyley, R., Goode, R. and Shephard, R.J. (1985). Sleep deprivation, chronic exercise and muscle performance. *Ergonomics* **28:** 591-601.

Tilley, A. (1984). The effects of a restricted sleep regime on the composition of sleep and on performance. *Psychophysiology* **21:** 406-411.

Van Cauter, E., Blackman, J.D. and Rolan, D. (1991). Modulation of glucose regulation and insulin secretion by rhythmicity and sleep. *J Clin Invest* **88:** 934-942.

Van Cauter, E., Kerkhofs, M. and Caufriez, A. (1992). A quantitative estimation of GH secretion in normal man: reproducibility and relation to sleep and time of day. *J Clin Endocr Metab* **74:** 1441-1450.

Van Cauter, E., Polonsky, K.S. and Scheen, A.J. (1997). Roles of Circadian Rhythmicity and sleep in human glucose regulation. *Endocr Rev* **18:** 716-738.

Van den Berg, J. and Neely, G. (2006). Performance on a simple reaction time task while sleep deprived. *Percept Motor Skill* **102:** 589-599.

VanHelder, T. and Radomski, M. (1989). Sleep deprivation and the effect on exercise performance. *Sports Med* **7:** 235-247.

Van Dongen, H.P., Maislin, G., Mullington, J.M. and Dinges, D.F. (2003). The cumulative cost of additional wakefulness: Dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. *Sleep* **26:** 117–126.

Van Dongen, H.P. and Dinges, D.F. (2005). Sleep, circadian rhythms, and psychomotor vigilance. *Clin Sports Med* **24:** 237–249.

Vgontzas, A.N., Pejovic, S., Zoumakis, E., Lin, H.M., Bixler, E.O., Basta, M., Fang, J., Sarrigiannidis, A. and Chrousos, G.P. (2007). Daytime napping after a night of sleep loss decreases sleepiness, improves performance, and causes beneficial changes in cortisol and interleukin-6 secretion. *Am J Phisiol Endocrinol Metab* **292:** E253-E261.

Walker, M.P. and Stickgold, R. (2004). Sleep-dependent learning and memory consolidation. *Neuron* **44:** 121–133.

Webb, W. (1975). The effects of subsequent sleep of an acute restriction of sleep length. *Psychophysiology* **12:** 367-370.

Weckowics, T.E. (1978). Review of the Profile of mood states. In: *The eighth mental measurements yearbook*, Buros, O.K. (Ed), Highland Park, NJ: Gryphon, pp. 1018-1019.

Williamson, A.M. and Feyer, A.M. (2000). Moderate sleep deprivation produces impairments in cognitive and motor performance equivalent to legally prescribed levels of alcohol intoxication. *Occup Environ Med* **57:** 649-655.

Wyndham, C.H., Strydom, J.S., Maritz, J.F., Morrison, J. and Potgieter, Z.U. (1959). Maximal oxygen intake and maximum heart rate during strenuous work. *J Appl Physiol* **14:** 927-936.

Zhang, J. (December 2004). Memory process and the function of sleep. *J Theor* **6**.

APPENDICES

GENERAL HEALTH QUESTIONNAIRE

GHQ-60

Please read this carefully:

We should like to know if you have had any medical complaints, and how your health has been in general, *over the past few weeks*. Please answer ALL the questions on the following pages simply by underlining the answer which you think most nearly applies to you. Remember that we want to know about present and recent complaints, not those you had in the past.

It is important that you try to answer ALL the questions.

Thank you very much for your co-operation.

HAVE YOU RECENTLY:

1 — been feeling perfectly well and in good health?	Better than usual	Same as usual	Worse than usual	Much worse than usual
2 — been feeling in need of a good tonic?	Not at all	No more than usual	Rather more than usual	Much more than usual
3 — been feeling run down and out of sorts?	Not at all	No more than usual	Rather more than usual	Much more than usual
4 — felt that you are ill?	Not at all	No more than usual	Rather more than usual	Much more than usual
5 — been getting any pains in your head?	Not at all	No more than usual	Rather more than usual	Much more than usual
6 — been getting a feeling of tightness or pressure in your head?	Not at all	No more than usual	Rather more than usual	Much more than usual
7 — been able to concentrate on whatever you're doing?	Better than usual	Same as usual	Less than usual	Much less than usual
8 — been afraid that you were going to collapse in a public place?	Not at all	No more than usual	Rather more than usual	Much more than usual
9 — been having hot or cold spells?	Not at all	No more than usual	Rather more than usual	Much more than usual
10 — been perspiring (sweating) a lot?	Not at all	No more than usual	Rather more than usual	Much more than usual
11 — found yourself waking early and unable to get back to sleep?	Not at all	No more than usual	Rather more than usual	Much more than usual
12 — been getting up feeling your sleep hasn't refreshed you?	Not at all	No more than usual	Rather more than usual	Much more than usual
13 — been feeling too tired and exhausted even to eat?	Not at all	No more than usual	Rather more than usual	Much more than usual

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HAVE YOU RECENTLY:

14 — lost much sleep over worry?	Not at all	No more than usual	Rather more than usual	Much more than usual
15 — been feeling mentally alert and wide awake?	Better than usual	Same as usual	Less alert than usual	Much less alert
16 — been feeling full of energy?	Better than usual	Same as usual	Less energy than usual	Much less energetic
17 — had difficulty in getting off to sleep?	Not at all	No more than usual	Rather more than usual	Much more than usual
18 — had difficulty in staying asleep once you are off?	Not at all	No more than usual	Rather more than usual	Much more than usual
19 — been having frightening or unpleasant dreams?	Not at all	No more than usual	Rather more than usual	Much more than usual
20 — been having restless, disturbed nights?	Not at all	No more than usual	Rather more than usual	Much more than usual
21 — been managing to keep yourself busy and occupied?	More so than usual	Same as usual	Rather less than usual	Much less than usual
22 — been taking longer over the things you do?	Quicker than usual	Same as usual	Longer than usual	Much longer than usual
23 — tended to lose interest in your ordinary activities?	Not at all	No more than usual	Rather more than usual	Much more than usual
24 — been losing interest in your personal appearance?	Not at all	No more than usual	Rather more than usual	Much more than usual
25 — been taking less trouble with your clothes?	More trouble than usual	About same as usual	Less trouble than usual	Much less trouble
26 — been getting out of the house as much as usual?	More than usual	Same as usual	Less than usual	Much less than usual
27 — been managing as well as most people would in your shoes?	Better than most	About the same	Rather less well	Much less well
28 — felt on the whole you were doing things well?	Better than usual	About the same	Less well than usual	Much less well
29 — been late getting to work, or getting started on your housework?	Not at all	No later than usual	Rather later than usual	Much later than usual
30 — been satisfied with the way you've carried out your task?	More satisfied	About same as usual	Less satisfied than usual	Much less satisfied
31 — been able to feel warmth and affection for those near to you?	Better than usual	About same as usual	Less well than usual	Much less well
32 — been finding it easy to get on with other people?	Better than usual	About same as usual	Less well than usual	Much less well
33 — spent much time chatting with people?	More time than usual	About same as usual	Less than usual	Much less than usual

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HAVE YOU RECENTLY:

34 — kept feeling afraid to say anything to people in case you made a fool of yourself?	Not at all	No more than usual	Rather more than usual	Much more than usual
35 — felt that you are playing a useful part in things?	More so than usual	Same as usual	Less useful than usual	Much less useful
36 — felt capable of making decisions about things?	More so than usual	Same as usual	Less so than usual	Much less capable
37 — felt you're just not able to make a start on anything?	Not at all	No more than usual	Rather more than usual	Much more than usual
38 — felt yourself dreading everything that you have to do?	Not at all	No more than usual	Rather more than usual	Much more than usual
39 — felt constantly under strain?	Not at all	No more than usual	Rather more than usual	Much more than usual
40 — felt you couldn't overcome your difficulties?	Not at all	No more than usual	Rather more than usual	Much more than usual
41 — been finding life a struggle all the time?	Not at all	No more than usual	Rather more than usual	Much more than usual
42 — been able to enjoy your normal day-to-day activities?	More so than usual	Same as usual	Less so than usual	Much less than usual
43 — been taking things hard?	Not at all	No more than usual	Rather more than usual	Much more than usual
44 — been getting edgy and bad-tempered?	Not at all	No more than usual	Rather more than usual	Much more than usual
45 — been getting scared or panicky for no good reason?	Not at all	No more than usual	Rather more than usual	Much more than usual
46 — been able to face up to your problems?	More so than usual	Same as usual	Less able than usual	Much less able
47 — found everything getting on top of you?	Not at all	No more than usual	Rather more than usual	Much more than usual
48 — had the feeling that people were looking at you?	Not at all	No more than usual	Rather more than usual	Much more than usual
49 — been feeling unhappy and depressed?	Not at all	No more than usual	Rather more than usual	Much more than usual
50 — been losing confidence in yourself?	Not at all	No more than usual	Rather more than usual	Much more than usual
51 — been thinking of yourself as a worthless person?	Not at all	No more than usual	Rather more than usual	Much more than usual
52 — felt that life is entirely hopeless?	Not at all	No more than usual	Rather more than usual	Much more than usual
53 — been feeling hopeful about your own future?	More so than usual	About same as usual	Less so than usual	Much less hopeful

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HAVE YOU RECENTLY:

54 — been feeling reasonably happy, all things considered?	More so than usual	About same as usual	Less so than usual	Much less than usual
55 — been feeling nervous and strung-up all the time?	Not at all	No more than usual	Rather more than usual	Much more than usual
56 — felt that life isn't worth living?	Definitely not	No more than usual	Rather more than usual	Much more than usual
57 — thought of the possibility that you might make away with yourself?	Not at all	I don't think so	Has crossed my mind	Definitely have
58 — found at times you couldn't do anything because your nerves were too bad?	Not at all	No more than usual	Rather more than usual	Much more than usual
59 — found yourself wishing you were dead and away from it all?	Not at all	No more than usual	Rather more than usual	Much more than usual
60 — found that the idea of taking your own life kept coming into your mind?	Definitely not	I don't think so	Has crossed my mind	Definitely has

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Profile of Mood States

Subject's Initials _____
Birth date _____
Date _____
Subject Code No. _____

Directions: Describe HOW YOU FEEL RIGHT NOW by checking one space after each of the words listed below:

FEELING	Not at all	A little	Mod.	Quite a bit	Extremely
Friendly	1	2	3	4	5
Tense	1	2	3	4	5
Angry	1	2	3	4	5
Worn Out	1	2	3	4	5
Unhappy	1	2	3	4	5
Clear-headed	1	2	3	4	5
Lively	1	2	3	4	5
Confused	1	2	3	4	5
Sorry for things done	1	2	3	4	5
Shaky	1	2	3	4	5
Listless	1	2	3	4	5
Peeved	1	2	3	4	5
Considerate	1	2	3	4	5
Sad	1	2	3	4	5
Active	1	2	3	4	5
On edge	1	2	3	4	5
Grouchy	1	2	3	4	5
Blue	1	2	3	4	5
Energetic	1	2	3	4	5
Panicky	1	2	3	4	5
Hopeless	1	2	3	4	5
Relaxed	1	2	3	4	5
Unworthy	1	2	3	4	5
Spiteful	1	2	3	4	5
Sympathetic	1	2	3	4	5
Uneasy	1	2	3	4	5
Restless	1	2	3	4	5
Unable to concentrate	1	2	3	4	5
Fatigued	1	2	3	4	5

Helpful	1	2	3	4	5
Annoyed	1	2	3	4	5
Discouraged	1	2	3	4	5
Resentful	1	2	3	4	5
Nervous	1	2	3	4	5
Lonely	1	2	3	4	5
Miserable	1	2	3	4	5
Muddled	1	2	3	4	5
Cheerful	1	2	3	4	5
Bitter	1	2	3	4	5
Exhausted	1	2	3	4	5
Anxious	1	2	3	4	5
Ready to fight	1	2	3	4	5
Good-natured	1	2	3	4	5
Gloomy	1	2	3	4	5
Desperate	1	2	3	4	5
Sluggish	1	2	3	4	5
Rebellious	1	2	3	4	5
Helpless	1	2	3	4	5
Weary	1	2	3	4	5
Bewildered	1	2	3	4	5
Alert	1	2	3	4	5
Deceived	1	2	3	4	5
Furious	1	2	3	4	5
Effacious	1	2	3	4	5
Trusting	1	2	3	4	5
Full of pep	1	2	3	4	5
Bad-tempered	1	2	3	4	5
Worthless	1	2	3	4	5
Forgetful	1	2	3	4	5
Carefree	1	2	3	4	5
Terrified	1	2	3	4	5
Guilty	1	2	3	4	5
Vigorous	1	2	3	4	5
Uncertain about things	1	2	3	4	5
Bushed	1	2	3	4	5