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# THE EFFECTS OF SIMULATED JET LAG ON SLEEP, EXERCISE AND COGNITIVE PERFORMANCE IN MALE ATHLETES

By

**Tamar Shira Goldin**

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*A dissertation submitted to the Faculty of Science, University of the Witwatersrand, Johannesburg, in fulfillment of the requirements for the degree of Master of Science.*

*Johannesburg, 2010*

# DECLARATION

I declare that this dissertation is my own, unaided work. It is being submitted for the Degree of Master of Science in the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination in any other University.

_____

Tamar Shira Goldin

____9th _____ day of _____February_____2010

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# ABSTRACT

Jet lag results in a temporary mismatch between the body's internal clock and local time cues. Travel across time zones negatively alters the sleep-wake cycle, body temperature, psychological status, cognitive performance, and athletic ability of the traveller. I was interested in investigating the impact of a laboratory simulated six hour phase advance shift in the sleep-wake cycle on core body temperature, sleep patterns, mood, cognitive and athletic performance in 10 male athletes (age, 23.8 $\pm$ 3.2 years; Mean $\pm$ SD). The athletes were studied over two randomized weekends; a local time ("home" weekend) and a six hour phase advance shift ("away" weekend). In the six hour phase advance shift ("away") weekend, all sleep-wake times, meal times, exercise tests, and cognitive tests were advanced by six hours. Body temperature, objective sleep parameters, subjective sleepiness, mood states, cognitive performance (reaction time), and athletic performance were measured over both weekends. Physical measures of athletic performance such as muscular strength, abdominal muscular endurance, flexibility, anaerobic ability, hand grip strength, and aerobic performance were measured. On both weekends, the results showed a circadian rhythm in body temperature; however, body temperature rhythm was significantly disrupted after the phase shift ($p<0.05$). After the phase shift, a significant decrease in total sleep time, percentage of rapid eye movement sleep, sleep efficiency, and increased time awake during sleep was observed ($p<0.05$). There was a significant decrease in muscular strength, abdominal muscular endurance, and anaerobic ability after the phase advance shift ($p<0.05$). Simple reaction time, a measure of cognitive performance, was not affected after the phase

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shift ($p>0.05$). The six hour phase shift resulted in altered mood states and elevated subjective daytime sleepiness ($p<0.05$). To conclude, a six hour phase advance shift of the sleep-wake cycle impaired sleep, anaerobic athletic performance and mood which may be problematic for athletes travelling across time zones.

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*In loving memory of my father*

*Dr. Cyril Goldin*

*1948-1999*

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# LIST OF ABBREVIATIONS

| | |
|---|---|
| BP | Blood Pressure |
| EEG | Electroencephalograph |
| EMG | Electromyograph |
| EOG | Electro-oculograph |
| ESQ | Environmental Symptoms Questionnaire |
| GHQ | General Health Questionnaire |
| GMT | Greenwich Mean Time |
| Hb | Haemoglobin |
| HR | Heart Rate |
| MSLT | Multiple Sleep Latency Test |
| MT | Movement Time |
| NREM | Non-Rapid Eye Movement |
| POMS | Profile of Mood States |
| PSG | Polysomnography |
| PSQI | Pittsburgh Sleep Quality Index |
| PVT | Psychomotor Vigilance Task |
| REM | Rapid Eye Movement |
| RER | Respiratory Exchange Ratio |
| RM-ANOVA | Repeated-measures analysis of variance |
| RM | Repetition Maximum |
| RPE | Rate of Perceived Exertion |
| RQ | Respiratory Quotient |

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| | |
|---|---|
| RSP | REM Sleep Propensity |
| RT | Reaction Time |
| SCN | Suprachiasmatic Nucleus |
| SD | Sleep Deprivation |
| SE | Sleep Efficiency |
| SOL | Sleep Onset Latency |
| SWS | Slow Wave Sleep |
| TIB | Time In Bed |
| TMD | Total Mood Disturbance |
| TST | Total Sleep Time |
| TWT | Total Wake Time |
| VAS | Visual Analogue Scale |
| $VCO_2$ | Maximal Carbon Dioxide Expired |
| VE | Ventilation |
| $VO_2max$ | Maximal Oxygen Uptake |

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# CHAPTER ONE

## *Literature Review*

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Exercise is a complex activity that integrates physiological and psychological components which contribute to success in athletic performance. The need for sleep as a restorative function is a vital component for the success of an athlete. Both the sleep-wake cycle and athletic performance exhibit a circadian rhythm and the need to maintain these two processes is of great importance to promote general well-being and success of athletes. However, several components of athletic performance and the sleep-wake cycle (sleep architecture and structure) are highly susceptible to external and internal disruptions such as transmeridian travel which may prove a problem for potential success in the competitive arena.

## 1.1 Exercise Physiology

Athletic performance depends on the integration of physiological (cardiovascular, respiratory, and metabolic systems) and psychological (mood, arousal, physical well-being, motivation, vigour, and fatigue) processes which contribute in different degrees to various types of athletic performance. Several components of athletic performance, such as muscular strength, flexibility, anaerobic power and capacity, aerobic capacity and endurance, speed, agility, and psychological factors, have been identified to optimize success in a selected sport (Lakomy 1994; McArdle *et al*. 2000; Hoffman 2002; Åstrand *et al*. 2003). Thus, the fundamentals of athletic performance stem from the relevance and importance of each component to a particular sport and thus provide a greater understanding of how to maximize and predict potential performance in athletes. Physical performance such as muscular

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strength, anaerobic and aerobic capacity can be measured, however the underlying psychological status and well-being of an athlete contributes to the overall performance.

The psychological status of an athlete such as mood, arousal, concentration, motivation, and fatigue play an important role in determining athletic performance. In order to optimize athletic performance, sport psychology has become a prominent field of research to predict potential performances in athletes. Generally, athletes are highly motivated individuals who display a positive mood; however the expectations to perform optimally at all times are a burden to an athlete, and could decrease athletic performance (Hardy and Jones 1994). Thus, there are significant benefits for a comprehensive, multidisciplinary approach, integrating physical and psychological tests, in evaluating athletic performance. Even though athletes may integrate physical, physiological, and psychological athletic components to produce their best possible performance, additional variables, such as the amount and quality of sleep, may impact on subsequent athletic performance.

## 1.2 Sleep Physiology

*"Sleep is a reversible behavioural state of perpetual disengagement from and unresponsiveness to the environment"* (Carskadon and Dement 2000, p.15).

Sleep is a dynamic, passive process, progressing through multiple stages in a cyclic pattern that represents the daily process of physiological restoration and recovery

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(Dement 2000). Polysomnography (PSG), an objective measure of sleep, uses electroencephalography (EEG), electro-oculography (EOG), and electromyography (EMG) to measure brain activity (EEG), eye movements (EOG), and muscle tone (EMG) during sleep (Moore-Ede *et al*. 1982; Carskadon and Dement 2000; Carskadon and Rechtschaffen 2000). The common terms used to analyze PSG recordings and to distinguish the underlying events of sleep, are summarized in Table 1 (Spriggs 2002).

**Table 1.** *Common terminology used in the analysis of polysomnographic recordings*

| **Common PSG terms** | **Definitions** |
|---|---|
| Total Sleep Period | The duration of time measured from SOL to final awakening, also consisting of the time taken up by arousals and MT |
| Time In Bed (TIB) | The total amount of actual time in bed during a sleep period |
| Total Sleep Time (TST) | The total amount of actual sleep time during a sleep period |
| Sleep Onset Latency (SOL) | The time it takes to fall asleep |
| Total Wake Time (TWT) | The total amount of time spent awake during a sleep period |
| Sleep Efficiency (SE) | Ratio of TST to TIB |
| Movement Time (MT) | The amount of actual body movement, when no distinguishable EEG activity of NREM or REM sleep stages are present for longer than 15 seconds |
| Arousals | A change in the EEG activity and frequency |

EEG- electroencephalography; NREM- non-rapid eye movement; REM- rapid eye movement.

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Sleep can be distinctively divided into two states; non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep that are distinct from each other as each is from wakefulness. NREM sleep is typically subdivided into four stages; stages 1, 2, 3, and 4, representing a progressive increase in the depth of sleep. According to Rechtschaffen and Kales (1968), each sleep stage displays a distinctive EEG, EOG, and EMG pattern. Stages 1 and 2 represent a lighter sleep, characteristic of a transition from wakefulness to sleep. Stage 2 is characteristic of sleep spindles and K complexes. Stages 3 and 4 are referred to as slow-wave sleep (SWS) due to their characteristic low frequency and high voltage EEG activity. REM sleep is characterized by a low voltage, high frequency EEG, muscle atonia, and episodic bursts of eye movements (Rechtschaffen and Kales 1968; Carskadon and Rechtschaffen 2000).

The NREM-REM sleep stages are distributed in cycles throughout the night (Figure 1). The normal adult enters NREM sleep (stages 1 and 2) before progressing rapidly to SWS. Generally, adults spend 2-3% of total sleep time (TST) in stage 1, 40-45% of TST in stage 2, 13-23% of TST in SWS, and 20-25% of TST in REM sleep. SWS dominates the first third of the night, thereafter REM sleep predominates in the latter third of the sleep period. The NREM-REM sleep cycle repeats every 90 to 120 minutes in humans (Carskadon and Dement 1980; Carskadon and Dement 2000; Carskadon and Rechtschaffen 2000).

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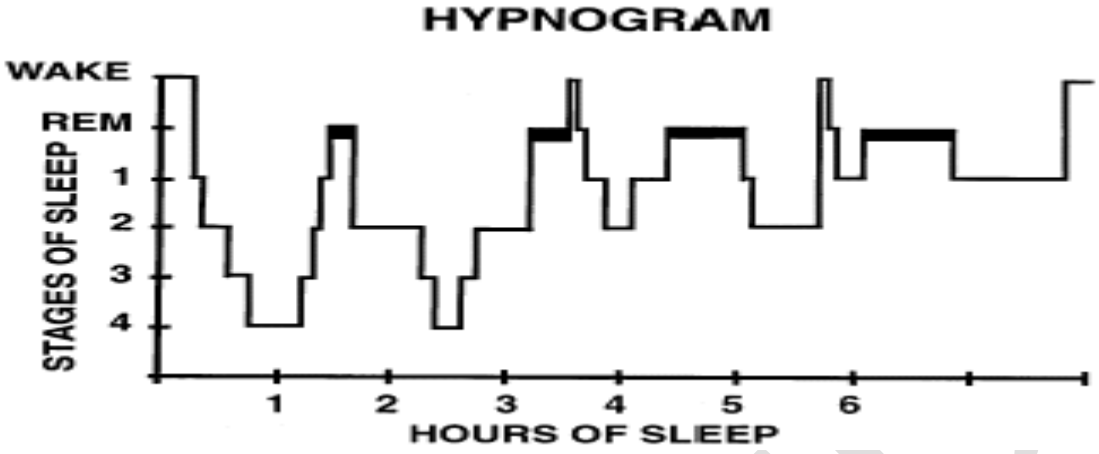



**Figure 1.** Hypnogram showing the progression of sleep stages in the normal young adult. Solid bars represent REM sleep (Strollo 1998).

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Sleep is a highly organized, complex sequence of events that follows a regular, cyclic pattern; however, lack of sleep has far-reaching effects (Moore-Ede *et al*. 1982; Roth and Roehrs 2000). Sleep deprivation studies have shown the negative consequences of sleep loss such as poor judgement, elevated fatigue and daytime sleepiness, and reduced cognitive performance (Bonnet 1985; Dinges *et al*. 1997; Roehrs and Roth 2000; Williamson and Feyer 2000; Phillip *et al*. 2004). Thus, the physiological and psychological need for sleep is a vital component to promote optimal functioning within the workplace, in our daily events, and in the sporting arena. The organization and regulation of sleep, and several other physiological systems, is partly sustained by circadian rhythms.

## 1.3 Circadian Rhythms

At rest, many biological and psychological systems in humans follow a daily, internally driven pattern, called circadian rhythms, which are synchronized with the 24 hour light-dark cycle. The meaning of the words, 'circadian' originates from the Latin words, *circa* ('about') and *dies* ('day'). Consequently, circadian rhythms can be described as a 'cycle of events', whereby maximum and minimum levels occur at specific times of the day. Most of the body's physiological variables, including body temperature, heart rate, hormonal secretions, cognitive performance, sleepiness, and subjective alertness, exhibit a circadian rhythmicity sustained within an approximate 24 hour period (Winget *et al*. 1984; Loat and Rhodes 1989; Atkinson and Reilly 1996; Mistlberger and Rusak 2000; Lack and Wright 2007).

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Most of these biological systems particularly the sleep-wake cycle and hormonal secretions are considered to be completely endogenous in nature; that is, they are controlled internally by the 'biological clock', found in the suprachiasmatic nucleus (SCN), located in the hypothalamus. Most mammals, isolated from environmental time cues, show a characteristic 'free-running' period which is entrained to approximately 24 hours (Mistlberger and Rusak 2000). However, circadian systems are sensitive to environmental time cues (exogenous variables) including photic (light-dark cycle) and non-photic (food availability, social interaction, physical activity, and ambient temperature) external time cues which are termed Zeitgebers ('time-givers') (Moore-Ede *et al*. 1982; Shephard 1984; Duffy *et al*. 1996; Mistlberger and Rusak 2000).

In summary, circadian rhythms can be defined by the following characteristics (Shephard 1984; Loat and Rhodes 1989):

1. The rhythm persists in the absence of external cues (darkness or meals) with a periodicity of 24 to 26 hours.
2. The rhythm persists in the presence of shifts of environmental cues (shift work or transmeridian travel across multiple time zones).
3. The rhythm persists in the presence of false time cues (clocks operating on a 21 or 27 hour cycle).

The most distinctive circadian rhythm is our daily cycle of sleep and wakefulness which is partly influenced by the endogenous body temperature circadian rhythm.

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### 1.3.1 Body Temperature and Sleep-Wake Cycle

Body temperature follows a distinct circadian rhythm, peaking in the early evening and reaching a nadir, or a trough, in the early morning (Reilly 1994). These fluctuations in core body temperature are self-sustained even in the absence of environmental time cues; thus measurement of core body temperature provides a good predictor of the phase of circadian rhythm (Monk *et al*. 1997). Several physiological variables such as cognitive performance, alertness, sleep and wakefulness and more recently, athletic performance, are coupled to the body temperature circadian rhythm (Reilly 1994; Van Dongen and Dinges 2000; Kline *et al*. 2007). Furthermore, the timing of sleep and the internal structure of sleep are closely related to the body temperature rhythm (Czeisler *et al*. 1980a; Czeisler *et al*. 1980b; Moore-Ede *et al*. 1982; Lack and Lushington 1996; Murphy and Campbell 1997; Czeisler and Khalsa 2000; Glotzbach and Heller 2000).

Sleep propensity (need to sleep) or subjective ratings of sleepiness are highest in the late evening, which coincides with a drop in body temperature and increase in melatonin secretion (Lack and Lushington 1996; Murphy and Campbell 1997). The tendency to fall asleep during the day is greatest at two specific times of the day; between 04:00-05:00 and 15:30-16:30, with the latter termed the 'post lunch dip' (Roehrs *et al*. 1996; Van Dongen and Dinges 2000; Monk 2005).

Roehrs *et al*. (1996) conducted a multiple sleep latency test (MSLT) throughout the waking period and showed that the greatest level of sleepiness (shortest SOL) was

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apparent in the late afternoon. Thus, sleep propensity follows a circadian rhythm, fluctuating during the day, with minimum and maximum sleep propensity occurring in the morning on the ascending limb of the temperature circadian rhythm and in the late evening on the descending limb of the temperature circadian rhythm, respectively (Graeber 1994; Lack and Lushington 1996; Murphy and Campbell 1997). Besides determining sleep propensity, the temperature circadian rhythm has been shown to regulate sleep architecture and structure.

Once asleep, the timing of the internal structure of the NREM-REM cycle has been shown to correlate with the circadian body temperature rhythm (Czeisler *et al*. 1980a; Czeisler *et al*. 1980b; Zulley 1980; Moore-Ede *et al*. 1982; Borbély and Achermann 2000). A study conducted by Czeisler *et al*. (1980b) demonstrated that the timing of REM sleep is controlled by an endogenous circadian oscillator which is coupled to the 24 hour body temperature circadian rhythm. Thus, the peak in REM sleep propensity (RSP) occurred on the ascending limb of the body temperature rhythm, coinciding with the phase of peak sleep tendency.

Despite the close relationship between sleep and body temperature circadian rhythm, the sleep-wake cycle and sleep structure is actually controlled by two mechanisms; the homeostatic drive for sleep (Process S) and the circadian process (Process C) (Borbély 1982; Dijk and Czeisler 1995; Borbély and Achermann 2000). The circadian process (Process C) represents the 24 hour body temperature circadian rhythm. The homeostatic drive for sleep (Process S) is a need for sleep that

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accumulates during the wakeful period (daytime) and declines during the sleep period. The timing and duration of sleep is therefore determined by the independence and the interaction of the homeostatic drive for sleep and the circadian system (Figure 2) (Borbély and Achermann 2000). The greatest tendency to sleep for Process C occurs during the trough of body temperature. The optimal time for sleep occurs when Process S is at its peak and Process C is at its trough, coinciding with the typical nighttime sleep period. Awakening from sleep typically occurs when Process C and Process S intersect (Figure 2) (Borbély and Achermann 2000). Furthermore, the interaction between the two processes has been shown to modulate neurobehavioural performances such as alertness, fatigue, and cognitive functioning (Daan *et al*. 1984; Van Dongen and Dinges 2003).

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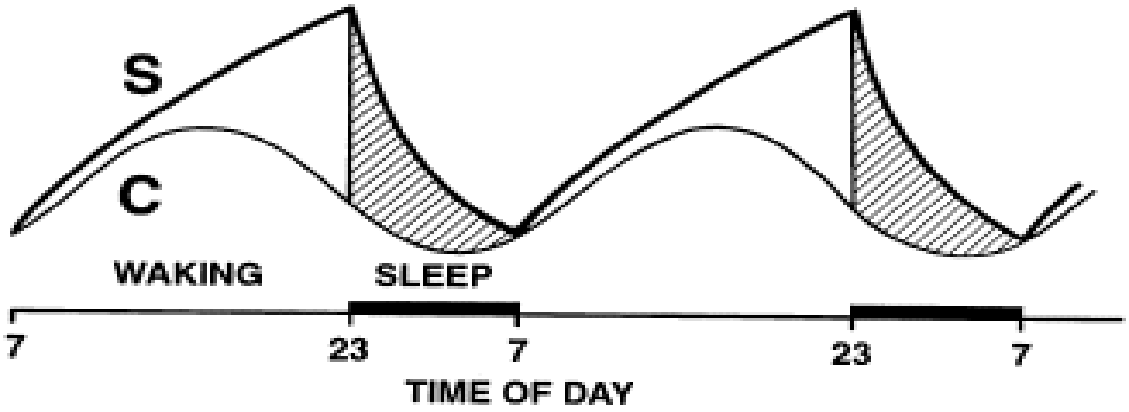



**Figure 2.** Two-process model of sleep regulation. Process S represents the homeostatic drive for sleep, which rises during the wakeful period (daytime) and declines during the sleep period. Process C represents the circadian system as measured by body temperature. The shaded area represents the optimal time for sleep (Borbély and Achermann 2000).

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### 1.3.2 Neurobehavioural Functions

Neurobehavioural functions such as alertness, cognitive performance and fatigue exhibit an endogenous circadian rhythm closely related to the body temperature rhythm, which reach a minimum after the trough in body temperature (Czeisler *et al*. 1980a; Dijk *et al*. 1992; Johnson *et al*. 1992; Monk *et al*. 1997; Czeisler and Khalsa 2000; Roehrs *et al*. 2000; Van Dongen and Dinges 2000; Wright *et al*. 2002). In addition to the circadian component, neurobehavioural measures such as subjective alertness and performance are highly vulnerable to the duration of prior nocturnal sleep and wakefulness (Bonnet 1985; Roehrs *et al*. 2000; Van Dongen and Dinges 2000; Wright *et al*. 2002; Van Dongen and Dinges 2005). As seen in sleep derived individuals, increased pressure for sleep (increased homeostatic drive for sleep) contributes to decrements in subjective alertness and performance (Van Dongen and Dinges 2000; Williamson and Feyer 2000; Van Dongen and Dinges 2005).

The maintenance of circadian rhythms for alertness, fatigue, and cognitive performance are complex (Van Dongen and Dinges 2003). Dijk *et al*. (1992) investigated the circadian rhythm in subjective alertness and cognitive performance and showed that the circadian rhythm for neurobehavioural functions was influenced both by the interaction between the circadian drive for wakefulness (Process C), homeostatic drive for sleep (Process S), as well as several endogenous and exogenous masking factors. These masking factors include demand characteristics of

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the experiment, distractions by irrelevant stimuli, boredom and motivational factors, stress, food intake, and posture (Van Dongen and Dinges 2000).

Several techniques such as subjective measures of sleepiness and mood have been employed to detect a circadian rhythmicity in neurobehavioural variables (McNair *et al*. 1971; Mitler *et al*. 2000). Alternatively, many studies have employed objective measures of performance such as simple and choice reaction times, sorting or memory tasks and psychomotor vigilance to assess circadian rhythmicity (Dinges *et al*. 1997; Van Dongen and Dinges 2000).

In the case of sleep-wake rhythm disorders (advanced phase syndrome) or circadian rhythm disruptions (transmeridian travel), the relationship between the homeostatic and circadian processes is important to understand the changes in neurobehavioural functions.

### 1.3.3 Athletic Performance

The impact of circadian rhythms on athletic performance may also be quite profound. Evaluation of various sport-specific physical components of athletic performance such as muscular strength, flexibility, aerobic endurance and capacity, anaerobic power and capacity, all combine to contribute to the success of an athlete (Hoffman 2002). At rest, several components of athletic performance such as physiological functions (heart rate, oxygen consumption, and ventilation rate), psychological functions (mood, arousal, and motivation), metabolic functions (body temperature),

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and behavioural functions (reaction time, hand-eye coordination) follow a daily rhythmic pattern (Winget *et al*. 1984; Drust *et al*. 2005). It would thus be expected that athletic performance "exhibits a rhythmic variation during the day" (Manfredini *et al*. 1998).

The study of the circadian variation in athletic performance has been extensively investigated for many decades. Many researchers agree that peak athletic performance or 'peak performance window', occurs in the early evening, coinciding with the peak of body temperature rhythm (Shephard 1984; Reilly 1994; Youngstedt and O'Connor 1999; Drust *et al*. 2005). Overall, athletic performance has been shown to improve over the course of the day. This peak in performance has been shown in studies that have utilized 'controlled' protocols or simulated competitions in competitive cycling (Reilly and Baxter 1983; Atkinson and Reilly 1995; Atkinson *et al*. 2005), and swimming performance (Rodahl *et al*. 1976; Baxter and Reilly 1983; Kline *et al*. 2007) during the day. Overall performance is dependent on the individual contributing components of athletic performance such as muscular strength, flexibility, anaerobic and aerobic capacity, which may also follow the same circadian rhythm.

Individual components of athletic performance exhibit a circadian rhythm similar to that of overall performance, also peaking in the afternoon or early evening. Muscular strength has been shown to peak in the early evening, irrespective of the type of muscle group tested or speed of muscle contraction. Elbow flexion strength

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(Coldwells *et al*. 1994; Callard *et al*. 2000), isometric grip strength (Gifford 1987), back strength (Coldwells *et al*. 1994), knee flexor strength (Bambaeichi *et al*. 2005), and isokinetic muscular leg strength (Coldwells *et al*. 1994; Wyse *et al*. 1994; Giacomoni *et al*. 2005) were all highest in the early evening. Controlled laboratory investigations have shown that other components of athletic performance such as joint flexibility (Baxter and Reilly 1983; Gifford 1987), joint stiffness (Reilly 1994; Atkinson and Reilly 1996; Drust *et al*. 2005), and ratings of perceived exertion (Atkinson and Reilly 1995; Martin *et al*. 2001) also show a circadian rhythm. Circadian rhythms have been reported for anaerobic power output and anaerobic capacity (Hill and Smith 1991a; Hill and Smith 1991b; Hill *et al*. 1992; Souissi *et al*. 2004); athletes recorded their best standing broad jump (Reilly and Down 1992) and multiple jump performances (Bernard *et al*. 1998) in the early evening when body temperature was highest.

In contrast, investigators have shown that shoulder and ankle flexibility (Baxter and Reilly 1983), grip strength (Baxter and Reilly 1983), anaerobic power and anaerobic output (Reilly and Down 1992), and running and cycling sprint performances (Bernard *et al*. 1998; Falgairette *et al*. 2003; Racinais *et al*. 2005) do not follow a circadian rhythm. Similarly, physiological responses to sub-maximal or maximal aerobic exercise, such as increased heart rate, oxygen uptake, minute ventilation and expired carbon dioxide, do not display a circadian rhythm (Martin *et al*. 2001). Therefore, sports performance is independent on the time of day and the body

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temperature rhythm and thus the best performance can occur irrespective of the time of day.

In summary, circadian rhythms are regulated, in part, by both endogenous and exogenous factors with a periodicity of 24 hours. Several rhythms such as sleep-wake cycle and athletic performance have been reported to closely follow the circadian rhythm of body temperature. Neurobehavioural functions such as alertness and cognitive performance, and more recently athletic performance, have been shown to peak in the early evening, coinciding with the body temperature maximum.

There is a wide range of research data suggesting that an endogenous component accounts for the circadian rhythmicity in athletic performance. Many components of athletic performance exhibit a circadian rhythm in parallel to the circadian body temperature rhythm, including muscle strength, anaerobic power output, joint flexibility and stiffness, and explosive tests. This temperature-dependent relationship supports the findings that maximum performance is attained in the late afternoon or early evening, coinciding with the peak of the body temperature rhythm.

Thus, since neurobehavioural functions such as subjective alertness, fatigue, and cognitive performance, and athletic performance peak in the early evening with an increase in body temperature rhythm, it is important to consider conditions which may interfere with the body temperature circadian rhythms.

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## 1.4 Disturbances in Circadian Rhythms

Circadian rhythm disturbances occur when the timing of the sleep-wake cycle is out of phase with the internal or external environment (Turek 2000). The disturbance may be either chronic or transient. Chronic phase disorders, such as occurs in delayed or advanced phase syndrome, occur as result of a failure of the 'body clock'; consequently the timing of sleep occurs at abnormal times of the day or night (Mahowald and Schenck 2005; Cardinali *et al*. 2006; Lack and Wright 2007). In contrast, transient phase disorders occur when environmental cues such as light-dark cycles or social cues do not correspond with established circadian rhythms (Mahowald and Schenck 2005; Lack and Wright 2007). Common causes of transient phase disorders are jet lag, shift-work or illness (Mahowald and Schenck 2005; Lack and Wright 2007).

In today's society, a great number of people are exposed to transient circadian disturbances as a result of rotational shift-work or travelling across time zones (Åkerstedt 2003). In turn, travellers and shift-workers experience common symptoms as a result of the slow adjustment of the 'body clock' to the new time cues. A temporary disturbance in circadian rhythm leads to altered sleep-wake cycles (Lowden and Åkerstedt 1999; Jamieson *et al*. 2001; Takahashi *et al*. 2002), elevated daytime sleepiness (Wright *et al*. 1983; Lowden and Åkerstedt 1999), fatigue (Wright *et al*. 1983; Waterhouse *et al*. 2000; Waterhouse *et al*. 2002; Waterhouse *et al*. 2003), and reduced alertness (Lowden and Åkerstedt 1999), making it difficult to

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function at one's maximum capacity or sustain a normal daily routine. Subsequently, transient circadian disturbances may hamper productivity in the working environment or impair athletic performances in competitive events. I was particularly interested in the changes that may occur in athletic performance due to rapid time zone traversal, a phenomenon, known as jet lag

## 1.5 Jet lag

### 1.5.1 Introduction

The primary function of circadian rhythms is to ensure that behavioural and physiological systems are appropriately timed with respect to daily events in the environment. Thus, a close relationship is maintained between exogenous Zeitgebers (light-dark cycle or social cues) and the internally driven physiological rhythms (body temperature or sleep-wake cycle) (Winget *et al*. 1984). Following transmeridian travel across many time zones, a temporary mismatch between the body's circadian rhythms and the local Zeitgebers occurs, commonly referred to as 'jet lag' (Atkinson and Reilly 1996; Meir 2002). Thus, jet lag symptoms arise when the body's circadian rhythms are still aligned with the circadian timing of the 'home' environment instead of local Zeitgebers (Reilly *et al*. 1997).

The common symptoms of jet lag include sleep disturbances (insomnia, premature awakenings, inability to initiate or maintain sleep), impaired performance, loss of motivation or concentration, somatic problems (gastrointestinal disturbance,

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headaches, irritability, anorexia) and poor daytime functioning (Klein *et al*. 1972; Preston *et al*. 1973a; Preston *et al*. 1973b; Wright *et al*. 1983; Loat and Rhodes 1989; O'Connor *et al*. 1991; Haugli *et al*. 1994; Reilly *et al*. 1997; Lowden and Åkerstedt 1998; Lowden and Åkerstedt 1999; Arendt *et al*. 2000; Daurat *et al*. 2000; Edwards *et al*. 2000; Waterhouse *et al*. 2000; Jamieson *et al*. 2001; Lagarde *et al*. 2001; Takahashi *et al*. 2002; Waterhouse *et al*. 2002; Waterhouse *et al*. 2003; Beaumont *et al*. 2004). The disruption in the internal 'body clock' persists for several days, after arrival, until resynchronization to the new local time cues are complete.

Several field studies have provided valuable information on the impact of transmeridian travel on physiological, behavioural, and psychological variables. However, air travel is time-consuming and expensive, and environmental variables such as exposure to daylight or social contact are difficult to assess and standardize. Also, the sleeping environment (hotel conditions, noise), timing of the flight, and the flight itself (uncomfortable seating, lack of sleep) may further aggravate the negative consequences of jet lag. Thus, controlled laboratory simulations provide an alternative method to study behavioural and physiological changes that occur following transmeridian travel. Laboratory simulated phase shifts allow for the objective measurement of sleep patterns (PSG), without confounding variables such as travel fatigue and hotel environment that could negatively impact on the results. Several investigators have developed laboratory models to mimic phase shifts that occur after transmeridian travel across several time zones (Elliott *et al*. 1972; Preston *et al*. 1973a; Mills *et al*. 1978; Hume 1980; Monk *et al*. 1988; Moline *et al*. 1992;

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Monk *et al*. 1993; Carrier *et al*. 1996; Deacon and Arendt 1996; Roehrs *et al*. 1996; Caufriez *et al*. 2002).

Most of these researchers have mimicked phase shifts associated with transmeridian travel by adjusting sleep-wake cycles and meal times. The protocol involves confining subjects into an isolation unit for a period of time. However, laboratory simulated phase shifts may not reflect the true circadian rhythm readjustment as seen after transmeridian travel. The isolated protocol prevents subjects being exposed to a wide range of environmental cues such as social interaction or exposure to natural daylight that may promote rapid adjustment of circadian systems. Thus, another method of simulating jet lag is the use of evening and morning exposure to bright light to produce a phase delay or phase advance (Minors *et al*. 1991; Gronfier *et al*. 2007). Deacon *et al*. (1996) developed a model to successfully mimic nine-hour phase shifts, similar to those that occur after transmeridian flight, by exposing subjects to different intensities of light at different times of the day, without isolating the subjects. However, both methods of laboratory simulated time zone transitions lack the stressors and sleep deficits associated with flight. Nevertheless, laboratory simulated phase shifts successfully cause a circadian desynchronization similar to that which occurs after travelling across time zones.

### 1.5.2 Severity of Circadian Desynchronization

An overview of field and simulated laboratory studies has generally confirmed that the severity of circadian desynchronization associated with transmeridian travel is

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influenced by multiple factors such as direction of travel, number of time zones crossed, physiological systems measured, individual variability and strength of local Zeitgebers.

#### *1.5.2.1 Direction of Travel*

The severity of circadian desynchronization associated with travel across time zones, and thus rate of resynchronization, is influenced by the direction of travel. Typically, resynchronization after transmeridian travel follows an *orthodromic response*, whereby physiological systems adapt in the same direction as the phase shift (Edwards *et al*. 2000). Thus, adaptation to either westward or eastward travel requires a phase delay or phase advance, respectively. However, several field studies that investigated eastward travel across eight to ten time zones (Gundel and Wegmann 1987; Edwards *et al*. 2000; Takahashi *et al*. 2002) as well as simulated phase advances (Mills *et al*. 1978; Wever 1980; Moline *et al*. 1992; Deacon and Arendt 1996) have shown that physiological systems adapted in reverse to the direction of travel or phase shift, known as an *antidromic response*.

It would seem logical that resynchronization of physiological systems would occur equally rapidly after westward or eastward travel over the same number of time zones. However, it has been well documented in field studies (Elliott *et al*. 1972; Klein *et al*. 1972; Gundel and Wegmann 1987; O'Connor *et al*. 1991; Hill *et al*. 1993; Suvanto *et al*. 1993a; Suvanto *et al*. 1993b; Lowden and Åkerstedt 1999; Takahashi *et al*. 2002) and simulated phase shifts (Hume 1980; Deacon and Arendt

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1996), that adaptation is significantly quicker after westward travel when compared to eastward travel. For example, Klein *et al*. (1972) showed that following eastward or westward travel across six time zones in both directions, complete resynchronization of the body temperature rhythm occurred within eight days and five days, respectively.

The difference in the rate of readjustment after east or west travel across time zones is influenced by the properties of the endogenous circadian system and the direction of travel (Winget *et al*. 1984; Loat and Rhodes 1989; O'Connor and Morgan 1990; Haimov and Arendt 1999). Typically, travelling in an eastward direction shortens the day, decreasing the homeostatic need for sleep (Process S) and thus results in difficulty falling asleep. Thus, eastward travel requires readjustment by a phase advance, forcing a condensing of the period of the circadian rhythm to be less than 24 hours. On the other hand, after westward travel, the day is lengthened, increasing Process S and thus promoting sleep. Thus, readjustment after westward travel requires a phase delay, meaning that the period of the circadian rhythm would have increased beyond 24 hours. As the 'free-running' periods tend to be longer than 24 hours, adaptation of rhythms is better suited to phase delaying rather than phase advancing. However, the extent of desynchronization created by the direction of travel will be further exaggerated by the number of time zones travelled (Moore-Ede *et al*. 1982; Haimov and Arendt 1999).

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#### *1.5.2.2 Number of Time Zones Crossed*

After crossing more than three time zones, a shift in physiological variables, leading to undesirable symptoms of jet lag, becomes significant (O'Connor and Morgan 1990; Reilly and Edwards 2007). In general, physiological readjustment to the new local time cues requires one day per time zone crossed (Aschoff *et al*. 1975; Wever 1980; Youngstedt and O'Connor 1999). However, physiological systems readjust to the new time zone at differing rates.

#### *1.5.2.3 Physiological Systems Measured*

A temporary phase shift associated with transmeridian travel, has been documented in most of the body's circadian rhythms, such as body temperature (Hauty 1967; Klein *et al*. 1972; Gundel and Wegmann 1987; Suvanto *et al*. 1993a; Daurat *et al*. 2000; Edwards *et al*. 2000; Lemmer *et al*. 2002; Waterhouse *et al*. 2002; Beaumont *et al*. 2004), hormonal levels (Désir *et al*. 1981; Golstein *et al*. 1983; Lemmer *et al*. 2002; Bullock *et al*. 2007), cardiovascular parameters (Lemmer *et al*. 2002; Tateishi and Fujishiro 2002), gastrointestinal function (Waterhouse *et al*. 2005), and sleep patterns (O'Connor *et al*. 1991; Hill *et al*. 1993; Lowden and Åkerstedt 1999; Daurat *et al*. 2000; Edwards *et al*. 2000; Waterhouse *et al*. 2000; Jameison *et al*. 2001; Takahashi *et al*. 2002; Waterhouse *et al*. 2002; Waterhouse *et al*. 2003; Beaumont *et al*. 2004).

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The rate of resynchronization differs among physiological systems (Elliott *et al*. 1972; Winget *et al*. 1984). Certain physiological variables adjust more rapidly to a time zone change, whereas other variables take longer to adjust. For example, in one study, body temperature returned to baseline rhythm after two days, while other physiological functions, such as sleeping patterns, mood and performance variables, took five days to adjust after a simulated phase shift of nine hours in an eastwardly direction (Deacon and Arendt 1996). Similarly, Elliott *et al*. (1972) observed that body temperature and urinary excretions of sodium and chloride adapted more readily than urinary excretion of potassium and plasma steroids after simulated exposure of 23 participants to six or eight hour phase shifts. While group effects have been shown in both the symptoms and physiology of jet lag, the individual response and susceptibility to travel across time zones has to be taken into account.

#### *1.5.2.4 Individual Variability*

Several field studies (Elliott *et al*. 1972; O'Connor *et al*. 1991; Lowden and Åkerstedt 1999; Waterhouse *et al*. 2002) and simulated phase shifts (Monk *et al*. 1988; Carrier *et al*. 1996; Deacon and Arendt 1996; Roehrs *et al*. 1996) have concluded that the course of adaptation to a time zone shift varies between individuals. Waterhouse *et al*. (2002) suggested a number of important factors such as gender, age, personality chronotype ('lark' versus 'owl'), physical fitness, previous travel experience, rhythm amplitudes, rhythm stability, exposure to local Zeitgebers (daylight or social contact), and sleeping habits which may all have a significant influence on the degree of individual desynchronization after

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transmeridian travel. For example, the degree of circadian desynchronization may be more persistent in 'larks' (morning people) compared to 'owls' (evening people), as 'larks' have larger body temperature amplitudes and are more susceptible to phase shifts (Winget *et al*. 1984; Suvanto *et al*. 1993b; Graeber 1994). Furthermore, Klein *et al*. (1972) demonstrated that limited exposure to outdoor activities and social contact by isolating subjects to hotel rooms, similar to isolation in laboratory simulated phase shifts, further delayed the resynchronization of systems after transmeridian travel across six time zones. The ability of the individual to cope with the degree of circadian desynchronization after transmeridian travel may also be influenced by the strength of local Zeitgebers.

#### *1.5.2.5 Strength of Local Zeitgebers*

Suvanto *et al*. (1993b) assessed the salivary melatonin circadian rhythm in 40 female flight attendants after bi-directional flights across 10 time zones. The study reported that adaptation after transmeridian travel is faster during summer than during winter due to the increased exposure to natural daylight. Thus, the amount of daylight exposure is a strong predictor for readjustment after transmeridian travel (Winget *et al*. 1984; Suvanto *et al*. 1993b; Duffy *et al*. 1996)

In summary, the dissociation between the endogenous components of the rhythm (body temperature) with the exogenous Zeitgebers (light-dark cycle) is apparent after transmeridian travel across multiple time zones. The temporary circadian

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desynchronization after transmeridian travel leads to undesirable symptoms (sleep disruptions, digestive problems, and general malaise), with worse symptoms reported after eastward travel, compared to westward travel. Various methods can be used to simulate 'experimental' jet lag, including timed exposure to bright light or adjustment of sleep-wake cycles. Many factors such as direction of travel, number of time zones crossed, physiological systems measured, individual variability, and strength of local Zeitgebers influence the severity of circadian desynchronization which to some degree contribute to the negative consequences of transmeridian travel.

### 1.5.3 The Consequences of Transmeridian Travel for Athletes

Several investigators have suggested that the desynchronization of the 'body clock' following transmeridian travel contributes to a "transient deterioration in sleep and mood" (Meir 2002), fatigue (Hill *et al*. 1993; Atkinson and Reilly 1996), and a more recently described decrement in athletic performance (Wright *et al*. 1983; Hill *et al*. 1993; Jehue *et al*. 1993; Recht *et al*. 1995; Steenland and Deddens 1997; Lagarde *et al*. 2001; Lemmer *et al*. 2002). Therefore, I was particularly interested in the relationship between sleep, mood, cognitive performance, and general athletic performance after a phase shift across several time zones.

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#### *1.5.3.1 Altered Sleep-Wake Cycle*

As mentioned previously, transmeridian travel causes a transient phase shift in many physiological rhythms including body temperature, endocrine, cardiovascular and renal functions (Suvanto *et al*. 1993a; Daurat *et al*. 2000; Lemmer *et al*. 2002; Beaumont *et al*. 2004). However, the most common consequence of transmeridian travel is a disturbance of the sleep-wake cycle which may lead to sleep deprivation, fragmented sleep patterns, premature awakenings, altered mood, or excessive daytime sleepiness or fatigue.

The transient sleep problems associated with transmeridian travel are partly due to the time at which sleep is scheduled in relation to the circadian body temperature rhythm (Shephard 1984; Graeber 1994; Haimov and Arendt 1999). As represented in Figure 3, immediately after a time-zone transition, the rhythm has not adjusted and is inappropriately phased. In Hong Kong (8 hour eastward shift), the traveller will have difficulty in getting to sleep at the new night-time (temperature high) and will feel tired, particularly in the morning (temperature low). In general, after an eastward flight, sleep is scheduled before the travellers' 'home' bedtime, and near the peak of the circadian body temperature rhythm. Thus, after eastward flight, travellers' will find it difficult to fall asleep, have more awakenings in the early part of the night, and have decreased REM sleep (Graeber 1994; Arendt *et al*. 2000). In Los Angeles (8 hour westward shift), the traveller will have difficulty remaining asleep in the morning (temperature rising) but will feel tired in the afternoon and evening (temperature low) (Figure 3) (Waterhouse *et al*. 1997). Conversely, after westward

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travel, sleep is scheduled after the travellers' 'home' bedtime, and near the trough of the circadian body temperature rhythm. The quality of sleep is usually good in the first part of the sleep episode, but early awakenings may be evident in the latter part of the sleep episode (Graeber 1994; Arendt *et al*. 2000).

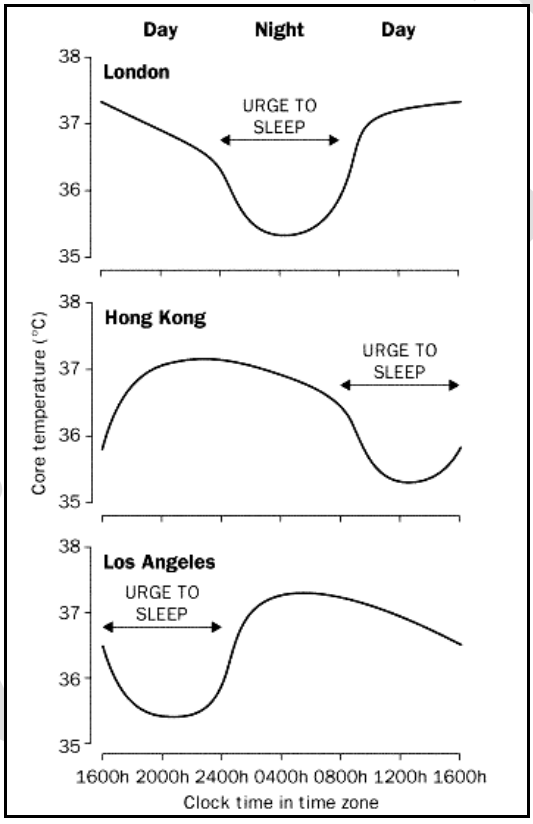



**Figure 3.** Diagrammatic representation of core (rectal) temperature circadian rhythm in an individual who generally sleeps from 24:00–08:00 hours and is mainly sedentary during the day. The 'urge to sleep' represents the time at which the traveller will normally go to sleep at home (e.g., London), coinciding with a drop in core body temperature (Waterhouse *et al*. 1997).

Several techniques can be employed to assess the sleep-wake pattern in travellers after transmeridian travel. The most common and reliable method of recording sleep objectively is by PSG. However, PSG is costly and may further disrupt sleep of the traveller, thus few field studies have used PSG measurements to record sleep patterns. Beaumont *et al*. (2004) recorded sleep using PSG recordings for nine nights in 27 healthy volunteers following eastward travel across seven time zones. The results showed that the duration of SOL was longer for three nights, SWS was increased for three nights, and REM sleep was decreased for one night, showing a rebound effect, with REM sleep increasing on night three after travel.

Similar sleep disturbances associated with the direction of travel have been reported in laboratory phase shift studies. Hume conducted a study in 1980 to investigate the immediate response and course of adaptation of the sleep pattern (PSG) after an eight hour phase delay (westward travel) and phase advance (eastward travel) of the sleep-wake cycle in 15 subjects. Sleep was significantly disrupted after both phase shifts. However, a phase advance of the sleep-wake cycle caused a greater disruption of the sleep episode with increased number of awakenings, body movements and stage 1 sleep. Furthermore, after a phase delay, there was a significant increase in the amount of REM sleep, whereas the opposite was observed after a phase advance. Similarly, Monk *et al*. (1988) measured sleep patterns (PSG) in eight male subjects after a six hour phase advance shift in the sleep-wake cycle and concluded that sleep duration, percentage of REM sleep and SWS decreased significantly after the phase shift. Carrier *et al*. (1996) objectively measured sleep (PSG) in 25 elderly subjects

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after a six hour phase shift, and the results showed a reduction in the percentage of SE, the duration of TST, SOL, and an increase in the number of awakenings in the first part of the sleep period. The sleep disturbances after laboratory simulated studies, as measured by PSG recordings, provide valuable insight into the sleep problems which occur after transmeridian travel. However no current study has investigated the combined impact of jet lag on objective sleep recordings (PSG) and assessment of athletic performance within a laboratory setting or a field study in athletes.

The use of PSG recordings for sleep patterns is ideal for laboratory simulated phase shifts; however this method is inadequate for the study of sleep patterns in field studies of jet lag. Consequently, few field studies have utilized PSG for the investigation of sleep disturbances after transmeridian travel, and researchers have used subjective assessment of sleep patterns such as sleep diaries or sleep logs, and monitored the activity levels of travellers after transmeridian travel using activity data loggers. Sleep diaries or sleep logs can be used to provide subjective assessments of the quality and quantity of sleep (sleep onset and offset times, number of awakenings, sleep duration) simultaneously in many subjects travelling across time zones (O'Connor *et al*. 1991; Edwards *et al*. 2000; Jamieson *et al*. 2001; Takahashi *et al*. 2002). However, the reliability of sleep diaries and sleep logs is purely at the discretion of the subject, and may potentially provide inaccurate results. On the other hand, activity data loggers worn on the wrist or ankle, provide an easy and accessible measurement of motor activity after transmeridian travel. Activity

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data loggers monitor movement which can be related to wakefulness; inactivity is presumed to occur during the sleep episode, and the quality of sleep can be correlated with the number of movements during a sleep episode (Ancoli-Israel 2000).

Subjective measures in sleep patterns after travel across multiple time zones have been extensively studied in the field in businessmen (Edwards *et al*. 2000), the military (Beaumont *et al*. 2004), travellers (Hill *et al*. 1993; Daurat *et al*. 2000; Jamieson *et al*. 2001), academics (Hill *et al*. 1993; Edwards *et al*. 2000; Takahashi *et al*. 2002), airline employees (Preston *et al*. 1973a; Preston *et al*. 1973b; Suvanto *et al*. 1993a; Haugli *et al*. 1994; Lowden and Åkerstedt 1998; Lowden and Åkerstedt 1999), and athletes (O'Connor *et al*. 1991; Hill *et al*. 1993; Lemmer *et al*. 2002).

The subjective changes in sleep, after eastward travel across time zones and laboratory simulated phase shifts include increased SOL (Lowden and Åkerstedt 1999; Jamieson *et al*. 2001), decreased TST (Deacon and Arendt 1996; Takahashi *et al*. 2002), increased wakefulness during the sleep period (Deacon and Arendt 1996; Lowden and Åkerstedt 1999; Jamieson *et al*. 2001; Takahashi *et al*. 2002), and reduced sleep quality (Deacon and Arendt 1996; Jamieson *et al*. 2001). However, no previous study has documented the impact of sleep disruption, as measured by PSG recordings, in athletes after transmeridian travel.

In summary, a rapid time zone change results in an unadjusted body temperature circadian rhythm, and subsequently sleep initiation may be problematic. Both

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eastward and westward travel impairs sleep. However the severity of sleep problems is influenced by the direction of travel, number of time zones crossed, and departure or arrival times of the flight. Furthermore, sleep disturbances are more persistent following eastward travel. Investigators have utilized objective (PSG) and subjective (daily sleep diaries) measurements to obtain sleep data after transmeridian travel and laboratory simulated phase shifts. However, few studies have investigated sleep parameters, using PSG recordings, after transmeridian travel and simulated phase shifts in athletes. The disturbance in the normal sleep pattern associated with transmeridian travel leads to sleep loss, and the impact thereof may be related to the impact on daytime activity, mood, fatigue, or cognitive performance of travelling athletes.

#### *1.5.3.2 Impaired Mood, Alertness and Cognitive Performance*

It has been suggested that transmeridian travel may impact negatively on cognitive performance (Cho *et al*. 2000; Katz *et al*. 2001), stress levels as measured by cortisol (O'Connor *et al*. 1991; Cho *et al*. 2000), and psychological factors such as mood, fatigue, and alertness (Wright *et al*. 1983; O'Connor *et al*. 1991; Hill *et al*. 1993, Edwards *et al*. 2000; Waterhouse *et al*. 2002). As previously mentioned these factors may also have an impact on athletic performance.

One of the first studies to investigate the relationship between jet lag, cognitive performance and mood was conducted in a laboratory simulated phase shift animal

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model (Tapp and Natelson 1989). Tapp and Natelson (1989) examined how a six hour phase advance shift affected cognitive performance (vigilance-discrimination task) in seven male Rhesus monkeys. The results showed that cognitive performance in monkeys was significantly decreased after a six hours phase shift. Similarly, a decrement in memory function, in humans, has been shown in laboratory and field studies (Deacon and Arendt 1996; Cho *et al*. 2000). In the laboratory study, efficiencies in both search and memory task were impaired for five days after a nine hour phase advance shift (Deacon and Arendt 1996). In the long-term jet lag, the study on aircrew, the levels of cortisol and memory function were negatively altered (Cho *et al*. 2000).

Several studies have investigated the psychological status of traveller's after transmeridian travel (Wright *et al*. 1983; O'Connor *et al*. 1991; Hill *et al*. 1993; Daurat *et al*. 2000; Edwards *et al*. 2000; Jamieson *et al*. 2001; Waterhouse *et al*. 2003). Examples of the different psychometric questionnaires used by investigators to measure mood disturbances after transmeridian travel include the Liverpool Jet lag Questionnaire (Edwards *et al*. 2000; Waterhouse *et al*. 2003), Visual Analogue Scale (VAS) measuring alertness, mood, motivation, and ability to concentrate (Daurat *et al*. 2000; Edwards *et al*. 2000; Jamieson *et al*. 2001), Environmental Symptoms Questionnaire (ESQ) (Wright *et al*. 1983), and Profile of Mood States (POMS) (O'Connor *et al*. 1991; Hill *et al*. 1993). A number of studies have shown worsened mood states following eastward or westward travel (Wright *et al*. 1983; Hill *et al*. 1993) as well as simulated phase shifts (Monk *et al*. 1988; Deacon and Arendt 1996).

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Presently, limited research has investigated the mood and sleep disturbances associated with transmeridian travel in athletes. Both O'Connor *et al*. (1991) and Hill *et al*. (1993) have examined the effect of transmeridian travel on sleep and mood in competitive elite athletes. O'Connor *et al*. (1991) compared the responses of male and female elite swimmers to westward and eastward flights across four time zones. In this study, sleep was measured subjectively and mood states were assessed, once a day, before training, using a POMS questionnaire. The investigators reported more sleep disturbances after eastward travel, compared to westward travel. As well, O'Connor *et al*. (1991) observed that mood states were significantly improved after travel in either westward or eastward travel. Hill *et al*. (1993) conducted a study to investigate the effects of westward travel across eight time zones in female athletes from the USA Women's National Soccer team. Sleeping habits (daily logs) and mood (POMS) were measured one week before departure and then again after arrival in Taiwan for 12 days. The results of this study showed a change in the athletes' sleeping habits and a marked increase in fatigue, total mood disturbance (TMD), and decreased vigour for several days after the flight.

The results of O'Connor *et al*. (1991) are relevant to my study on the impact of transmeridian travel on mood in athletes. The study conducted by O'Connor *et al*. (1991) presents conflicting outcomes of mood following travel across time zones. However, a number of explanations can be suggested to clarify the discrepancy:

1. *Baseline measurement.* Firstly, assessment of mood before departure may have been inconclusive and confounded the results. The competitive swimmers were

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engaged in heavy training sessions and the measured mood before departure may have already been disrupted due to over-training (O'Connor *et al*. 1991). In order to assess the impact of transmeridian travel on the psychological aspects of athletes, one has to take into account the intensity of training before and after transmeridian travel.

2. *Time zone traversal.* The severity of jet lag is influenced by the direction of travel and number of time zones crossed (Winget *et al*. 1985; Youngstedt and O'Connor 1999). Elite swimmers travelled eastward and westward across four time zones (O'Connor *et al*. 1991). In agreement with the present literature, circadian desynchronization is apparent after crossing three or more time zones (O'Connor and Morgan 1990; Reilly and Edwards 2007). Thus, the more time zones crossed, the greater the severity of circadian desynchronization.
3. *Psychological perception of the destination.* The travel destination may impact on the psychological status of a traveller. Elite swimmers travelled from winter to a tropical destination (Hawaii) which may have had a positive effect on the quality of mood.

The mood improvement seen in the study conducted by O'Connor *et al*. (1991) cannot be attributed to the effects of transmeridian travel or the effects of sleep changes. Since few studies have evaluated the mood disturbances in athletes after transmeridian travel, it is unclear whether transmeridian travel alters the psychological status of athletes.

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The negative consequences of transmeridian travel on mood, alertness and cognitive performance may be attributed to disrupted or altered sleep patterns, and the resultant sleep deprivation (Shephard 1984; Loat and Rhodes 1989; Meir 2002). Several investigators have studied the effects of partial or total sleep deprivation on human cognitive and motor performance, and psychological status (Bonnet 1985; Dinges *et al*. 1997; Meney *et al*. 1998; Bonnet 2000; Williamson and Feyer 2000; Phillip *et al*. 2004; Vgontzas *et al*. 2004; Scott *et al*. 2006). Slower reaction times (Williamson and Feyer 2000; Phillip *et al*. 2004; Scott *et al*. 2006), impaired psychomotor performance (Dinges *et al*. 1997), elevated sleepiness (Dinges *et al*. 1997; Phillip *et al*. 2004), and mood alterations (Bonnet 1985; Dinges *et al*. 1997; Meney *et al*. 1998; Scott *et al*. 2006) have been reported following partial or total sleep deprivation. The disturbance in mood after sleep deprivation is similar to the mood alterations that occur after transmeridian travel. As discussed previously, transmeridian travellers' are exposed to restricted sleep schedules until their sleep-wake cycles adjust to new local times.

In summary, the present literature confirms that travel across time zones or laboratory simulated phase shifts result in decreased performance, reduced alertness, increased fatigue, and mood alterations that can partly be a result of the temporary impairment in the quality and quantity of sleep. Presently, there is limited research investigating the combined impact of the sleep loss, impaired cognitive performance, and mood disturbances associated with transmeridian travel in athletes, and whether these undesirable symptoms of jet lag may impair athletic performances.

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### *1.5.3.3 Decrement in Athletic Performance*

As far as athletic performance is concerned the adverse consequences of jet lag, such as disrupted sleep-wake cycle, associated disturbances in mood, increased fatigue, and increased daytime sleepiness, have attracted considerable interest. Several authors have suggested that rapid transmeridian travel has detrimental consequences on athletic performance (Wright *et al*. 1983; Hill *et al*. 1993; Jehue *et al*. 1993; Recht *et al*. 1995; Lagarde *et al*. 2001). However, despite the increasing number of investigations focused on athletic performance after transmeridian travel, the available literature is both confounding and inconclusive. A review of the extensive literature related to athletic performance following rapid traversal of multiple time zones noted that the majority of jet lag investigations are flawed with "a number of methodological problems" (O'Connor and Morgan 1990).

A number of studies have investigated the effects of transmeridian travel across time zones on team performance and individual athletic performance. These studies are summarized in Table 2.

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**Table 2.** *The effects of transmeridian travel on athletic performance*

| Author | Subjects | Direction of travel/number of time zones crossed | Results |
|---|---|---|---|
| Wright *et al*. (1983) | Military personnel | Eastward travel/six time zones | ↔ HRmax, VE, $VO_2$max<br>↓ RPE<br>↓ dynamic arm strength, performance in 270m sprint and 2.8km run |
| Lagarde *et al*. (2001) | U.S. Air Force Reservists | Eastward travel/seven time zones | ↓ grip strength, squat jump<br>↔ multiple jump test |
| Hill *et al*. (1993) | Students | Eastward travel/six time zones | ↔ grip strength<br>↓ anaerobic power and capacity |
| Jehue *et al*. (1993) | National Football League (NFL) teams | Intra-continental westward and eastward travel/two to three time zones | Eastward travel negatively impacts on team performance as a result of a shift in 'peak performance window' |
| Recht *et al*. (1995) | American Baseball teams | Intra-continental westward and eastward travel/three time zones | Eastward travel negatively impacts on team performance as a result of a shift in 'peak performance window' |
| Steenland and Deddens (1997) | National Basketball Association (NBA) teams | Inter-continental westward and eastward travel/one to two time zones | Team performance was significantly improved after eastward travel compared to westward travel |
| O'Connor *et al*. (1991) | Elite swimmers | Westward and eastward travel /four time zones | ↑ HR (resting) (W-E travel)<br>↔ RPE<br>↓ muscle soreness |
| Bullock *et al*. (2007) | Elite athletes | Eastward travel/eight time zones | ↔ 30m sprint performance |

↓, ↔ ↑ represents a decrease, no change, and increase in measured variables. HR- heart rate; VE- ventilation; $VO_2$max- maximal oxygen uptake; RPE- rate of perceived exertion.

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Several studies have investigated the impact of transmeridian travel on athletic performance in non-athletes (Wright *et* al. 1983; Hill *et al*. 1993; Lagarde *et al*. 2001). A review by O'Connor and Morgan (1990) commented, in detail, about the investigations reported by Wright *et al*. (1983) on athletic performance after transmeridian travel across multiple time zones. They suggested that the soldiers' physical performance and physiological structure (muscle fibre composition, maximal oxygen uptake) may differ to that of athletes and the repetitive testing could have caused a further increase in fatigue and muscle soreness which may have consequently contributed to a reduced performance. Thus, athletic impairment after transmeridian travel, as reported in studies conducted on non-athletes (Wright *et al*. 1983; Hill *et al*. 1993; Lagarde *et al*. 2001), cannot be "generalized to situations involving the performance of athletes in competition" (O'Connor and Morgan 1990, p.24).

Several studies have examined the pattern of team wins and losses to determine whether the effect of travel across time zones negatively impacts on team performance. Jehue *et al*. (1993) and Recht *et al*. (1995) concluded that the direction of travel, especially in an eastward direction, negatively impacted on team performance. The authors suggested that the greater loss record was partly due to the shift in their circadian rhythm, ultimately affecting their optimal time for peak performance (Jehue *et al*. 1993; Recht *et al*. 1995). In contrast, Steenland and Deddens (1997) disagree that team performance deteriorates after eastward travel. The authors concluded that, compared to westward travel, teams travelling in an

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eastward direction performed significantly better as night games coincided with their optimal time of peak performance (Steenland and Deddens 1997).

The results of the above mentioned studies conducted by Jehue *et al*. (1993), Recht *et al*. (1995), and Steenland and Deddens (1997) reported that travel across time zones has a negative impact on athletic performance in athletes. However, the results of these retrospective studies of team performance makes it is difficult to conclude that time zone traversal adversely affects team performance. For a number of reasons, these studies fail to provide direct evidence that jet lag adversely affects athletic performance.

1. *Team versus individual performance*. Each study investigated the performance 'decrements' in team sports which neglects individual variations in performance. Several contributing factors such as age, personality chronotype or physical fitness influence the severity of jet lag (Waterhouse *et al*. 2002). Therefore, some individual members of the team may have improved in their individual performance or effects of jet lag may have had no impact in their performance decrement.
2. *Opposing team performance*. The performance of the opposing teams is not taken into account. For instance, a loss may have been a result of the opposing team being a stronger and more-skilled team. Thus, poor performance after transmeridian travel may be partly attributed to superior opponents, and not because of jet lag.

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3. *Number of time zones crossed*. The negative effects of jet lag are experienced after three or more changes in time zones of westward or eastward travel (O'Connor and Morgan 1990). Most of the studies travelled across one time zone (Steenland and Deddens 1997), two time zones (Jehue *et al*. 1993; Steenland and Deddens 1997), or at most three time zones (Jehue *et al*. 1993; Recht *et al*. 1995). Each study fails to provide substantial evidence that the teams experienced jet lag and that subsequent team losses were a result of jet lag.
4. *Travel fatigue*. Lastly, the authors neglected the role of travel-related stress, fatigue and anxiety that may have partially contributed to a decline in team performance (Preston *et al*. 1973a; Reilly *et al*. 1997; Waterhouse *et al*. 2004).

Even though performance was investigated in a competitive nature by means of win-loss game statistics, the results of these retrospective studies (Jehue *et al*. 1993; Recht *et al*. 1995; Steenland and Deddens 1997) fail to provide substantial evidence that travel across time zones has a negative effect on athletic performance. Thus, research on the impact of jet lag on athletic performance needs to focus on individual variability and the impact of jet lag on various components of athletic ability in athletes.

To date, few studies have assessed individual athletic performance after transmeridian travel in athletes (O'Connor *et al*. 1991; Hill *et al*. 1993; Bullock *et al*. 2007). O'Connor *et al*. (1991) investigated the impact of transmeridian travel on

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athletic performance in 40 college swimmers travelling across four time zones and concluded that air travel across four time zones did not impair athletic performance in competitive swimmers.

On the other hand, Hill *et al*. (1993), in three studies to investigate the negative effects of jet lag on factors related to sports performance, noted a decline in athletic performance after transmeridian travel. In both the first and third studies performance was not assessed before transmeridian travel and therefore the accuracy may be compromised. In Hill *et al*.'s (1993) second study, the grip strength and anaerobic power of students travelling in an eastward direction across six time zones was measured two days before, and four days after the flight. Hill *et* al. (1993) concluded that grip strength remained unchanged after air travel. However, anaerobic power and capacity were significantly altered for up to two days after air travel.

I have reviewed that the literature related to athletic performance may be temporarily impaired after transmeridian travel as a result of disrupted circadian rhythm of athletic performance, training sessions or competitive events not coinciding with peak performance time, loss of motivation to train or compete, or as a result of altered mood. Furthermore, transient sleep problems associated with transmeridian travel may lead to sleep deprivation and thus further impair athletic performance.

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It is well documented that sleep deprivation negatively affects mood and mental performance (Bonnet 1985; Dinges *et al*. 1997; Meney *et al*. 1998; Bonnet 2000; Williamson and Feyer 2000; Vgontzas *et al*. 2004; Scott *et al*. 2006); however, the impact of sleep deprivation on subsequent athletic performance is both inconclusive and not compelling. The impact of sleep deprivation and partial sleep loss on subsequent exercise performance has been investigated and summarized in Table 3.

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**Table 3.** *Sleep deprivation and athletic performance*

| Authors | Length of sleep deprivation | Type of exercise | Parameters measured | | | |
|---|---|---|---|---|---|---|
| | | | **Physiological** | **Metabolic** | **Physical performance** | **Psychological** |
| Martin and Gaddis (1981) | 30 hours | Cycle ergometer at 25, 50, and 75% $VO_2$max | ↔ resting HR, $VO_2$max, $VCO_2$, VE, BP<br>↓ peak HR | - | - | ↑ RPE |
| Horne and Pettitt (1984) | 72 hours | Cycle ergometer at 40, 60, and 80% $VO_2$max | ↔ maximal HR, $VO_2$max, $VCO_2$, RQ | - | - | - |
| McMurray and Brown (1984) | 24 hours | 20 minute sub-maximal treadmill run | ↔ resting HR, resting VE, $VO_2$max | ↔ lactate, blood glucose, plasma volume | - | - |
| Takeuchi *et al*. (1985) | 64 hours | 40m dash, vertical jump test, isometric handgrip strength, isokinetic leg strength and endurance, balance | - | - | ↔ 40m dash, grip strength, balance, or peak torque of knee extension (180 and 300°$s^{-1}$)<br>↓ vertical jump height, and peak torque of knee extension by 20% (60°$s^{-1}$) | - |
| Symons *et al*. (1988) | 60 hours | Wingate anaerobic test<br>Muscular strength and endurance<br>70% $VO_2$max cycle ergometer<br>70 and 80% $VO_2$max treadmill run | ↔ cardiovascular and respiratory responses | ↔ blood lactate | ↔ peak power, velocity, and muscular strength | - |

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↓, ↔, ↑ represents a decrease, no change, and increase in measured variables. HR- heart rate; $VO_2$max- maximal oxygen uptake; $VCO_2$- maximal carbon dioxide expired; VE- ventilation; BP- blood pressure; RPE- rate of perceived exertion; RQ- respiratory quotient.

**Table 3.** *Sleep deprivation and athletic performance-continued*

| Authors | Length of sleep deprivation | Type of exercise | Parameters measured | | | |
|---|---|---|---|---|---|---|
| | | | Physiological | Metabolic | Physical performance | Psychological |
| Goodman *et al.* (1989) | 60 hours | Maximal aerobic cycle ergometer protocol | ↔ peak VE, RER, HR | ↔ maximal blood lactate<br>↑ Plasma volume<br>↓ Hb concentration, red blood cell count | - | - |
| Reilly and Piercy (1994) | Sleep restricted to 3 hours for 3 successive nights | Maximal lift (bicep curls, bench press, leg press, and dead lift) | - | - | ↓ maximal bench, leg press, and dead lift | Altered mood states (POMS)<br>↑ confusion, fatigue<br>↓ vigour |
| Mougin *et al.* (1996) | Sleep delayed until 3am | Wingate anaerobic test | ↔ maximal VE | ↔ pH, lactate | ↔ peak velocity, output | - |
| Souissi *et al.* (2003) | One night's SD | Wingate anaerobic test (measured at 06:00 and 18:00) | - | - | ↓ maximal power and capacity (36hrs SD) | - |
| Bambaeichi *et al.* (2005) | Sleep restricted to 2.5 hour for one night | Muscular strength (isometric and isokinetic strength of knee extensors and flexors) | - | - | ↔ muscular peak torque strength | - |

↓, ↔, ↑ represents a decrease, no change, and increase in measured variables. VE- ventilation; RER- respiratory exchange ratio; HR- heart rate; Hb- haemoglobin; POMS- Profile of Mood States; SD- sleep deprivation.

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As seen in Table 3, in response to sub-maximal or maximal exercise, sleep deprivation of 24 hours up to 70 hours has no effect on physiological parameters such as heart rate, blood pressure, maximal oxygen uptake ($VO_2$max), and ventilation rate (Martin and Gaddis 1981; Horne and Pettitt 1984; McMurray and Brown 1984; Goodman *et al*. 1989; Mougin *et al*. 1996). Similarly, muscular strength, maximum isometric contraction, and power output or capacity remains unaffected following partial or total sleep deprivation (Takeuchi *et al*. 1985; Symons *et al*. 1988; Mougin *et al*. 1996; Bambaeichi *et al*. 2005). On the other hand, sleep deprivation results in an increased rate of perceived exertion (RPE), decreased explosive power, decreased muscular strength, and decreased anaerobic capacity (Martin and Gaddis 1981; Takeuchi *et al*. 1985; Reilly and Piercy 1994; Souissi *et al*. 2003). The impact of sleep deprivation associated with jet lag on athletic performance has not been previously investigated. However, judging from these investigations, it would appear as though the effect of sleep deprivation on athletic performance after transmeridian travel can be discounted.

In summary, the current evidence on the impact of jet lag on athletic performance is "based on a number of unconfirmed hypotheses" that lacks significant scientific facts (O'Connor and Morgan 1990). The present literature confirms that static strength, dynamic strength and endurance, anaerobic power and capacity, and performances in sprint and distance running, are impaired after transmeridian travel across multiple time zones. In contrast, athletic performance is unaffected or can improve after transmeridian travel. The present assessment of athletic performance after

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transmeridian travel is based on team performance, untrained subjects, and athletes. Thus, the inconsistent and conflicting results of the impact of transmeridian travel on athletic performance may be partly due to subject recruitment and the type of athletic assessment. Further research needs a more controlled, effective assessment of individual athletic performance after transmeridian travel in athletes.

## 1.6 Conclusion

Circadian rhythms such as sleep-wake cycles, cognitive, and athletic performance, which closely follow the body temperature rhythm, are all interrelated. The present literature confirms that travel across time zones negatively alters the circadian body temperature rhythm, sleep-wake cycle, psychological status, cognitive performance, and athletic ability of the traveller.

In summary, athletes are faced with two major problems after transmeridian travel across time zones:

1. *Shift in the circadian 'peak performance window'.* The misalignment of body temperature rhythm with the local time cues implies that maximum performance during training sessions or competitive events may be decreased because the event does not coincide with peak circadian performance.
2. *Symptoms associated with jet lag.* The decrement in sports performance may be attributed to the undesirable psychological and physiological symptoms associated with jet lag. Jet lag results in sleep deprivation, which may

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negatively impact the mood and motivation of an athlete and in turn may affect the mental and physical performance of an athlete.

A reduction in sports performance after travelling across time zones has focused primarily on the symptoms associated with jet lag while neglecting the role of travel-related stress, fatigue, and anxiety. Additional non-circadian factors such as flight conditions (restricted physical mobility, dehydration, quality of cabin air), uncomfortable sleeping environment, and time of arrival or social differences at competing destinations may partially account for a decline in performance (Hauty 1967; Reilly *et al*. 1997; Youngstedt and O'Connor 1999; Waterhouse *et al*. 2004).

Laboratory simulated jet lag studies have been able to mimic the effects of transmeridian travel across time zones by showing disrupted sleep patterns and altered neurobehavioural variables (mood, fatigue, alertness, and cognitive ability) that are similar to field studies on jet lag. The need to further understand the impact of jet lag on athletic performance is relevant; however transporting athletes across time zones is costly. Furthermore, athletes travelling across several time zones may be exposed to a wide range of physiological, psychological, and behavioural variables; therefore, simulating jet lag within a laboratory setting would eliminate a number of these variables associated with travel and allow for the assessment of 'pure' time zone change on athletic performance. To date, no study has investigated the impact of simulated phase shifts on various components of athletic performance

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within a controlled laboratory environment while measuring sleep and mood as interacting variables.

## Objectives

1. To assess the impact of a laboratory simulated six hour phase advance shift on
    a) sleep patterns as measured by PSG recordings
    b) cognitive performance as measured by simple reaction time
    c) athletic performance as measured by muscular strength, abdominal muscular endurance, hamstring muscular flexibility, hand grip strength, anaerobic and aerobic performance in male athletes.
2. To gain some insight into the interaction between these three variables.

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# CHAPTER TWO

## *Materials and Methods*

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## 2.1 Materials and Methods

### 2.1.1 Subjects

Ten male subjects voluntarily participated in this study. Subjects were recruited from various sporting clubs and were amateur athletes who regularly participated in soccer, marathon running or triathlon. Ethical clearance was obtained from the University of the Witwatersrand's Committee for the Research on Human Subjects (Protocol Number M060221); all the subjects gave written consent for participation.

### 2.1.2 Experimental Protocol

The study was divided into three separate parts: an adaptation night, a two day "home" weekend and a two day six hour phase advanced ("away") weekend. Each subject spent the two separate weekends, consisting of three nights and two days each, in the Witwatersrand Dial-A-Bed Sleep Laboratory which is housed in the School of Physiology, University of the Witwatersrand, Johannesburg, South Africa (1700m above sea level).

#### *2.1.2.1 Adaptation night*

Approximately two to three days before the first experimental weekend, each subject spent one night in the Sleep Laboratory. The aim of this night was to familiarize the

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subjects with the living conditions, sleeping facilities, and measuring techniques that they would experience during the experimental weekends.

After arrival at the Sleep Laboratory (18:00), each subject performed the battery of exercise and cognitive tests to be used in the experimental weekends (please refer to exercise session and simple reaction time tests). The subjects were also required to complete the General Health Questionnaire (GHQ), the Morningness-Eveningness questionnaire (Horne and Östberg 1976), and Pittsburgh Sleep Quality Index (PSQI) (Buysse *et al*. 1989), which were used to screen for psychological abnormalities, personality traits and sleep disorders respectively.

Before sleeping, the subjects were prepared for polysomnography (PSG) recordings; including measurements of electroencephalography (EEG), electro-oculography (EOG) and electromyography (EMG) (please refer to polysomnographic recordings and measurements). Subjects were then allowed to sleep for the night but no recordings were performed. The following morning all electrodes were removed and the subjects were free to leave.

#### *2.1.2.2 Study design*

On arrival in the Sleep Laboratory, all possible time cues (watches, cell phones) were taken away from the subjects and returned after each study period. As there are no windows within the Sleep Laboratory, the subjects were isolated from any external time cues (sunrise, sunset), and physical and communicative interaction with the

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outside world. The subjects had no access to 'real-live' television or radio broadcasts and were not informed of the 'actual' time over either weekend.

For the duration of both weekends, each subject followed a fixed routine of bedtime and waking schedules, meal times, and exercise and cognitive sessions, which were initiated by the primary investigator. The 'daytime' period commenced at 07:00 (waking of subjects) with a light intensity of approximately 950 lux, measured with a digital light meter (Model YF-170, Yu Fong Electronics, Singapore). At 19:00, the light intensity was lowered to approximately 488 lux, representing the 'nighttime' period, until 22:00 (bedtime), when lights were turned off until morning waking time (07:00).

The subjects were not allowed to take naps during the 'daytime' period. The subjects were served breakfast at 08:00, lunch at 13:00 and dinner at 19:00 (Table 4). Snacks and refreshments were freely available throughout the day. During the wakeful time, the subjects were allowed to engage in non-physical activities such as watching videos, watching DVDs, and reading or playing video or computer games.

The "home" and "away" weekends were randomized, and the subject was unaware of which timeline he followed for each experimental weekend. Additionally, the subjects were not informed about the direction or magnitude of the phase shift for the "away" weekend.

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The subjects were instructed to refrain from exercising for at least 48 hours, and to abstain from consuming alcohol or caffeine for at least one week, before each experimental weekend. On both weekends, assessment of core body temperature, objective and subjective sleep parameters, subjective sleepiness, mood states, and measurements of cognitive and athletic performance were obtained.

#### *2.1.2.3 "Home" weekend*

The "home" weekend corresponded to a Johannesburg, South Africa, timeline (Greenwich Mean Time, GMT +2 hours). The subjects arrived at the Sleep Laboratory at approximately 20:00 on Friday night and were fitted with EEG, EOG and EMG electrodes, and retired to bed at 22:00. The next morning, the subjects were woken up at 07:00 and ate breakfast at 08:00. The subjects performed cognitive testing at 15:00 followed by a battery of exercise tests conducted between the hours of 16:00-18:00 and filled in various questionnaires throughout the wakeful period (please refer to subjective sleepiness and mood states). The subjects retired to bed at 22:00. The subjects followed the above routine for the entire "home" weekend (Table 4).

#### *2.1.2.4 "Away" weekend*

The subjects arrived at the Sleep Laboratory at approximately 16:00 on Friday afternoon and were fitted with EEG, EOG and EMG electrodes for PSG recordings, and retired to bed at approximately 17:00. The subjects were woken up at 01:00 (local laboratory clock time, GMT +2 hours), the following morning, corresponding to a

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morning rising time of 07:00 in Perth, Australia (GMT +8 hours). For the remainder of the weekend, sleep-wake times, meals, exercise and cognitive tests occurred at the correct times for the six hour phase advance. The six hour phase shift corresponded to simulating eastward travel from Johannesburg, South Africa, to Perth, Australia. On the final night, the subjects went to bed at 22:00 (GMT +8 hours) and were allowed to sleep for as long as required to ensure minimal effects of sleep deprivation that may have occurred over the weekend.

**Table 4.** *Daily routine for "home" and "away" weekends*

| Time | Activity |
|---|---|
| 07:00 - 07:30 | Wake up, lights on, morning sleep questionnaire |
| 08:00 - 09:00 | Breakfast, Pictorial Sleepiness Scale |
| 09:00 - 13:00 | 'Free' time |
| 13:00 - 14:00 | Lunch |
| 14:00 - 15:00 | 'Free' time |
| 15:00 - 16:00 | PVT, Pictorial Sleepiness Scale, POMS |
| 16:00 - 18:00 | Exercise |
| 18:00 - 19:00 | 'Free' time |
| 19:00 - 21:00 | Dinner, 'free' time |
| 21:00 | Pictorial Sleepiness Scale, placement of electrodes for PSG recordings |
| 22:00 - 07:00 | Retire to bed, lights off |

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| | |
|---|---|
| 07:00 | Wake up, lights on |

Daily representation of the fixed routine that subjects followed for the "home" and "away" weekends. The times indicated represent the actual laboratory clock time for the "home" weekend and advanced by six hours for the "away" weekend, being the correct times for the "away" weekend if the subject had travelled across six time zones in an eastward direction (GMT +8 hours). 'Free' time represents the periods where the subjects engaged in non-physical activities (described in the text). PVT- Psychomotor Vigilance Task; POMS- Profile of Mood States; PSG- Polysomnography.

## 2.1.3 Measurements and Recordings

### *2.1.3.1 Core body temperature*

Rectal core body temperature was continuously monitored for the duration of each weekend, using indwelling rectal thermistors connected to small portable temperature loggers (Stowaway XT1, Onset Computer Corporation, Pocasset, MA, USA). The thermistors were covered in a polythene sheath and were calibrated in a water bath against a quartz thermometer, with an accuracy of 0.1°C (Quat 100, Heraeus, Hanau, Germany), before commencement of the study. When arriving in the Sleep Laboratory, the subjects self-inserted the indwelling thermistor 10cm into the rectum. They removed the rectal probe during each exercise session and when defecating and showering. Rectal temperature was recorded at five minute intervals and data was downloaded onto a personal computer (BoxCar Pro, Version 4.3). As a result of technical problems (rectal thermistor insertion difficulties or missing values), the data of one subject were lost.

### *2.1.3.2 Polysomnographic recordings and measurements*

The standard PSG recording including EEG, EOG and EMG was recorded on a digital EEG system (Easy EEG version 2.0.2, Cadwell Laboratories Inc., Kennewick WA) at

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a recording speed of 30mm.s$^{-1}$. Standard EEG electrode placement (at C3, C4, A1 and A2) was used according to the International 10/20 system (Jasper 1958). Muscle tone (EMG) was recorded by placement of two electrodes on either side of the lower jaw bone (mental, submental). EOG activity was recorded by placing one electrode under the right eye and one electrode above the left eye. Two reference electrodes were placed behind the ears on the mastoid bone. Two ground electrodes were placed in the middle of the forehead above the nose.

Electrodes for PSG recordings were applied an hour before scheduled bedtime for each night of both weekends. Each site for placement of electrodes was cleaned and exfoliated with a typical toner and exfoliator, respectively. All electrodes were filled with an electrically conductive paste (Ten20 Conductive, D.O. Weaver & CO., Aurora, USA) and secured onto the specific sites of the face with micropore tape. As a result of technical difficulties (external interference or loss of data), the complete sleep data of only five subjects was obtained.

Sleep stages were scored according to the "Manual of Standardised Terminology, Techniques and Scoring System for Sleep Stages in Human Sleep" (Rechtschaffen and Kales 1968). The stages were scored epoch-by-epoch at 30 second intervals. The following objective sleep variables were calculated for all three nights of both weekends: Total sleep time (TST); amount of actual sleep time during a sleep period, Sleep onset latency (SOL); time from 'lights out' to the second epoch of stage 2 sleep, Rapid eye movement (REM) onset latency; time taken from SOL to the first

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epoch of REM sleep, Sleep efficiency (SE); ratio of TST to time in bed, and Total wake time (TWT); duration of time awake during the sleep period. Each stage of sleep was analyzed by measuring the total duration and percentage (%) of TST.

#### *2.1.3.3 Subjective assessment of sleep*

Morning sleep questionnaires consisted of a 10-point scale, assessing subjective sleep quality such as refreshing sleep, time to fall asleep, number of awakenings, and morning alertness, were completed every morning for both weekends within 30 min of arising. Each subject was asked to mark from the left end to the right end with time to fall asleep as being shorter than normal to longer than normal, refreshing sleep as being worse than normal to better than normal, awakenings as being less than normal to more than normal, and morning alertness as being less than normal to more than normal.

#### *2.1.3.3 Subjective sleepiness*

To assess subjective sleepiness during the wakeful period of each weekend, the subjects completed a validated Pictorial Sleepiness Scale questionnaire (Maldonado *et al*. 2004) at 08:00, 15:00, and 21:00. Each subject was asked to mark the one face which best expressed how he felt at that specific time.

#### *2.1.3.5 Mood states*

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To evaluate mood states, subjects completed the Profile of Mood States (POMS) (Mcnair *et al*. 1971) questionnaire, once a day at 15:00 for each weekend. The subjects were required to complete the POMS questionnaire based on how they felt "right now". Total mood disturbances (TMD) for each subject was calculated by adding each subjects scores for tension, fatigue, depression, anger and confusion and then subtracting the vigour score from the total. The higher the TMD value was, the worse the mood.

#### *2.1.3.6 Simple reaction time*

Visual reaction time (alertness) was assessed using the Psychomotor Vigilance Task-192 (PVT) (Ambulatory Monitoring Inc., Ardsley, New York). Each subject completed a PVT test once a day at 15:00 for both weekends. For each PVT test session for both weekends, we calculated the following performance measures:

- Mean reaction time (RT) – average correct response times less or equal to 500ms (measure of speed).
- Number of false response – the number of RT less than 100ms (measure of performance accuracy).
- Number of lapses – the number of RT greater than 500ms (measure of alertness).

#### *2.1.3.7 Exercise session*

The exercise session comprising of a standard battery of exercise tests was conducted between 16:00-18:00 each day. The subjects performed a short warm-up including

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standard stretching exercises before the commencement of each exercise session. The following exercise tests were conducted on each day of both weekends:

**1) Muscular strength test.** The one repetition maximum (1RM) was assessed using the leg press and bench press. The 1RM test is a maximum amount of weight (kg) that a subject is able to leg and bench press once only (McArdle *et al*. 2000).

a) *Leg press*: - The subject was seated on the pad of the leg press machine (Cardio Genesis Fitness Systems, South Africa) at an angle approximately 45° in relation to the leg press machine. The subject completed a standard 1RM leg press test, resting for approximately three minutes between each attempt, until maximum weight was attained.

b) *Bench press*: - The subject lay flat on his back, on the bench press machine (Maxicam Muscle Dynamics, Southern California, USA), with his feet flat on the ground. The subject performed a typical 1RM bench press test until maximum weight was reached, resting for approximately three minutes between each attempt.

**2) Anaerobic ability.** The anaerobic ability of each subject was measured using a 10x10 metre shuttle run test. The total time (seconds) taken to complete ten sprints over a distance of 10 metres, was recorded.

**3) Anaerobic leg power.** Each subject performed a standing long jump test which required them to jump, from a static position, as far forward as possible, landing with both feet on the floor (Kearney *et al*. 2000). Each subject performed three successive jumps, resting for a few seconds between each jump, and their longest jump distance (cm) was recorded.

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**4) Hamstring muscular flexibility.** The flexibility of the hamstring muscle group of each subject was measured using the sit and reach 'box' (Model 01285, Lafayette Instrument Company, Indiana, USA). The subjects were instructed to reach forward and push the lever on top of the box as far away from them as possible (McArdle *et al*. 2000). Each subject performed three successive trials, resting for a few seconds between each attempt, and their longest distance (cm) was recorded.

**5) Abdominal muscular endurance.** The number of standard sit-ups each subject was able to perform in 30 seconds was used as a measure of their muscular endurance (McArdle *et al*. 2000). The subjects lay on their back with their knees bent. The subject's feet were held flat on the ground with their arms folded across their chest. A standard sit-up technique was enforced whereby each subject lifted his torso to a 90° position (knee height) and then returned back down without allowing their head or shoulders to touch the ground.

**6) Hand grip strength.** The hand grip strength of each subject's dominant hand was measured using a hand-held dynamometer (Model 78010, Lafayette Instrument Company, Indiana, USA). Each subject performed three successive trials, resting for a few seconds between each attempt, and their highest value (kg) was recorded.

**7) Aerobic exercise.** Each subject completed a multi-stage running protocol on a motorized treadmill (Powerjog E10, Birmingham, England) set at a constant gradient (0%) (Leger *et al*. 1988). A starting speed of 8.5km.h$^{-1}$ was maintained for one minute; thereafter the speed was increased by 0.5km.h$^{-1}$every minute until the subject reached exhaustion and voluntarily stopped running. The total distance run (km) and time (min) to exhaustion was recorded. During the run, the subjects assessed their rate

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of perceived exertion (RPE), at one-minute intervals, using the Borg Scale (Borg 1973).

## 2.1.4 Data Analysis

### *2.1.4.1 Core body temperature data*

To determine whether the subjects phase advanced over the 'away' weekend, edited raw temperature data for each subject was analyzed by determining the nadir (time of temperature trough) for each day of both weekends. The nadir for body temperature for each day of the "away" weekend was compared to the corresponding day of the "home" weekend and then analyzed using the Wilcoxon signed rank test for non-parametric data.

### *2.1.4.2 Polysomnographic data*

A repeated-measures analysis of variance (RM-ANOVA), with a Tukey-Kramer post-hoc test, was used to compare the objective sleep variables of all six recording nights ("home" and "away" weekends). A Paired T-test was used to compare corresponding nights of the "home" weekend to the "away" weekend.

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### *2.1.4.3 Subjective assessment of sleep*

A non-parametric RM-ANOVA (Friedman Test), with a Dunn post-hoc test, was used to compare subjective morning assessment data for each day of both weekends. Subjective morning assessment data was analyzed, using the Wilcoxon matched-pairs signed-ranked test for non-parametric data, by comparing the "home" weekend to the corresponding "away" weekend data for each day.

### *2.1.4.4 Subjective sleepiness*

A RM-ANOVA, with a Tukey-Kramer post-hoc test, was used to compare the subjective sleepiness data, as measured by the Pictorial Sleepiness Scale, at each time (08:00, 15:00 and 21:00) for each day of both weekends. Additionally, subjective sleepiness data was analyzed by comparing the actual laboratory time of day of the "home" weekend to the perceived time of day for the "away" weekend (i.e. actual "home" time 15:00 and actual "away" time 15:00), using the Paired T-test.

### *2.1.4.5 Mood states*

POMS data were analyzed using the Wilcoxon matched-pairs signed-ranked test for non-parametric data. The TMD, calculated from the POMS data, for each day of each weekend, and corresponding days of the "home" and "away" weekends were analyzed.

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### *2.1.4.6 Simple reaction time*

A Paired T-test was used to compare the PVT response data for each day of each weekend, and compare corresponding days of the "home" and "away" weekends.

### *2.1.4.7 Exercise tests*

Each exercise variable was analyzed using the Paired T-test (leg press, bench press, flexibility test, shuttle run, standing long jump test, hand grip strength, and treadmill run) or the Wilcoxon matched-pairs signed-ranked test for non-parametric data (sit-ups, RPE) by comparing data for each day of each weekend and comparing corresponding days of the "home" and "away" weekends.

Statistical analysis was performed using the computer software program Graphpad, Instat (GraphPad Software, version 3, California, USA). Statistical significance was set at $P \leq 0.05$ and all data are expressed as Mean $\pm$ SD, except otherwise were stated.

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# CHAPTER THREE

## *Results*

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## 3.1 Results

Ten male subjects aged 23.8 $\pm$ 3.2 years with an average body mass index of 23.0 $\pm$ 2.8 $kg.m^2$ voluntarily participated in the study. All subjects were in good health and good sleepers (SOL, 19.5 $\pm$ 6.9 minutes; TST, 7.4 $\pm$ 0.7 hours/night) as assessed by the GHQ and the PSQI, respectively.

All of the subjects were of good mental health, were neither morning nor evening type personalities, and did not have any sleep disorders.

### 3.1.1 Core body temperature data

Figure 4 represents the core body temperature rhythm for the "home" and "away" weekends. The subjects showed a normal circadian body temperature rhythm over the "home" weekend, with a trough in body temperature before waking (03:00-05:00), peak body temperature occurring between 18:00-21:00 and a decline in body temperature when sleep was initiated by the subjects (Figure 4).

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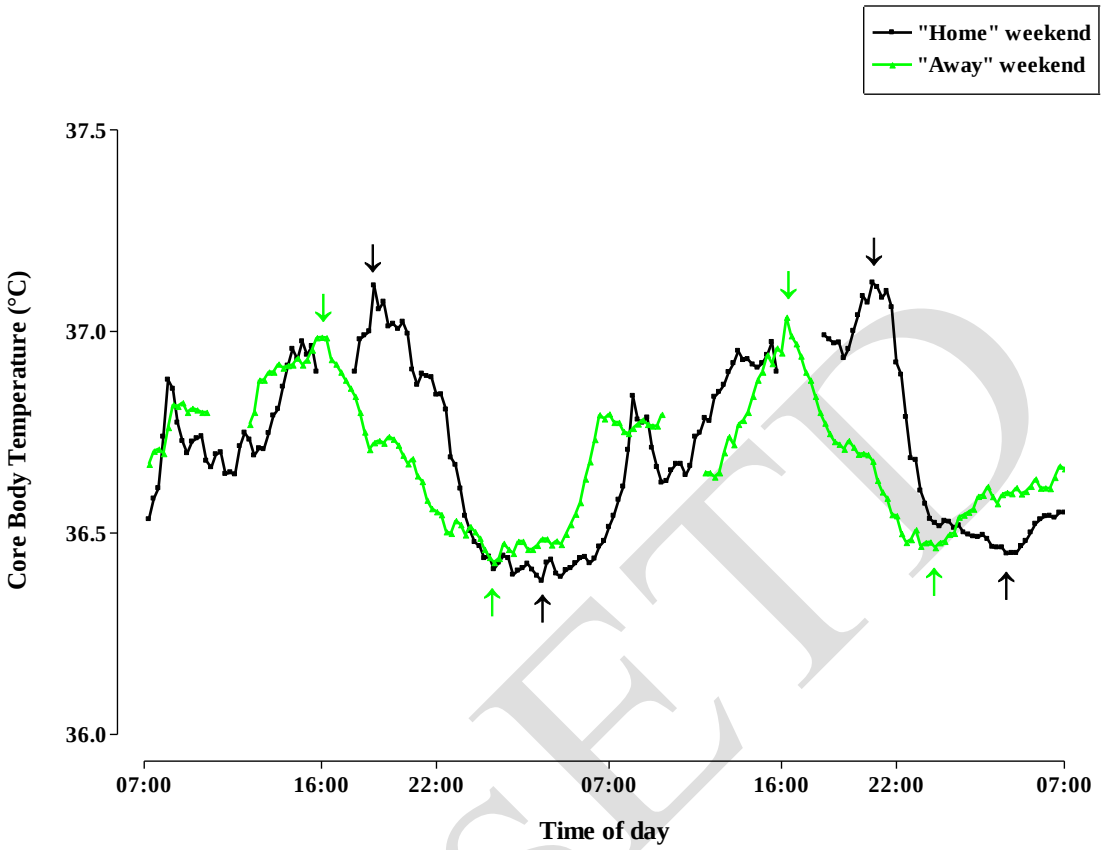



**Figure 4.** Mean core (rectal) body temperature for nine subjects for "home" and "away" weekends (48 hours). Time of day represents the local laboratory clock time (Johannesburg, South Africa, GMT +2 hours) for the "home" and "away" weekends. Arrows indicate peak and trough times of body temperature for both weekends. Sleep was scheduled between 22:00-07:00 and 16:00-01:00 for the "home" and "away" weekends, respectively. Exercise sessions were conducted between the hours of 16:00-18:00 and 10:00-12:00 for the "home" and "away" weekends, respectively. Missing data points represent the time when subjects were engaged in exercise sessions (removal of rectal probe).

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After two days on the six hour phase advance routine, the body temperature rhythm was disrupted (Figure 4); the lowest body temperature occurred at approximately 01:00 and peak body temperature occurring at approximately 16:00 (local laboratory clock time, GMT +2 hours). The trough body temperature for each night of both weekends was compared using the Wilcoxon signed rank test. A significant advance in body temperature troughs was evident for nights two ($p=0.05$; Wilcoxon signed rank test) and three ($p=0.004$; Wilcoxon signed rank test) of the "away" weekend compared to the corresponding nights of the "home" weekend.

### 3.2.2 Objective measurements of sleep

Table 5 shows objective measurements of sleep, as determined by polysomnography (PSG) recordings, for all three nights of both weekends.

There was no significant differences in TST, SOL, TWT, % stages 1, 2, % SWS, REM latency, % of REM sleep, and SE between any of the "home" weekend nights ($p>0.05$; RM-ANOVA).

After the six hour phase shift, there were no significant differences in SOL, TWT, % stages 1, 2, % SWS, REM latency, and SE between any of the nights of the "away" weekend ($p>0.05$; RM-ANOVA).

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**Table 5.** *Descriptive statistics of polysomnographic recordings for "home" and "away" weekends*

| Weekend | "Home" Night 1 (n=5) | "Home" Night 2 (n=5) | "Home" Night 3 (n=5) | "Away" Night 1 (n=5) | "Away" Night 2 (n=5) | "Away" Night 3 (n=5) |
|---|---|---|---|---|---|---|
| **Sleep variable** | | | | | | |
| **Total sleep time (min)** | 388.2 ± 44.1 | 439.0 ± 48.3 | 351.6 ± 68.1 | **196.6 ± 57.0 #** | **394.6 ± 45.6 **** | **308.0 ± 65.0 *** |
| **Sleep onset latency (min)** | 29.3 ± 22.1 | 28.9 ± 22.5 | 29.6 ± 12.0 | 41.9 ± 24.9 | 32.6 ± 27.5 | 32.8 ± 35.1 |
| **REM latency (min)** | 99.1 ± 29.6 | 103.3 ± 48.6 | 102.1 ± 40.4 | 108.8 ± 31.4 | 107.1 ± 11.3 | 113.9 ± 108.9 |
| **Total wake time (min)** | 49.0 ± 25.8 | 40.4 ± 15.2 | 49.5 ± 22.4 | **96.5 ± 47.6 #** | **119.2 ± 47.4 #** | **158.9 ± 86.4 #** |
| **Stage 1 sleep (% TST)** | 9.4 ± 1.5 | 7.8 ± 1.9 | 8.2 ± 2.9 | 11.6 ± 1.9 | 11.2 ± 4.0 | 10.6 ± 2.4 |
| **Stage 2 sleep (% TST)** | 48.8 ± 5.3 | 54.6 ± 3.2 | 50.4 ± 4.6 | 51.6 ± 7.5 | 53.2 ± 2.2 | 47.0 ± 8.3 |
| **SWS (% TST)** | 21.6 ± 5.6 | 16.8 ± 5.9 | 23.6 ± 1.3 | 24.2 ± 8.3 | 19.6 ± 4.0 | 22.4 ± 6.7 |
| **REM sleep (% TST)** | 18.4 ± 4.8 | 21.2 ± 6.7 | 17.6 ± 2.6 | **12.6 ± 2.1 **#** | **15.6 ± 4.2 *#** | 20.0 ± 6.1 |
| **Sleep Efficiency (%)** | 78.4 ± 3.2 | 83.4 ± 5.0 | 80.2 ± 5.1 | **59.6 ± 11.1 #** | **68.8 ± 3.0 #** | **60.2 ± 15.5 #** |

Data are expressed as Mean ± SD. *p<0.05; **p<0.01; ***p<0.001 comparing nights within weekends for both "home" and "away" weekends, RM-ANOVA, Tukey Kramer post-hoc test. # p<0.05 comparing "home" night to corresponding "away" night, Paired *t* test. TST- total sleep time; REM- rapid eye movement; SWS- slow wave sleep.

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TST ($F_{14}$=15.1, p=0.002; RM-ANOVA) and % of REM sleep ($F_{14}$=6.7, p=0.02; RM-ANOVA) were significantly different for the three nights of the 'away' weekend. A significant decrease in TST was observed on night one compared to night two ($p<0.01$) and night three ($p<0.05$) of the "away" weekend. The % of REM sleep was significantly increased on night three when compared to night one ($p<0.01$) and night two ($p<0.05$) of the "away" weekend.

There was no significant differences in SOL, % stages 1, 2, and SWS and REM latency between the nights of the "home" weekend and corresponding nights of the "away" weekend ($p>0.05$; Paired *t* test).

While TST for all nights of the "away" weekend were shorter than the corresponding nights of the "home" weekend, only TST on night one of the "away" weekend was decreased significantly compared to night one of the "home" weekend ($t_4$=9.5, p=0.0007; Paired *t* test).

The amount of time spent awake during the sleep period on nights one ($t_4$=4.1, p=0.01; Paired *t* test), two ($t_4$=4.04, p=0.02; Paired *t* test) and three ($t_4$=2.9, p=0.04; Paired *t* test) of the "away" weekend was significantly higher compared to the corresponding nights of the "home" weekend.

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The % of REM sleep was significantly lower for night one ($t_4$=3.8, p=0.02; Paired *t* test) and night two ($t_4$=3.2, p=0.03; Paired *t* test) of the "away" weekend compared to nights one and two of the "home" weekend, respectively.

The six hour phase advance shift significantly decreased SE for nights one ($t_4$=4.1, p=0.01; Paired *t* test), two ($t_4$=4.8, p=0.009; Paired *t* test) and three ($t_4$=3.0, p=0.04; Paired *t* test) of the "away" weekend when compared to the corresponding nights of the "home" weekend.

### 3.2.3 Subjective assessment of sleep

Table 6 shows the subjective morning assessment data of sleep quality for the "home" and "away" weekends. There were no significant differences in refreshing sleep, awakenings, and morning alertness for both weekends (p>0.05; Friedman Test). The subjects reported that their time to fall asleep was significantly different on night two of the "away" weekend compared to nights one and three of the "away" weekend (Fr=10.3, p=0.006; Friedman Test). The time to fall asleep was significantly shorter on night two of the "away" weekend compared to night one (p<0.05) and night three (p<0.05) of the same weekend. There were no significant differences in refreshing sleep, time to fall asleep, awakenings, and morning alertness between the corresponding days of the "home" and "away" weekends (p>0.05; Wilcoxon matched-pairs signed-rank test).

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**Table 6.** *Subjective morning assessment of sleep quality for "home" and "away" weekends*

| Weekend | "Home" Morning 1 (n=10) | "Home" Morning 2 (n=10) | "Home" Morning 3 (n=10) | "Away" Morning 1 (n=10) | "Away" Morning 2 (n=10) | "Away" Morning 3 (n=10) |
|---|---|---|---|---|---|---|
| **Subjective data** | | | | | | |
| **Refreshing sleep** | 4.7 (3.4, 5.7) | 4.8 (3.4, 6.2) | 5.1 (4.1, 6.1) | 5.3 (3.5, 7.1) | 5.8 (4.8, 6.8) | 5.1 (4.2, 6.0) |
| **Time to fall asleep** | 6.2 (5.0, 7.4) | 4.9 (3.5, 6.3) | 5.3 (4.2, 6.4) | **6.2 (4.7, 7.7) *** | 4.0 (2.8, 5.2) | **5.3 (4.3, 6.3) *** |
| **Awakenings** | 6.6 (5.4, 7.9) | 5.1 (3.1, 7.1) | 4.9 (3.5, 6.3) | 5.7 (3.9, 7.5) | 5.1 (4.1, 6.1) | 6.0 (5.0, 7.0) |
| **Morning alertness** | 5.6 (4.1, 7.1) | 5.6 (4.2, 7.0) | 5.9 (5.6, 7.2) | 5.2 (3.4, 7.0) | 6.1 (4.7, 7.5) | 5.7 (4.4, 7.1) |

Data are expressed as Median (lower 95% CI, upper 95% CI). Data was analyzed using the Wilcoxon matched-pairs signed-ranked test and Friedman test (non-parametric RM-ANOVA) with a significance level of $p<0.05$. * $p<0.05$ comparing days within each weekend for both "home" and "away" weekends, Friedman Test, Dunn post-hoc test.

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### 3.2.4 Subjective sleepiness

Figure 5 shows the perceived sleepiness, as assessed using the Pictorial Sleepiness Scale questionnaire, throughout the day for days one and two of the "home" (Figure 5a) and "away" (Figure 5b) weekends.

Day one of the "home" weekend showed a significant gradual increase in subjective sleepiness throughout the wakeful period ($F_{29}$=11.3, p=0.0007; RM-ANOVA), with subjects feeling sleepier at 15:00 ($p<0.05$) and 21:00 ($p<0.001$) compared to 08:00 (Figure 5a). The subjects reported feeling sleepier, upon rising (08:00), on day two compared to day one of the "home" weekend ($t_9$=2.5, p=0.04; Paired *t* test) (Figure 5a).

Day one of the "away" weekend also showed a significant difference in sleepiness throughout the day ($F_{29}$=6.1, p=0.01; RM-ANOVA), with a significant increase in subjective sleepiness at 21:00 ($p<0.01$) compared to 08:00 (Figure 5b). The subjects reported feeling less sleepy, before bedtime (21:00), on day two compared to day one of the "away" weekend ($t_9$=3.5, p=0.007; Paired *t* test) (Figure 5b).

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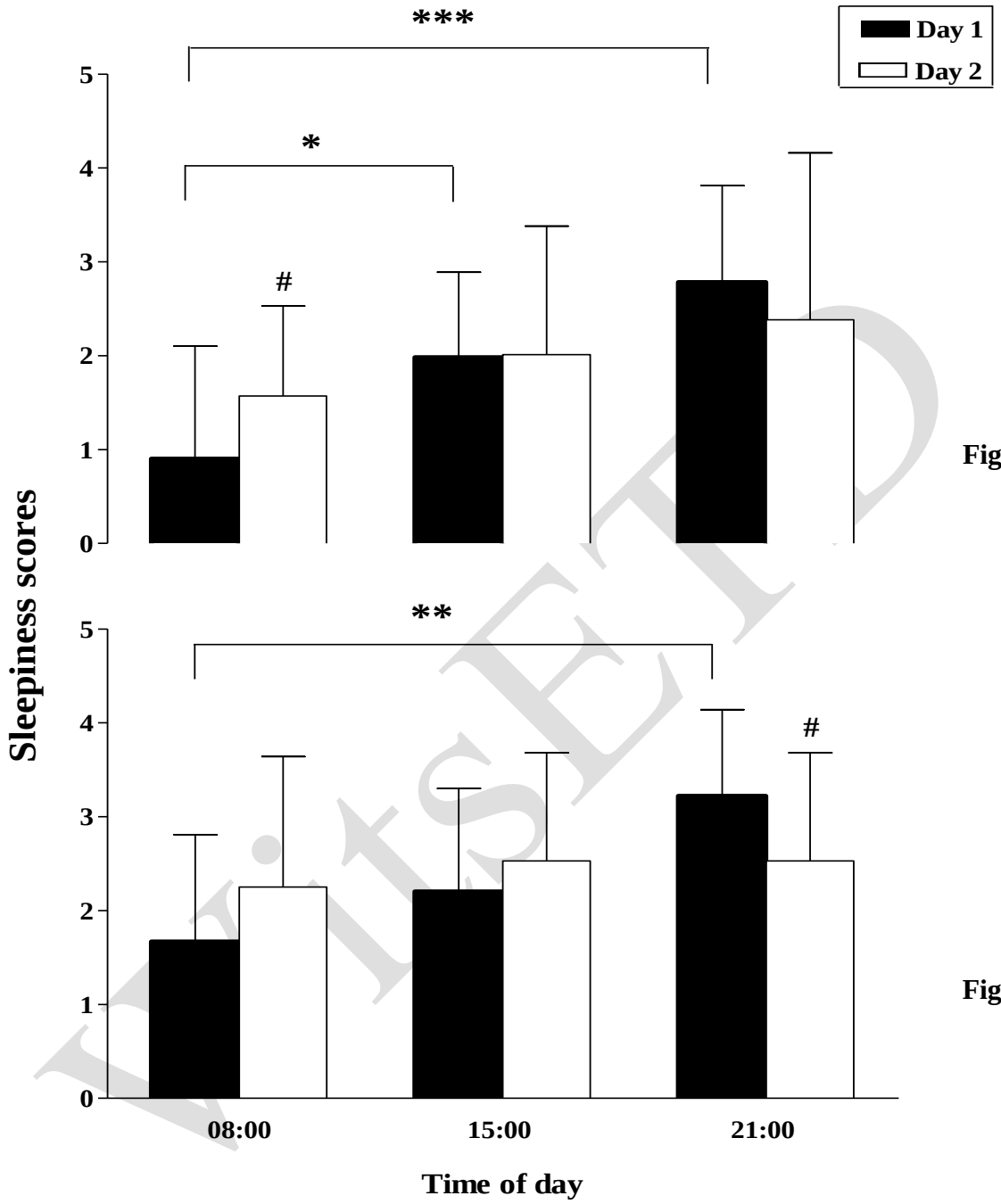



**Figure 5a**

**Figure 5b**

**Figure 5.** Subjective sleepiness during the day (Mean + SD) for days one and two of the "home" (Figure 5a) and "away" (Figure 5b) weekends. Time of day represents the actual laboratory clock time. The "home" weekend represents local time of day (Johannesburg, South Africa, GMT +2 hours). The "away" weekend time represents the athlete's perceived time of day, local "home" time advanced by six hours, being the correct times if the athletes had travelled across six time zones in an eastward direction (Perth, Australia, GMT +8 hours). *$p<0.05$, **$p<0.01$, ***$p<0.001$ comparing different times for each day of both weekends, RM-ANOVA, Tukey Kramer post-hoc test. # $p<0.05$ comparing day one to day two on both weekends at the same time, Paired *t* test.

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Figure 6 shows the subjective sleepiness of day one (Figure 6a) and day two (Figure 6b) for both weekends.

When comparing the actual time of day across weekends, there was a significant increase in subjective sleepiness in the morning, 08:00, for day one ($t_9$=2.4, p=0.04; Paired *t* test) (Figure 6a) and day two ($t_9$=2.7, p=0.02; Paired *t* test) (Figure 6b) of the "away" weekend compared to the corresponding time and day of the "home" weekend. There was no significant difference between the two weekends for any other time of day (p>0.05; Paired *t* test).

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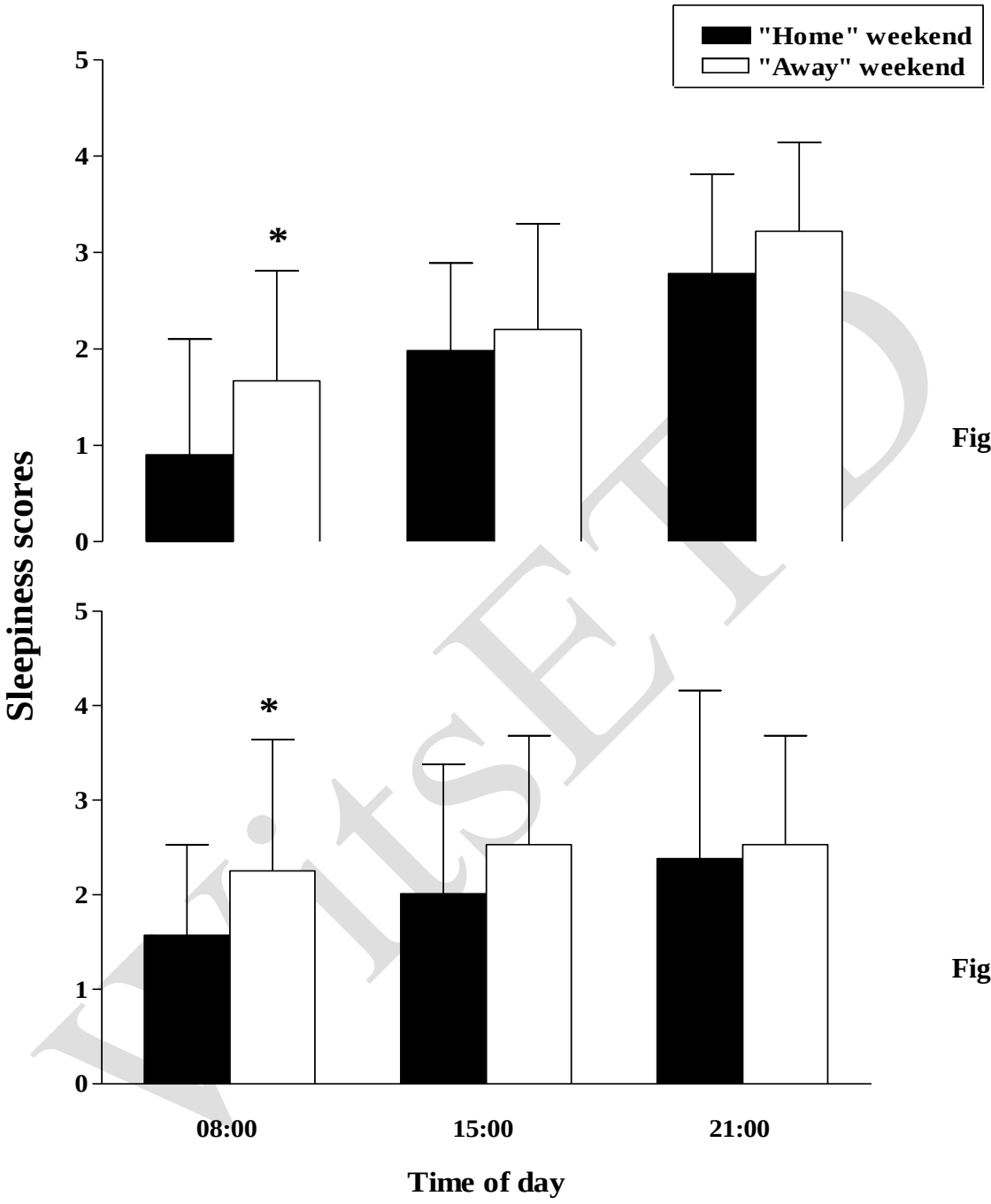



Figure 6a

Figure 6b

**Figure 6.** Subjective sleepiness during the day (Mean + SD) for day one (Figure 6a) and day two (Figure 6b) of both weekends. Time of day represents the actual laboratory clock time. The "home" weekend represents local time of day (Johannesburg, South Africa, GMT +2 hours). The "away" weekend time represents the athlete's perceived time of day, local "home" time advanced by six hours, being the correct times if the athletes had travelled across six time zones in an eastward direction (Perth, Australia, GMT +8 hours). * $p<0.05$ comparing different times for days one and two for both weekends, Paired *t* test.

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### 3.2.6 Mood states

Figure 7 shows the total mood disturbance (TMD) for each day of both weekends. The TMD analysis showed that there were significant mood changes between the days of each weekend. TMD was significantly higher on day one of the "away" weekend compared to day one of the "home" weekend ($p=0.002$; Wilcoxon matched-pairs signed-ranked test) with the subjects feeling more tense, fatigued, and confused on the first day of the "away" weekend. Similarly, TMD was significantly higher on day two of the "away" weekend compared to day two of the "home" weekend ($p=0.004$; Wilcoxon matched-pairs signed-ranked test) with the subjects feeling more fatigued on the second day of the "away" weekend.

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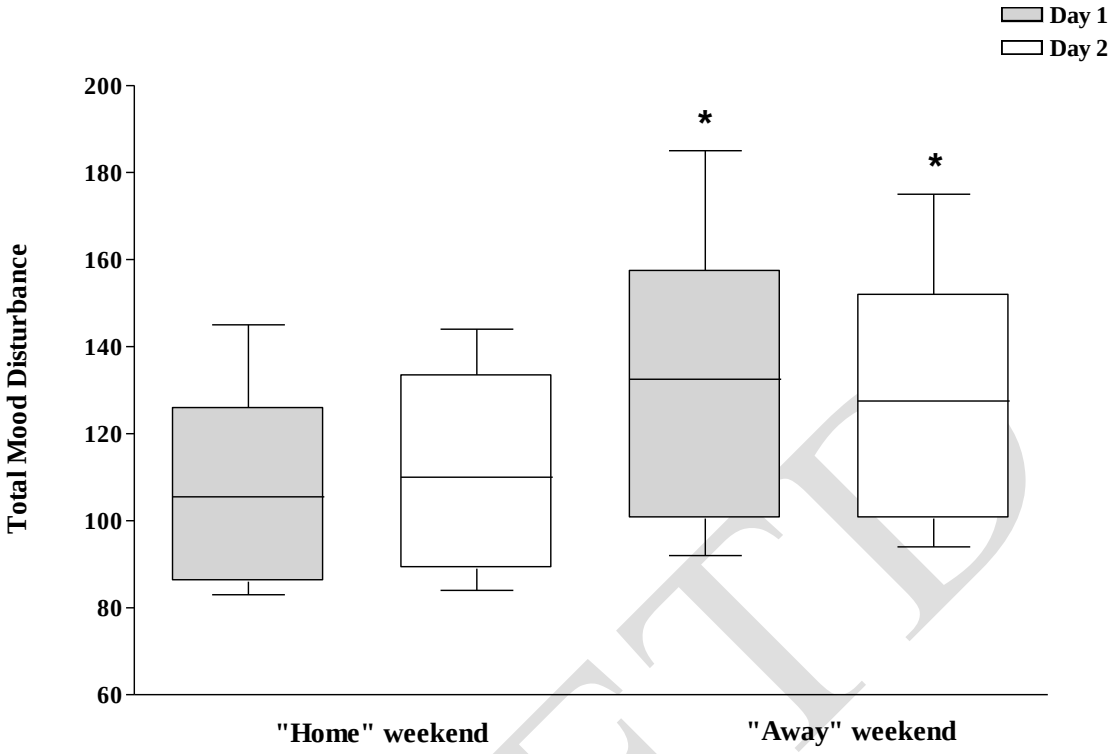



**Figure 7.** Total mood disturbance (TMD) of ten male subjects for both days of the "home" and "away" weekends. Data are expressed as Median $\pm$ CI. * $p<0.05$ when each day of the "home" weekend was compared to the corresponding day of the "away" weekend, Wilcoxon matched-pairs signed-ranked test.

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### 3.2.6 Simple reaction time

Table 7 shows the Psychomotor Vigilance Task (PVT) results for both days of the "home" and "away" weekends. The phase shift of six hours did not produce any significant differences for any of the PVT measures ($p>0.05$; Paired *t* test).

**Table 7.** *Characteristics of Psychomotor Vigilance Task (PVT) for "home" and "away" weekends*

| Weekend | "Home" Day 1 (n=10) | "Home" Day 2 (n=10) | "Away" Day 1 (n=10) | "Away" Day 2 (n=10) |
|---|---|---|---|---|
| **PVT Response** | | | | |
| **Mean RT (ms)** | 223.0 ± 21.1 | 226.6 ± 24.0 | 235.8 ± 32.5 | 238.0 ± 39.3 |
| **Number of false responses** | 35.4 ± 25.4 | 37.4 ± 29.4 | 38.0 ± 21.4 | 33.7 ± 22.1 |
| **Number of lapses (RT > 500ms)** | 0.6 ± 0.5 | 0.8 ± 0.7 | 0.9 ± 0.8 | 0.9 ± 0.7 |

Data are expressed as Mean ± SD. All data was analyzed using the Paired *t* test with a significance level of $p<0.05$. PVT- Psychomotor Vigilance Task; RT- reaction time.

### 3.2.7 Exercise

The change in the timing of the temperature circadian rhythm for the two weekends meant that exercise sessions were occurring at different times of the circadian phase of body temperature. Although the timing of the exercise sessions were fixed, the six hour phase advance shift meant that, in respect to the local laboratory "home" time (Johannesburg, South Africa, GMT +2 hours), exercise sessions were initiated between the hours of 10:00-12:00 for the "away" weekend compared to 16:00-18:00 for the "home" weekend. When combined with the shift in the body temperature

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rhythms that occurred due to phase advance shift, the exercise sessions over the "away" weekend occurred when the athlete's body temperature rhythm was on the rise, whereas the exercise sessions, over the "home" weekend, were initiated at or near the peak of body temperature (Figure 4). Table 8 shows the exercise data for each exercise session for both days of the "home" and "away" weekends.

There were no significant differences between day one and day two of the "home" weekend for upper and lower body muscular strength, anaerobic ability, anaerobic leg power, hand grip strength, aerobic endurance performance ($p>0.05$; Paired *t* test) and abdominal muscular endurance and RPE ($p>0.05$; Wilcoxon matched-pairs signed-ranked test). However, hamstring muscular flexibility significantly increased on day two compared to day one of the "home" weekend ($t_9=2.2$, $p=0.05$; Paired *t* test).

There were no significant differences between day one and day two of the "away" weekend for any of the exercise measurements for parametric data ($p>0.05$; Paired *t* test) and non-parametric data ($p>0.05$; Wilcoxon matched-pairs signed-ranked test).

There were no significant differences in anaerobic leg power, hand grip strength, hamstring muscular flexibility, aerobic endurance performance ($p>0.05$; Paired *t* test), and RPE ($p>0.05$; Wilcoxon matched-pairs signed-ranked test) between the corresponding days of the two weekends.

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**Table 8.** *Characteristics of exercise tests for "home" and "away" weekends*

| Weekend | "Home" Day 1 (n=10) | "Home" Day 2 (n=10) | "Away" Day 1 (n=10) | "Away" Day 2 (n=10) |
|---|---|---|---|---|
| **Exercise measures** | | | | |
| **Leg Press 1 RM (kg)** | 225.5 ± 96.1 | 242.5 ± 114.0 | 203.1 ± 72.1 | **200.2 ± 79.0 #** |
| **Bench Press 1 RM (kg)** | 66.5 ± 13.7 | 67.5 ± 14.7 | **62.0 ± 15.4 #** | **62.0 ± 15.2 #** |
| **Anaerobic ability (s)** | 29.3 ± 1.4 | 29.0 ± 1.7 | **30.5 ± 1.7 #** | **30.9 ± 2.4 #** |
| **Anaerobic leg power (cm)** | 203.0 ± 34.5 | 203.9 ± 31.8 | 197.2 ± 34.9 | 196.7 ± 38.7 |
| **Hamstring muscular flexibility (cm)** | 60.5 ± 2.5 | 61.2 ± 2.0 * | 59.0 ± 5.7 | 59.3 ± 4.6 |
| **Abdominal muscular endurance** | 26.0 (23.2, 29.6) | 27.0 (24.1, 31.7) | **22.0 (20.6, 27.2) #** | **23.5 (21.3, 28.1) #** |
| **Hand grip strength (kg)** | 46.6 ± 8.1 | 47.9 ± 6.8 | 46.2 ± 6.6 | 46.3 ± 4.8 |
| **Aerobic endurance (km)** | 2.9 ± 1.4 | 3.2 ± 1.8 | 3.0 ± 1.4 | 2.9 ± 1.4 |

Data are expressed as Mean ± SD, apart from abdominal muscular endurance expressed as Median (lower 95% CI, upper 95% CI). * $p<0.05$ when exercise sessions on day one were compared to day two of "home" or "away" weekends. # $p<0.05$ when the exercise session on a specific day of the "home" weekend were compared to the same exercise session on the "away" weekend day. All data was analyzed using Paired *t* test apart from abdominal muscular endurance (Wilcoxon matched-pairs signed-ranked test). 1RM- one repetition maximum.

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There was a significant decrease in lower body muscular strength, as measured using leg press 1RM, for day two of the "away" weekend compared to day two of the "home" weekend ($t_9$=2.4, p=0.04; Paired *t* test). A significant decrease in the weight lifted for the bench press was observed for day one ($t_9$=2.9, p=0.02; Paired *t* test) and day two ($t_9$=2.7, p=0.02; Paired *t* test) of the "away" weekend compared to corresponding days of the "home" weekend.

The athlete's anaerobic ability, as assessed using the 10x10 shuttle run, was better for the "home" weekend compared to the "away" weekend for both days. Day one ($t_9$=3.03, p=0.01; Paired *t* test) and day two ($t_9$=2.4, p=0.04; Paired *t* test) of the "away" weekend showed significantly slower shuttle run times in anaerobic performance compared to corresponding days of the "home" weekend.

Abdominal muscular endurance performance, as measured by the number of sit-ups completed in 30 seconds, was significantly lower on day one (p=0.008; Wilcoxon matched-pairs signed-ranked test) and day two (p=0.01; Wilcoxon matched-pairs signed-ranked test) of the "away" weekend when compared to the corresponding "home" weekend days.

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# CHAPTER FOUR

## *Discussion*

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## 4.1 Discussion

Athletes often travel across many time zones to compete in athletic events. As a result of transmeridian travel, their sleep patterns and athletic performance are often compromised. The primary aim of my study was to investigate the effects of a laboratory simulated six hour phase advance shift in the sleep-wake cycle on body temperature rhythms, objective and subjective sleep parameters, subjective sleepiness, mood states, and cognitive and exercise performance in male athletes. Ten male athletes spent two separate weekends in a controlled laboratory setting. On the one weekend, the athletes followed a local timeline (Johannesburg, South Africa, GMT +2 hours; "home" weekend), and on the other weekend the laboratory environment was altered to induce a six hour phase advance ("away" weekend), as would occur if they had travelled eastward by air across six time zones. The data within the same weekend was used to assess whether the laboratory simulated "home" weekend, and the phase advance shift ("away" weekend) had any effect on the circadian body temperature rhythm, sleep, sleepiness, mood, and cognitive and anaerobic exercise performance in the male athletes. The data between the corresponding day and night of the "home" and "away" weekends was analyzed to assess the magnitude of any observed changes in the measured variables, between the two weekends.

As expected, I did not observe any objective or subjective sleep disturbances, mood disturbances, or decreases in exercise performance for the "home" weekend. The athlete's body temperature circadian rhythm followed a normal pattern over the

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weekend; the peak in body temperature occurred at approximately 20:00 and the lowest body temperature occurred between 03:00-05:00 (Figure 4). After the six hour phase advance, the athlete's body temperature, which was entrained to the Johannesburg time-line (GMT +2 hours), was desynchronized with respect to the "new" time-line. The peak in body temperature now occurred at approximately 16:00, and the lowest body temperature occurred at approximately 01:00 (Figure 4). Objective and subjective measures of sleep and exercise performance were disrupted after the six hour phase advance shift. On the "away" weekend, the athletes had a lower percentage of REM sleep on nights one and two compared to night three of the same weekend (Table 5). Also, the athletes felt that they took longer to fall asleep on nights one and three of the "away" weekend compared to night two of the same weekend (Table 6). The athletes felt less sleepy on night three of the "away" weekend compared to night two of the same weekend (Figure 5b). The athlete's athletic performance decreased during the "away" weekend; their upper body, lower body and abdominal muscle strength, as well as anaerobic ability, was lower on the second day of the "away" weekend, compared to the first day of the same weekend (Table 8). There was no change in their mood states, as assessed by the Profile of Mood States (POMS) questionnaire, or cognitive ability, as measured by a visual reaction time test, on the days of the "away" weekend.

A comparison between the corresponding days and nights of the two weekends revealed that sleep architecture, athletic performance, and mood were negatively affected by the six hour phase advance shift. Compared to the "home' weekend, total

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sleep time, percentage of REM sleep and sleep efficiency decreased, and total wake time during the sleep period increased after the phase shift. Compared to the "home" weekend, the phase shift of six hours negatively affected mood states, as assessed by the Profile of Mood States (POMS) questionnaire, and subjective measures of sleepiness, as assessed by Pictorial sleepiness scale. Athletic performance was decreased after the phase shift; compared to the "home" weekend; muscular strength (upper and lower body), abdominal muscular strength, and anaerobic ability decreased after the phase shift.

I managed to recruit ten male athletes who successfully fulfilled all the criteria for participation in the study, and who were prepared to spend two weekends (six nights in total) away from home, confined in the sleep laboratory. I acknowledge that my sample size and the duration of the laboratory simulated phase shift were relatively small and short, compared to other studies where laboratory simulated phase shifts were induced for longer periods of time (Hume 1980; Monk *et al*. 1988; Monk *et al*. 1993; Carrier *et al*. 1996). More significant results with respect to changes in certain sleep variables and physical measures of athletic performance after a six hour phase advance shift might be obtained with a larger sample size and a longer experimental period. However, a longer experimental period may also reverse the changes seen in my study as the subjects adapt to the phase shift. Additionally, the choice of exercise tests (based on availability of equipment and size of the experimental area) may not be directly relevant to the sporting community (e.g. hand grip strength). Furthermore, the choice of cognitive performance testing, as measured by reaction

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time, may have been ineffective in my study, although the Psychomotor Vigilance Task (PVT) has been shown in other studies to be sensitive to the effects of sleep deprivation (Dinges *et al*. 1997; Van Dongen and Dinges 2003). In my study, a more effective measure of cognitive performance, such as search or memory tasks, may have produced a greater change in cognitive performance after the six hour phase advance shift. As a result of technical problems (external interference, rectal thermistor insertion difficulties), some data was lost for temperature recordings and objective measurements of sleep. A full data set may have revealed more, or even greater, changes in sleep variables, mood states, cognitive and athletic performance after the phase advance shift. However, despite these limitations, I was still able to effectively use a laboratory setting to induce a six hour phase advance shift in the athletes, and measure the effect of the phase advance shift, and the subsequent desynchronization of the body temperature circadian rhythm, on their sleep patterns, mood and athletic performance.

Several other studies have shown a desynchronized circadian rhythm, by measuring melatonin and body temperature rhythms, after a laboratory phase shift (Monk *et al*. 1988; Monk *et al*. 1993; Carrier *et al*. 1996; Deacon and Ardent 1996), or after transmeridian travel (Suvanto *et al*. 1993a; Daurat *et al*. 2000; Edwards *et al*. 2000; Caufriez *et al*. 2002; Lemmer *et al*. 2002; Beaumont *et al*. 2004). Since body temperature plays an important role in the sleep-wake cycle, a change in body temperature circadian rhythm, as would occur after transmeridian travel, can have a negative impact on sleep patterns (Czeisler and Khalsa 2000). In my study, the six

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hour phase advance shift had a negative impact on objective (PSG) and subjective (questionnaire) measures of sleep, both within the "away" weekend and between corresponding nights of both weekends.

Homeostatic (Process S) and circadian (Process C) processes determine the initiation and maintenance of sleep at different times of the day (Borbély 1982; Graeber 1994; Dijk and Czeisler 1995; Lack and Lushington 1996; Murphy and Campbell 1997; Borbély and Achermann 2000). Typical nighttime sleep is initiated when Process S is at its peak and Process C is at its trough; whereas awakening from sleep occurs when Process S is at its trough and Process C is beginning to rise (Borbély and Achermann 2000). However, with a rapid change in time zones, circadian rhythms take time to adapt to the new time zone, and sleep episodes will be initiated at different times of the phase of body temperature circadian rhythm (Shephard 1984; Graeber 1994; Haimov and Arendt 1999).

On the nights of the "away" weekend, sleep was scheduled at or near the peak of the body temperature rhythm (Figure 4), which may have resulted in the reduced total sleep time on the three nights. However, when comparing the corresponding nights of the two weekends, total sleep time was only significantly decreased on night one of the "away" weekend, although total sleep time for night two and three of the "away" weekend were lower than the corresponding "home" weekend nights. Other investigators have reported a decrease in total sleep time after a time zone transition (Daurat *et al*. 2000; Jamieson *et al*. 2001; Takahashi *et al*. 2002) or after laboratory

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phase shifts (Preston *et al*. 1973a; Hume 1980; Monk *et al*. 1988; Carrier *et al*. 1996; Deacon and Arendt 1996). In contrast to these studies, O'Connor *et al*. (1991) observed an increase in total sleep time in elite swimmers after eastward travel across four time zones. However, O'Connor *et al*. (1991) concluded that sleep was confounded by the intensity of training schedules before and after travel.

The duration of time spent awake during the sleep episode, as assessed by PSG recordings, although not significantly increased during the "away" weekend, was significantly increased for all nights of the "away" weekend compared to corresponding "home" weekend nights. Sleep initiated during the rising phase, or peak of the body temperature circadian rhythm, will be shorter and associated with more awakenings in the early part of the sleep episode (Graeber 1994; Arendt *et al*. 2000). Following eastward travel, as simulated in my study, sleep was scheduled in advance of the 'home' bedtime, corresponding to the rising phase or peak of the temperature circadian rhythm (Graeber 1994; Haimov and Arendt 1999). An increased number of awakenings, in the first part of the sleep episode, are characteristic after a phase advance (Monk *et al*. 1988; Monk *et al*. 1993; Carrier *et al*. 1996) or after eastward travel (Lowden and Åkerstedt 1998; Lowden and Åkerstedt 1999; Jamieson *et al*. 2001; Takahashi *et al*. 2002). The increased number of awakenings during the "away" weekend nights is reflected in the sleep efficiency which, when compared to the corresponding "home" weekend nights, was significantly decreased on all the "away" weekend nights. Decreased sleep efficiency, or poor sleep quality, has been observed in young and middle-aged

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subjects after a six hour phase advance shift (Monk *et al*. 1993; Carrier *et al*. 1996) and after transmeridian travel (Lowden and Åkerstedt 1999; Beaumont *et al*. 2004).

The six hour phase advance of the sleep-wake cycle also decreased the percentage of REM sleep on the "away" weekend nights, and on night one and night two of the "away" weekend compared to the corresponding "home" weekend nights. REM sleep is controlled by an endogenous circadian oscillator which is coupled to the temperature circadian rhythm (Moore-Ede *et al*. 1982; Czeisler *et al*. 1980a; Czeisler *et al*. 1980b; Zulley 1980; Borbély and Achermann 2000). Thus, REM sleep propensity occurs in the latter part of the sleep episode (early morning), corresponding to the trough or ascending limb of the temperature rhythm (Czeisler *et al*. 1980b; Graeber 1994). In my study, the percentage of REM sleep was significantly reduced for nights one and two of the "away" weekend compared to corresponding "home" weekend nights. After a simulated phase advance shift or transmeridian travel, the timing of REM sleep propensity is out of phase with the body temperature circadian rhythm (Winget *et al*. 1985; Monk *et al*. 1988). Similar reductions in REM sleep have been observed in laboratory simulated phase shifts (Hume 1980; Monk *et al*. 1988; Moline *et al*. 1992; Monk *et al*. 1993) and after transmeridian travel (Beaumont *et al*. 2004). Thus, the change in the sleep-wake cycle by six hours prevented the subjects from consolidating the full amount of REM sleep as waking times were earlier than normal.

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In my study, an increase in the percentage of REM sleep was observed on the third night of the "away" weekend compared to nights one and two of the same weekend. A 'rebound effect' of REM sleep has been observed several nights after a six hour phase shift (Monk *et al*. 1988) and after field studies of jet lag (Beaumont *et al*. 2004). Beaumont *et al*. (2004) observed a compensatory increase in REM sleep, on the third night, after eastward travel across seven time zones. Monk *et al*. (1988) suggested that after a phase shift, sleep parameters follow a 'zig-zag' recovery pattern representing an internal struggle between the circadian and homeostatic processes which will further disrupt sleep and performance measures. Monk *et al*. (1988) observed a 'zig-zag' recovery pattern of total sleep time, slow wave sleep and REM sleep after a six hour phase advance, and complete recovery of REM sleep was achieved eight to nine days after the phase shift. Thus, the compensatory increase in the percentage of REM sleep, as seen in my study, on night three post-shift, further demonstrates the misalignment of the circadian rhythm with the new time schedule.

In my study, after the six hour phase advance shift, subjective assessment of sleep quality data such as 'refreshing sleep', 'number of awakenings', and perceived time to fall asleep did not reflect the objective measurements of sleep (PSG). Compared to the corresponding "home" weekend nights, subjective quality of sleep did not differ after the phase shift, thus athletes perceived that they had a relatively good night's sleep. It is interesting to note that the athletes perceived their sleep after the phase shift as not being different from the nights of the "home" weekend. However, compared to corresponding nights of the "home" weekend, objective measurements

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of sleep (PSG) such as reduced percentage of REM sleep, sleep efficiency, and increased total wake time during the sleep period were different on all nights of the "away" weekend. The discrepancies in the objective (PSG recordings) and subjective (questionnaires) measurements of sleep suggest that people are inaccurate in assessing their own sleeping patterns (Baker *et al*. 1999; Richmond *et al*. 2004). Thus, these findings highlight the importance of using objective measurements, in addition to subjective measures, to investigate the effect of transmeridian travel on sleep patterns. It is also unclear whether the objective or subjective criteria are more important when assessing the impact of changes in sleep on daytime performance.

A reduction in sleep time, changes in sleep architecture, and poorer sleep quality may influence daytime sleepiness, mood states and performance. In my study, subjective sleepiness during the day for both weekends was assessed using the Pictorial Sleepiness Scale. After the phase shift, the athletes reported feeling less sleepy on night three of the "away" weekend compared to night two of the "away" weekend. This would be expected since the 'body clock' is still entrained to local Johannesburg, South Africa (GMT +2 hours) time (16:00) and the body temperature rhythm was at its peak prior to bedtime over the "away" weekend, which correlates to high levels of alertness (Van Dongen and Dinges 2000). Upon rising, sleepiness was significantly higher on both days of the "away" weekend compared to corresponding "home" weekend days. Morning sleepiness might be attributed to the lack of recuperative sleep, sleep loss, reduced sleep quality, or altered sleep architecture (Monk *et al*. 1988; Lowden and Åkerstedt 1999; Beaumont *et al*. 2004).

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Furthermore, researchers have shown that the circadian rhythm of sleepiness is highest at the trough of the body temperature rhythm (Lack and Lushington 1996; Murphy and Campbell 1997). In my study, after the phase shift, the morning waking time occurred at a trough in the body temperature rhythm. Similar findings have been reported after simulated phase shifts (Monk *et al*. 1988; Deacon and Arendt 1996) and after transmeridian travel (Lowden and Åkerstedt 1999; Beaumont *et al*. 2004).

The circadian rhythm of cognitive performance mimics the body temperature circadian rhythm; a higher body temperature is associated with increased alertness and better cognitive performance (Johnson *et al*. 1992; Van Dongen and Dinges 2000; Wright *et al*. 2002). In my study, simple reaction time, as measured by the Psychomotor Vigilance Task (PVT), was unchanged during the "away" weekend. In contrast to my study, cognitive performance has been shown to decrease after simulated phase shifts and after transmeridian travel (Preston *et al*. 1973a; Tapp and Natelson 1989; Deacon and Arendt 1996; Cho *et al*. 2000). Other studies have shown that reaction time, as measured using the PVT, is sensitive to the effects of sleep deprivation (Dinges *et al*. 1997; Van Dongen and Dinges 2003). Thus, in my study, the absence of a significant effect of the phase shift on PVT measures in the athletes may be a result of the lack of sleep deprivation induced by the protocol. Additionally, the PVT test may not have been sensitive enough to detect cognitive performance changes after a phase shift.

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In my study, I assessed the impact of the six hour phase advance shift of the sleep-wake cycle on physical measures of athletic performance such as muscular strength, anaerobic ability, anaerobic leg power, hamstring flexibility, abdominal muscular endurance, hand grip strength, and aerobic endurance. Physical measures of athletic performance were not different on the days of the "home" weekend and thus no learning or training effect of the exercise tests was observed. In comparison, muscular strength, anaerobic ability, and abdominal muscular endurance were lower on both days of the "away" weekend compared to the corresponding days of the "home" weekend. These changes in anaerobic athletic performance may have occurred due to the changes in body temperature during the two exercise sessions.

It has generally been accepted that athletic performance tends to follow a circadian rhythm which is closely related to the circadian body temperature rhythm (Reilly 1994). For instance, nerve conduction is faster, and muscular contractile properties are optimal at higher body temperatures (38.3°C) (Åstrand *et al*. 2003). Poorer athletic performance after transmeridian travel may be attributed to competing in athletic events, or performance of exercise testing, at inappropriate phases of the temperature circadian rhythm, when temperature is lowest (Jehue *et al*. 1993; Recht *et al*. 1995; Youngstedt and O'Connor 1999).

In my study, athletic performance was measured at different times of the body temperature circadian rhythm for the "home" and "away" weekends. A better performance was expected for the "home" weekend as the two exercise sessions

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coincided with the 'peak performance window'. After the phase advance shift, the assessment of athletic performance was conducted at a lower body temperature compared to the "home" weekend (Figure 4). Typically, the majority of competitive events are scheduled in the evening, coinciding with the 'peak performance window' where performance is optimal (Shephard 1984; Reilly 1994; Youngstedt and O'Connor 1999; Drust *et al*. 2005). However, after transmeridian travel, this performance window is shifted, and teams are more likely to lose evening games as a result of a shift in the 'peak performance window' (Jehue *et al*. 1993; Recht *et al*. 1995). After eastward travel across six time zones, as simulated in my study, an event scheduled in the afternoon or evening in the new time zone would correspond to 10:00 'body' clock time and a poorer performance would be expected. Thus, the importance of the 'peak performance window' has to be acknowledged as athletes travelling across time zones may be at a disadvantage as their 'body clock' is still entrained to home time.

In my study, the decline in muscular strength, anaerobic ability, and abdominal muscular endurance occurred on both days after the six hour phase advance shift of the sleep-wake cycle. My results are in agreement with Wright *et al*. (1983) where the performance of soldiers in a 270m sprint decreased by 8-12% following eastward travel. Hill *et al*. (1993) reported a decline in anaerobic power of 14% and anaerobic capacity of 6% for up to two days after eastward travel across six time zones. After transmeridian travel, circadian rhythms are desynchronized and in turn athletic ability may be impaired until re-entrainment is established. The disruption of circadian

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rhythms associated with transmeridian travel may contribute to changes in the excitation and contraction properties of the muscle fibre (motor control and fibre recruitment), hormonal secretions (cortisol), and metabolic functions (body temperature), which may result in poorer performances (Wright *et al*. 1983; Winget *et al*. 1985; Loat and Rhodes 1989; Hill *et al*. 1993; Reilly 1994).

Athletic performance not only depends on physiological processes, but is susceptible to the psychological status of athletes (Winget *et al*. 1985; Hardy and Jones 1994). After the six hour phase shift, a marked disruption in mood states, as measured by the POMS questionnaire, was reported on both days of the "away" weekend compared to corresponding "home" weekend days. Researchers have reported that a reduction in anaerobic performance, as was the case in my study, can occur as a result of increased feelings of fatigue, sleepiness and reduced motivation and willingness to participate in exercise sessions (Wright *et al.* 1983; Hill *et al*. 1993). Also, partial sleep deprivation studies have shown a decline in upper and lower muscular strength, and explosive tests, as a result of decreased quality of mood similar to mood disturbances that occur after transmeridian travel (Reilly and Piercy 1994; Takeuchi *et al*. 1985). Thus, a combination of the objective changes in sleep, the worsening of mood states and an increased fatigue may have reduced the anaerobic athletic performance in my subjects.

I believe that the results of my study are of interest and relevance to athletes who have to travel across time zones to participate in athletic events. My study used a six

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hour phase advance shift of the sleep-wake cycle to simulate eastward air travel across six time zones. As a result of the desynchronization of the body temperature circadian rhythm, the athletes' anaerobic ability and muscular strength was decreased after the simulated jet lag, and this could decrease athletic performance in the field. Even though, in my study, the assessment of physical measures of performance may not be sport-specific, some results may be relevant to actual performance in competitive events. For instance, professional rugby players require muscular power, muscular strength, and aerobic and anaerobic power to participate in games (Brewer and Davis 1995). During a rugby game, players are required to interchange between aerobic activity and intense anaerobic activities such as sprinting and tackling (Brewer and Davis 1995). After eastward travel across time zones, as simulated in my study, upper and lower body muscular strength, muscular endurance and anaerobic sprinting performance were compromised; thus a poorer performance may be expected in rugby players in some components of the game. Athletes, coaches and sports management should be made aware of the negative effects of transmeridian travel on sleep, mood, and athletic performance, so that they would be able to schedule athletic events at the best times of day.

To my knowledge, this is the first study to investigate the interaction between multiple variables such as body temperature, objective and subjective sleep parameters, mood states and athletic performance after a laboratory simulated phase shift in athletes. Even though some of the physical measures of athletic performance are relevant to all-round sport success, future studies need to employ more controlled,

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sport-specific performance tests to investigate the impact of simulated phase shifts on athletic performance in elite athletes. Unfortunately, as a result of time constraints, I was unable to induce 'jet lag' for a longer experimental period. Thus, it would be interesting to investigate the readjustment time of the sleep patterns, mood states, and athletic performance after a phase shift in athletes over a longer period of time.

Furthermore, it would be interesting to investigate the impact of transmeridian travel in elite athletes in actual sporting events. Within a controlled laboratory setting, I was unable to impose the wide range of physiological, psychological, and behavioural variables that an athlete may be exposed to after transmeridian travel, and the consequences of travel 'fatigue' itself on athletic performance. Thus, some of the symptoms associated with jet lag such as fatigue, loss of motivation or irritability may be overridden by the enthusiasm and excitement of an actual sporting event. Only by tracking athletes through the travel and the build-up to an actual sporting event could the impact of these variables be analyzed.

In conclusion, I successfully induced 'jet lag' within a controlled laboratory environment. A six hour phase advance shift of the sleep-wake cycle disrupted objective sleep parameters, negatively altered mood states, and resulted in a poorer anaerobic athletic performance. The deterioration of physical measures of athletic performance after a six hour phase advance shift indicates that there is a strong circadian influence which may be relevant to success in athletic performance after transmeridian travel.

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