THE EFFECTS OF BODY TEMPERATURE AND OXYGEN CONSUMPTION ON SLEEP ARCHITECTURE

THE EFFECTS OF BODY TEMPERATURE AND OXYGEN CONSUMPTION ON SLEEP ARCHITECTURE

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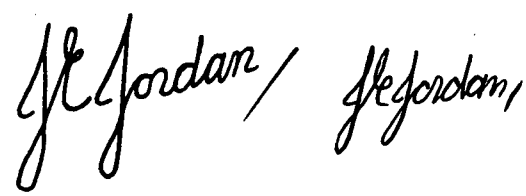
I certify that this thesis contains no material which has been accepted for the award of any other higher degree or graduate diploma in any university, and that to the best of my knowledge the thesis contains no copy or paraphrase of material previously published or written by another person, except where due reference is made in the text of the thesis.



ABSTRACT

Two related theories have had a substantial impact on our understanding of the nature and function of sleep. These are the energy conservation and restorative theories of sleep. Both theories predict an increase in both slow wave sleep (SWS) and sleep duration following increased energy expenditure. These predictions form the background to the empirical work of this thesis, and it is the effect of wake-period metabolism on both sleep metabolism and architecture that holds the main research focus.

Three studies were designed to evaluate the effects of either metabolic rate (MR), body temperature (T_b), or both, on sleep architecture, in particular SWS. The first and second studies imposed variations in waking activity in order to assess the effects of MR and T_b on sleep architecture. The first study actively increased MR by physical exercise, while the second study used passive heating in a warm bath. The third study compared T_b, MR and sleep in endurance athletes and sedentary individuals.

In the first study, the first sleep cycle of 10 young fit subjects (mean age = 21.8 years) was assessed after a 19km run, either immediately before bedtime or a few hours before retirement. There were four conditions: a no exercise condition; a late afternoon exercise session with evening meal; a late afternoon exercise session without evening meal; and a late evening exercise session with an evening meal. The results showed no evidence of an exercise-induced SWS effect, and found that exercise transiently increased wake-period MR which returned to control levels by bedtime. Furthermore, there was some evidence to suggest that a rise in wake-period energy expenditure may have a negative effect upon sleep properties.

The second study investigated the effect of passive heating on sleep architecture by using a method similar to that described by Horne and Staff (1983), Sewitch (1987) and Berger and Phillips (1988a,b).

It also tested the hypotheses that SWS levels increase following heating due to either a compensatory drop in T_b at sleep onset (Sewitch, 1987), or a sustained elevation in absolute T_b at sleep onset, and during sleep (Berger & Phillips, 1988; Berger, Palca, Walker & Phillips, 1988). Five healthy young male subjects (mean age= 20.4 years) were passively heated in a 42-43°C warm bath to induce elevated T_b and MR. A repeated measures design with two conditions was employed. These conditions included a control (no passive heating), and a passive heating condition in the late afternoon. Rectal temperature (T_{re}) was monitored from the early afternoon until the awakening period on the following morning. Metabolic rate was recorded for 20 minutes prior to, and immediately after the passive heating, and then across the sleep period. Sleep recordings also were monitored over the night. Results showed that passive heating significantly increased T_{re}, MR and SWS levels. Rectal temperature increases were sustained into, and across the sleep period, whereas MR increases were only transient and did not continue into the sleep period. SWS levels were significantly elevated in the first 150 minutes of sleep. The direct relationship of T_b to SWS supported the theory proposed by Berger and Phillips (1988a,b).

The third and final study determined whether the characteristically higher SWS levels and longer sleep durations of endurance athletes (Trinder, Paxton, Montgomery & Fraser, 1985) may be attributable to the effect of T_b on sleep. It was designed to assess the role of T_b on sleep by comparing the laboratory sleep of endurance athletes and sedentary individuals with T_b at sleep onset held constant between the two groups based upon evidence from another study (Hedges, 1989) that showed higher average T_{re}s and earlier sleep onset times in athletes compared to sedentary individuals. It was thus considered that the higher T_{re}s reported for athletes in this study may have been a consequence of their earlier sleep onset times. In the final study eight male endurance athletes (mean age= 21.5 years) and eight male non-athletes (mean age= 22.6 years) were compared under conditions of no-exercise.

The results showed SWS levels to be higher and sleep duration longer in the endurance athletes as compared to sedentary subjects, despite T_b at sleep onset being the same for the two groups, as a result of sleep onset being held constant. The results suggest elevated T_b at sleep onset may not be the mechanism causing particular sleep characteristics of endurance athletes. Rather it is proposed that the sleep properties of endurance athletes are due to a phase delay of the circadian oscillator, which in this group is achieved by an advance of the sleep-wake cycle (earlier usual sleep onset time).

In conclusion, it is argued that there is a relationship between metabolism and sleep architecture where SWS can be facilitated by either (a) high metabolism during sleep onset and the early part of sleep; or (b) the phase angle in the circadian temperature rhythm at sleep onset, or both.

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Do but consider what an excellent thing sleep is:
it is so inestimable a jewel that, if a tyrant would give his crown for an hour's slumber, it cannot be bought:
of so beautiful a shape is it, that though a man lie with an Empress, his heart cannot beat quiet till he leaves her embracements to be at rest with the other:
yea, so greatly are we indebted to this kinsman of death, that we owe the better tributary, half of our life to him:
and there is good cause why we should do so:
for sleep is that golden chain that ties health and our bodies together.
Who complains of want? of wounds? of cares? of great men's oppressions? of captivity? whilst he sleepeth? Beggars in their beds take as much pleasure as kings:
can we therefore surfeit on this delicate Ambrosia? Can we drink too much of that whereof to taste too little tumbles us into a churchyard, and to use it but indifferently throws us into Bedlam? No, no look upon Endymion, the moon's minion, who slept three score and fifteen years, and was not a hair the worse for it.

THOMAS DEKKER

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CHAPTER 1

INTRODUCTION

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INTRODUCTION

There are many theories of sleep function. They vary in their breadth of explanation; some attempting to give a global explanation of sleep, while others concentrate on particular aspects of sleep, such as rapid-eye movement (REM) sleep. However, it is clear that sleep does not serve just one function and a complete understanding of sleep requires a description of the full range of the functional roles of this state.

Two theories have had a substantial impact on our understanding of the nature and function of sleep. These are the energy conservation and restorative theories (Adam & Oswald, 1977, 1983; Berger, 1975; Berger & Phillips, 1988a; Feinberg, 1974; Hartmann, 1973; Horne, 1977, 1979, 1980, 1981, 1983, 1988, 1989; Oswald, 1969, 1974, 1980; Zepelin & Rechtschaffen, 1974). The two theories are similar in several respects such that empirical evidence is often supportive of both theories. Both argue that sleep primarily serves a compensatory role in respect to wakefulness, restoring bodily processes following daily activity, and both claim that stages 3 and 4 of slow-wave sleep (SWS) are particularly important for energy conservation and restoration.

There are two variants of the restorative theory; the bodily restorative (Adam & Oswald, 1977; Hartmann, 1973; Oswald, 1974), and the central nervous system (CNS) restorative theories (Feinberg, 1974; Horne, 1979, 1981, 1983, 1988, 1989). The first proposes that the sleep process is a necessary state for restoration of total body function including the CNS, while the other suggests that sleep is essential for the restorative processes of the CNS. Central to both theories, is the argument that energy expenditure is reduced during sleep in compensation to previously heightened daytime metabolism.

The inter-relationships between wake-period metabolism, sleep metabolism and sleep architecture hold particular relevance to the current thesis. These inter-relationships are investigated in three studies that directly or indirectly manipulate wake-period metabolism. Before reporting the experiments, important theoretical and methodological issues are considered in Chapters 2, 3, and 4.

Chapter 2 reviews the status of restorative and energy conservation theories, and considers in particular the relationship between MR and SWS. Chapter 3 reviews the adaptive metabolic changes associated with physical aerobic training and considers how they may contribute to SWS changes. It also outlines the changes in metabolic activity during sleep, and considers how such processes are affected by exercise. Chapter 4 presents the relationship between thermoregulation and sleep, focusing on the role of T_b and sleep.

The first study used exercise as an active means of MR elevation to assess the relationship between MR before and during sleep, and SWS, and is reported in Chapter 5. A second study reported in Chapter 6 used a passive method of elevating wake-period metabolism in order to assess the relationship between metabolism and sleep architecture. This study investigated the effects of body heating on T_b, MR and SWS during the sleep period. Such an investigation allowed closer examination of the effect of increased wake-time T_b, and possibly cerebral temperature, on both sleep metabolism and architecture through thermoregulatory changes. This is particularly relevant, since passive heating has previously been reported to be associated with changes in SWS (Bunnell, Agnew, Horvath, Jopson & Wills, 1988; Bunnell & Horvath, 1985; Horne & Moore, 1985; Horne & Reid, 1985; Horne & Shackell, 1987; Horne & Staff, 1983). This implies a direct relationship between thermoregulatory metabolism and sleep architecture. Chapter 7 reports the third study which compared the MR, T_b and sleep architecture of athletes and sedentary individuals.

Finally, the experimental outcome of the three research studies is assessed in Chapter 8. In addition, the implications of the

findings for metabolism and sleep architecture, as well as proposals for future investigation, will be presented.

CHAPTER 2

THEORIES OF SLEEP FUNCTION

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Both energy conservation and restorative theories of sleep were introduced in Chapter 1. Since this thesis is concerned with relationships whose origins are derived from these theories, an outline of the theories, with particular emphasis upon the nature of the relationship between MR and SWS, will be presented.

THE ENERGY CONSERVATION HYPOTHESIS

There have been a number of proponents of the energy conservation view including Allison and Van Twyver (1970), Snyder (1966), Zepelin and Rechtschaffen (1974), Horne (1977), Walker and Berger (1980) and Shapiro (1982). However, a large and recent contribution to this hypothesis has been the work of Berger (1975, 1984); Walker and Berger (1980); Berger, Palca, Walker and Phillips, (1988); and Berger and Phillips (1988a). Much of the evidence supporting the energy conservation hypothesis is derived from phylogenetic and ontogenetic comparisons of sleep data. These will be described in the following sections where the relationship of MR and sleep will also be considered.

Phylogenetic Comparisons

A major concern for all organisms, in particular homeothermic animals, is to attain and maintain an energy balance suitable for maximal functioning of bodily processes (Berger, 1975). The homeotherms are organisms which generate heat through high rates of energy metabolism that allows them to maintain a relatively constant T_b within the range of 36-40°C (Berger, 1984). In order to maintain energy balance, homeotherms need to balance food intake, activity, and other processes involved in metabolism. One of the processes involved in the energy equation is sleep. Berger (1984) postulates that sleep is a state of energy conservation. During this

period the organism is forced to remain relatively immobile ensuring that it undergoes minimal energy expenditure, providing periods of lowered T_b during sleep (Allison & Van Twyver, 1970; Berger, 1984; Kreider & Buskirk, 1957; Milan & Evonuk, 1967; Snyder, 1966; Walker & Berger, 1980). Thus, it is advocated that sleep's essential function is the reduction of energy expenditure below the level attainable by rest alone.

It is the homeotherms, such as birds and mammals, with their advanced cortical development, that have been found to uniquely exhibit the complete physiological and behavioural manifestations of sleep. It has been suggested that the process of sleep has evolved in parallel with homeothermy. Phylogenetic assessment reveals that the complete physiological and behavioural manifestations of sleep appear to be an exclusive characteristic of homeotherms. Only they demonstrate sleep comprising both rapid-eye movement (REM) and an electrophysiological pattern of large slow waves (SWS) (Allison & Van Twyver, 1970) leading to a proposal by Berger (1975) of parallel evolution between sleep and homeothermy in response to the need for energy conservation (Walker & Berger, 1980). Within sleep architecture, it is the component of SWS that has specifically been shown to coincide with the development of homeothermy and appears to be associated with reduced MR (Brebbia & Altschuler, 1965; Haskell, Palca, Walker, Berger & Heller, 1981a; Shapiro, Goll, Cohen & Oswald, 1984). If this is the case, then it could be assumed SWS would be especially energy conserving and an important and highly efficient automatic regulatory conserving mechanism within the homeotherms (Parmeggiani, 1989).

There are a number of findings that provide support for the notion of sleep as an energy conserving period. First, the results from Zepelin and Rechtschaffen's (1974) correlational study of sleep parameters, body weight, MR, predicted life-span and other constitutional variables, showed a high positive correlation (0.65) between total sleep time (TST) and MR in 53 mammalian species. This essentially supports the view that species with high MR during wake

require a greater need for energy conservation that is effectively fulfilled by increasing food intake, or alternatively, spending more of their time in inactive periods of reduced metabolism such as those associated with sleep, torpor or hibernation (Heller, Walker, Florant, Glotzbach & Berger, 1978). These reduce the time available for activity (Zepelin & Rechtschaffen, 1974), and thus provides a period during which energy can be conserved.

Second, Walker and Berger (1980) report that individuals with high MRs show longer sleep durations compared to those with low energy expenditure, regardless of the prolonged periods of relaxed wakefulness experienced by humans (Horne, 1977). It also has been reported that body weight shows a high negative correlation with MR (Allison & Cicchetti, 1976) and both body and brain weight have strong negative correlations with SWS while MR is highly positively correlated with SWS (Allison & Cicchetti, 1976).

Finally, a general conclusion from the phylogenetic studies is that quiet sleep, torpor, and hibernation appear to be related dormant states lying on a continuum of reduced energy expenditure and lowered T_b, all serving the purpose of energy conservation (Berger, 1984). In the framework of the current thesis then, the energy conservation hypothesis would predict increased sleep duration and levels of SWS as a function of high rates of energy expenditure.

Ontogenetic Comparisons

An analysis indicates that SWS is related to activity levels, and hence to energy conservation. The evidence on sleep changes over the life-span supporting this notion is as follows.

The high proportion of sleep evidenced in infancy (Williams, Karacan & Hursch, 1974) and the increased levels of SWS following acute starvation in adults (Kreider, Buskirk & Bass, 1958; Karacan, Rosenbloom, Londono, Salis, Thornby & Williams, 1973; MacFadyen, Oswald & Lewis, 1973) strongly infers SWS to have

energy conserving properties that may allow stored energy to be utilised during growth. Furthermore, there is a general decline in brain and body metabolism with age (Feinberg & Carlson, 1968) when relatively little SWS or sleep-related growth hormone (GH) secretion occurs in elderly humans (Mendelson, 1989). Therefore, since it has been reported that the very high SWS levels during infancy and childhood appears to decrease with age, especially stage 4 sleep, and TST (Agnew, Webb & Williams, 1967b; Feinberg, 1974; Feinberg, Koresko & Heller, 1967; Kahn & Fisher, 1969; Kales, Wilson, Kales, Jacobson, Paulson, Kollar & Walter, 1967; Webb & Agnew, 1971; Williams et al., 1974) it is suggested that such evidence further lends support for the notion of SWS as having an energy conserving function.

Other Evidence

There are three other sources of evidence from humans that provides further indirect evidence for the energy conserving properties of sleep, especially SWS. They are that body temperature decreases during the first one to two hours of sleep (Day, 1941; Geschichter, Andrews & Bullard, 1966) an effect that is largely independent of the circadian phase of sleep onset (Gillberg & Akerstedt, 1982); that depth of sleep is inversely related to the rate of oxygen (O_2) consumption (Brebbia & Altschuler, 1965) and that there is an associated drop in MR (Brebbia & Altschuler, 1965; Fraser, Trinder, Colrain & Montgomery, 1989; Webb & Hiestand, 1975) or O_2 consumption (Kreider et al., 1958); and that heart rate (HR) (Kleitman, 1963; Mason & Benedict, 1934) decreases across the night.

Summary

Evidence derived from the analysis of the phylogenetic correlations between sleep and decreased metabolism; between sleep and MR within species; and correlations collected from the available human studies, appear to strongly support the notion of individuals with high MR undergoing longer sleep durations compared to those

with low rates (Walker & Berger, 1980). The finding that longer nocturnal sleepers have associated higher daytime T_bs, and thus possible increased daytime MR compared to controls (Taub & Berger, 1976) is consistent with the view of increased daily MR requiring a longer sleep period for compensation and general restoration. Even one hour of extra wakefulness during the night has been found to be followed by extra SWS and plasma human growth hormone (hGH) levels in the later part of the night (Beck, Brezinova, Hunter & Oswald, 1975). These findings demonstrate that an important association exists between metabolism and SWS.

The energy conservation hypothesis predicts a positive relationship between MR and SWS. Berger (1984) claimed that SWS has evolved as a state of reduced metabolism which has further been thought to be a period during which the increased energy requirements of homeothermy are partially offset (Walker & Berger, 1980). Hence, this theoretical model postulates that the primary function of sleep is to conserve energy and that an elevation in the energy conserving processes of TST and SWS is a direct response to increased levels of activity.

THE RESTORATIVE THEORIES

There are contrasting viewpoints among restorative theorists, but all have assigned a restorative function to SWS (Adam, 1980; Feinberg, 1974; Hartmann 1973; Horne, 1977, 1979, 1980, 1981, 1983, 1988, 1989; Oswald, 1969, 1970, 1974, 1980). However, each researcher has emphasised different aspects of the theory which has led to the development of two main forms of restorative theory. These include the more general or bodily restorative theories (Adam & Oswald, 1977, 1983; Hartmann, 1973; Oswald, 1969, 1974, 1980), and the cerebral or CNS restorative theories (Feinberg, 1974; Horne, 1977, 1979, 1980, 1981, 1983, 1988, 1989). Essentially, the bodily restorative theories propose a general function of sleep that is restorative to both body and brain. In contrast, the cerebral or CNS restorative theories propose that sleep is primarily important for the

restoration of brain function. These variations in emphasis are outlined.

Bodily Restorative Theories

The main proponents of this theory include Hartmann (1973), Adam (1980), Adam and Oswald (1977, 1983) and Oswald (1969, 1974, 1980), with the later authors providing the greatest empirical support for this view.

Hartmann's Theory of Restoration

Hartmann (1973) proposed that the process of sleep requires two qualitatively different sleep states in mammals. These are identified as non-rapid eye movement (NREM) and REM states that occur together in "cyclic alternation" and as such may ultimately be interrelated in their function. He extrapolated from the findings of studies reporting evidence for chemical changes during the sleep period to suggest that SWS in particular, may serve in the anabolic process of macromolecular synthesis (e.g., protein or RNA). As to the primary function of REM sleep, Hartmann (1973) hypothesised that it is perhaps most directed at utilising the synthetic products from SWS during the repair and restoration of synaptic connections. REM sleep also may serve in the formation of new connections in the catecholaminergic neuronal systems required for focused attention and learning. Furthermore, REM sleep has a CNS restorative role, actively involved in memory storage or consolidation and susceptible to prior levels of emotional or mental stress. Hartmann's view of the restorative properties of the two separate and yet interactive sleep phases, with macromolecular synthesis activated during the SWS period of the NREM state culminating in the use of these products for the renewal and building of synaptic connections later in sleep during the REM state, is shared by other authors such as Oswald (1969).

Oswald's Theory of Restoration

Oswald (1969) endorsed the theory proposed by Hartmann (1973), accepting the existence of two distinct types of sleep that function to promote synthesis and repair body tissues. However, he extended the theory by proposing that REM sleep is also important in the growth and renewal of the brain. Thus, Oswald's version of the sleep restorative theory highlighted SWS as dealing in widespread bodily restitution while REM sleep chiefly operated in the synthetic processes of brain reorganisation and repair. He maintained that the brain and bodily tissues experience different forms of synthesis during REM and SWS with the process of restitution or repair based upon protein synthesis. For example, it was assumed that there is continual repair and renewal of the existing cells of the brain by means of protein synthesis occurring within the REM phase, with no new cells being produced during this period. In contrast, he proposed that the continual restoration of bodily tissues requires synthesis for growth and repair by the formation of new cells during SWS. Oswald concluded that SWS is a critical sleep component that increases in response to high wake catabolism in order to allow body tissue restoration to proceed efficiently. Sleep provides an unresponsive state during which the whole body including the nervous system can regenerate (Adam & Oswald, 1977; Ramm & Smith, 1987)

Adam and Oswald's Theory of Restoration

Adam and Oswald (1977) and Adam (1980) also introduced the concept of "cellular energy charge" (EC) which they proposed as playing a fundamental role in metabolic coordination. This concept of an "energy charge" relating to the energy state of the cell, was based upon the notion that a cell's activity is controlled via its chemical energy and this is stored in the form of a high energy molecule adenosine triphosphate (ATP) with adenosine diphosphate (ADP) and adenosine monophosphate (AMP) representing metabolic products of the release of energy from ATP. The fine balance of EC maintained within a cell during the continual circular processes of

energy usage and depletion of ATP as well as the production of ATP which is then stored as energy, was defined in the formula by Atkinson (1968):

$$EC = \frac{(ATP + ADP/_2)}{ATP + ADP + AMP}$$

Adam and Oswald (1977) postulated that the cycles of inactivity and activity seen in the more "primitive" organisms, as well as those of sleep and wake associated with the higher animals, may in fact mirror the cycles in available cell energy. Since the great demand for increased energy levels needed to compensate for the high levels of activity during the wake state only can be fulfilled by processes involved in the degradation of available energy stores, there are minimal facilities available allowing restoration to occur. Therefore, since cellular, and thus total O_2 consumption, is determined by the energy state of the cell, and therefore by the ATP level in the cell, reduced activity associated with sleep reduces ATP depletion. O_2 consumption, and hence MR, would thus be expected to fall (Adam & Oswald, 1977) providing a period conducive to restoration. Sleep has been reported to be associated with high cellular ATP levels (van den Noort & Brine, 1970). It is argued then, that low levels of MR during sleep enable a net increase in protein synthesis due to both an increase in synthesis and a reduction in protein degradation during sleep as compared to the wake period (Adam, 1980).

During SWS, MR is at its lowest level creating suitable conditions for high EC which ultimately is conducive to rapid rates of protein synthesis, anabolic repair and mitosis. The bodily restorative theory so far elucidates the notion of sleep as providing a time of most restoration which compensates for the high catabolic processes associated with waking activity. Thus, although the process of restoration/degradation is continual, the ratio of restoration to degradation varies according to whether the individual is in either the sleep or wake state. It has been assumed that SWS provides a stage of most restoration unlike any other in the whole sleep process (Adam, 1980; Adam & Oswald, 1977; Oswald, 1980). In fact, it has been postulated that the rate of synthesis is accelerated during SWS

(Adam, 1980) which suggests that restoration processes are reliably maximised under conditions of increased SWS or sleep intensity. Consequently, TST and SWS represent key factors in the bodily restoration theory. The restorative model proposed by Adam and Oswald predicted that in times of marked energy depletion there is an associated reduction in MR. An increase in EC parallels this MR decrease, thus acting as a precursor to anabolic processes, SWS and sleep duration.

Evidence Consistent With Bodily Restorative Theories

Physical Exercise

It has been argued that physical exercise results in an increase in SWS and in sleep duration (Baekland & Lasky, 1966; Browman, 1980; Buguet, Roussel, Angus, Sabiston, & Radomski, 1980; Bunnell, Bevier & Horvath, 1983a; Griffin & Trinder, 1978; Horne, 1981; Horne & Moore, 1985; Horne & Staff, 1983; Ryback & Lewis, 1971; Shapiro, Griesel, Bartel & Jooste, 1975; Shapiro, Trinder, Paxton, Oswald, Warren, Catterall, Flenely, East & Harvey, 1981). If correct, this finding would be consistent with bodily restorative theories.

Weight Loss Studies

Other evidence derived from studies investigating the effects of acute starvation on sleep architecture, have shown significant increases in SWS levels (Crisp, Stonehill, Fenton & Fenwick, 1973; MacFadyen et al., 1973) as well as corresponding increases in hGH (Parker, Rossman, & Vanderlaan, 1972) following periods of fasting. Chronic starvation has been characterised by reduced SWS and REM sleep (Lacey, Crisp, Kalucy, Hartmann & Chen, 1975) that improves with weight gain as a possible measure of tissue regeneration and restoration representative of an anabolic state. A clear example of this effect is found in the case of individuals suffering from "anorexia nervosa". These patients have been found to show increased SWS levels during a re-feeding period (Lacey et al., 1975). Once these patients reach a stable "normal" weight, SWS levels diminish. The

results suggest a greater need of restoration during the weight gain period.

Weight loss during fenfluramine (an amphetamine derivative) administration also has been associated with elevated SWS levels (Lewis, Oswald & Dunleavy, 1971). Additional studies conducted by Rechtschaffen and Maron (1964) have observed SWS increases following amphetamine administration, a drug known for its ability to stimulate metabolic activity. Such administration also has led to increases in hGH (Besser, Butler, Landon & Rees, 1969; Dunleavy, Oswald & Strong, 1973) supporting the notion of SWS as possessing a restorative function.

Human Growth Studies

Another index of body restitution that is consistent with Adam and Oswald's (1977) hypothesis, is the process of cell mitosis for growth and tissue regeneration. It has been found that the rate of mitosis in skin (Fisher, 1968) and bone marrow (Mauer, 1965) is greater during the sleep period. Furthermore, the circadian peak in mitosis, for at least some animal and human tissue has been found to occur within the sleep period, especially during that stage of the night when SWS predominates. However, on further investigation, these peaks have been found to be not sleep dependent since they arise in the absence of sleep, and are therefore possibly unrelated to the sleep-hGH release. Nevertheless, Oswald (1974) maintained that after mitotic division, hGH should be available to assist in the growth of new cells.

Studies investigating growth patterns in children have generally found growth to be optimal during sleep as a result of increases in the secretion of the anabolic hGH promoting protein synthesis and the associated need for an accelerated mobilisation of free fatty acids (Hartmann, 1973).

A correlation between GH secretion and SWS has been reported, with the hormone's secretion being at a maximum during

stages 3 and 4 (Honda, Takahashi, Takahashi, Azumi, Irie, Sakuma, Tsushima & Shizume, 1969; Horne, 1979; Parker, Gotlin & Rossman, 1968; Parker, Sassin, Mace, Gotlin & Rossman, 1969; Takahashi, Kipnis & Daughaday, 1968). It also has been shown that exogenous administration of GH to normal volunteers results in a decrease in SWS. This finding has led to further speculations that GH secretion and SWS processes are linked by a feedback mechanism (Mendelson, Slater, Gold & Gillin, 1980). The discovery that anabolic hormones are secreted at a higher rate (i.e., over 70% of 24 hour GH release) during the first two hours of sleep provides further support for the notion of restorative processes and growth occurring primarily during sleep. This also is upheld by those findings showing the catabolic hormones, such as the catecholamines, to be predominantly secreted during wake periods. Investigation into the effects of specific SWS deprivation (Agnew, Webb & Williams, 1964) has led to reports of increased lethargy and physical discomfort in subjects following SWS deprivation (Agnew, Webb & Williams, 1967a). This finding further supports Hartmann's (1973) proposal that the function of SWS is primarily to prevent lethargy by restoring the body after physical fatigue.

The hGH-SWS association has therefore led to further suggestions that SWS may provide for high levels of macro-molecule synthesis and related anabolic processes (Horne, 1979; Karacan, Rosenbloom, Londono, Williams & Salis, 1974; Parker et al., 1969). In fact, a study by Alford, Baker and Burger (1973) showed that while relaxed wakefulness only induces small elevations of plasma hGH levels (approximately 17%) during sleep, these levels increase by approximately 250%, emphasising the importance of sleep for physical growth.

Thyroid Abnormality Studies

Research with hyperthyroid patients has shown them to exhibit excesses of SWS as well as raised nocturnal plasma GH levels that gradually return towards normal after treatment (Adamson,

Dunleavy, Hall, Lewis, MacFadyen, Ogunremi, Oswald & Strong, 1973; Dunleavy, Oswald, Brown & Strong, 1974). Conversely, hypothyroid patients have significantly decreased SWS levels which can be rectified using appropriate thyroid treatment for this condition (Kales, Heuser, Jacobson, Kales, Hanley, Zweizig & Paulson, 1967). Since the administration of thyroid hormones increases the rate of energy expenditure, and correlates with increased SWS levels (Ruiz-Primo, Jurado, Solis, Maisterrena, Fernandez-Guardiola & Valverde, 1982), this provides additional evidence supporting the model of bodily restoration assigned to SWS function.

Summary

The theory developed by Adam and Oswald assigning the primary function of bodily restitution to SWS, has been well substantiated by the findings of a relatively wide spectrum of research literature. In part, the evidence shows elevations of SWS and TST under conditions of increased wake-period metabolism. The bodily restorative theories thus predict that SWS and sleep duration will increase in order to compensate for the effects of heightened daily energy expenditure. The theories also require that metabolic activity be decreased during sleep so that restorative activity can proceed at an increased rate. There have been no studies however, that report a direct link between MR and sleep architecture.

Central Nervous System Restorative Theories

There are a number of alternative models contending that the restorative role of sleep is in the CNS. Three of these models will be outlined in this section; those by Feinberg (1974), Borbely (1982), and Horne (1977, 1979, 1980, 1981, 1983, 1988, 1989).

Feinberg's Model of Restoration

Feinberg (1974) viewed the process of sleep as imperative in counteracting the effects of waking activity or metabolism in the CNS. This idea was based upon the cyclical nature of sleep, where SWS precedes REM sleep with each emerging as discrete episodes. That is, EEG activity reverses the effect of wakefulness on the brain in some way. Other factors considered to make an important contribution to his model relate to the diminishing SWS levels, especially stage 4, with an associated earlier displacement of the first REM sleep, as chronological age increases. In particular, he viewed the decline in SWS over a night's sleep as reflective of the decay of some factor that has steadily accumulated during wakefulness. Feinberg also suggested that the onset of REM is functionally significant in precipitating SWS and offered a model based on the existence of two neuronal-metabolic states which elucidated the dynamics of the relationship between REM and SWS. According to this model, State 1 (S1) is achieved at the end of a sleep cycle, while State (S2) is a state of degradation resulting from a period of waking. Restoration of the CNS from S2 to S1 in response to the metabolic and neuronal consequences of waking brain activity, commences with the early NREM period. However, to ensure the conversion to be totally complete, there needs to be an intervention process promoting this transition. Feinberg assigned this function to REM, which was assumed to produce a specific substance inducing further SWS production and hence increasing S1 levels. Therefore, under conditions of increased S2 there is evidence of more intense NREM. During the early NREM cycles, stage 4 predominates as S2 is converted to S1. After a number of cycles, optimal levels of S1 are achieved and this is shown by the large proportion of stage 2 occupying NREM and decreased SWS period duration in these later cycles. At this stage, restoration has been achieved as a result of the complete transformation of S2 to S1.

In conclusion, probably the strongest points to emerge from Feinberg's (1974) CNS theory of restoration lies in his

acknowledgement of the cyclical nature of sleep and the account of the continual changes that occur within the sleep cycle. However, due to the limited descriptive characteristics of sleep upon which Feinberg's model has been based, it fails to address possible mechanisms specifically involved within the sleep cycle as well as defining which specific physiological processes are restored. As a consequence, this restorative theory of sleep function remains speculative.

Borbely's Theory of Restoration

In the development of a two-process mathematical model of sleep regulation and restoration, it was postulated that these processes interact in a specific manner affecting such factors as the length and timing of sleep (Borbely, 1982; Daan, Beersma & Borbely, 1984). In this attempt to describe the SWS changes over the night, Borbely unlike Feinberg has quantified delta wave activity in the EEG using spectral analysis based on fast Fourier transforms (Borbely, Baumann, Brandeis, Strauch & Lehmann, 1981). One process termed "S", can be regarded as an hourglass process maintaining sleep homeostasis (Daan, 1987). The variable S was described as a mechanism or substance similar in nature to that of Feinberg's, that increases during wakefulness and decreases exponentially during sleep independent of the circadian phase at which wakefulness or sleep occurs (Dijk & Beersma, 1989; Dijk, Beersma & Daan, 1987; Dijk, Brunner & Borbely, 1991). Within the framework of the current thesis, it could be assumed that this substance "S" may be produced in greater quantities if waking metabolism is elevated, and reflects the decrease in SWS levels over the night. Borbely's model was more advanced than Feinberg's model, since it acknowledged a broad range of influential factors other than just the length of prior wakefulness and the shape of decay of SWS across the night. It also incorporated a powerful circadian influence on sleep length (Broughton, 1988; Dijk, Brunner & Borbely, 1990; Gagnon, De Koninck & Broughton, 1985; Webb, 1986), excluding SWS, which Borbely defined as process "C". This process was believed to be

strongly associated with circadian T_b changes, and was used to describe the circadian influence on sleep length.

Horne's Theory of Restoration

Horne (1977, 1979, 1980, 1981, 1983, 1988, 1989) argued that the primary function of sleep is to restore brain function. He rejected body restitution on the grounds that there was no conclusive evidence in support of the theory. The bases for Horne's proposition can be outlined.

1. Horne stated that psychological, cognitive and behavioural deficits develop after sleep deprivation rather than physiological deficits. For example, behaviours such as irritability, speech slurring, minor visual misperceptions and disorientation suggest a facilitation of CNS rather than bodily restitution (Horne, 1978).

2. He pointed out that the EEG measures brain activity rather than changes in body tissue, such as those associated with tissue restitution (Horne, 1978, 1981), and concluded the brain but not the body is "off line" and being repaired during human SWS sleep. He acknowledged circumstantial evidence that indicates the recovery of the cerebrum during human SWS. This includes (a) the unique ability of the cerebrum to enter into a condition of isolation from both sensory input and from subcortical structures (Velasco, Velasco, Cepeda & Munoz, 1982); (b) the finding of minimal cerebral neuronal firing rates during stage 4 sleep; and (c) a positive correlation exists between human SWS (especially stage 4) and length of prior wakefulness (Webb & Agnew, 1971), and this may reflect a form of recovery.

3. He also argued with respect to exercise, that the human SWS effect depends on a brain temperature increase with elevated brain metabolism, and this increases the need for brain restoration.

4. He disputed the empirical association between GH release and sleep arguing that it is equivocal phylogenetically, and hence does not offer substantial support for the body restoration hypothesis of sleep (Horne, 1983). In humans, he suggested that although GH and SWS are linked, many functions have been assigned to growth hormones such as promotion of protein synthesis and fat mobilisation, yet there is still no clear function of GH release during sleep. The sleep-related hGH output may be a preventative measure for sparing tissue protein during the extended fasting state of sleep. This is supported by the dietary behaviour of carnivores in which their high consumption of protein appears not to be associated with sleep-related GH release.

5. The primary stimulus to body restitution is food intake and amino acid absorption from the gut. The few recent studies measuring protein turnover in humans over a 24-hour period (Clugston & Garlick, 1982; Garlick, Clugston, Swick & Waterloo, 1980) have found food intake to be particularly important in facilitating protein synthesis. Horne thus considered the fasting state generally associated with sleep to prohibit general body restitution. He based this claim on the finding that protein breakdown or tissue dissolution tends to exceed the overall protein content of body tissue during sleep. This evidence strongly opposes the notion of sleep as serving a body restitutive role.

6. Horne (1983) contested that the low MR associated with sleep (Brebbia & Altschuler, 1965; Fraser et al., 1989; Kreider et al., 1958; Shapiro et al., 1984; Stothers & Warner, 1977a,b, 1978; Webb & Hiestand, 1975) is incompatible with the high energy costs of any substantial rise in protein synthesis. This low MR in association with high cellular EC strongly suggests decreased synthesis and thus restoration. Consequently, if high levels of protein synthesis were to exist then an associated high MR level would be expected during sleep. Horne (1983) thus concluded that the arguments based on increases in cell EC during sleep are misleading and lack the necessary evidence.

7. Finally, since physical rest facilitates body restitutive processes, Horne noted that humans usually obtain sufficient rest periods for such purposes during wakefulness, and thus the period of sleep would not be purely defined as a solitary period of body restoration.

In conclusion, Horne (1983) considered that there is very little evidence available to support the view of sleep as a period when tissue and general body restitution is heightened. He viewed the evidence favouring the body restitutive theory such as increases in rates of cell division during sleep, the sleep-related hGH releases, and the associated elevations in human SWS following daytime exercise, as being incorrectly interpreted by many researchers who use these findings to support the general bodily restorative model. He claimed that these inaccuracies have developed from using the theory as a foundation for interpretation of the findings rather than adopting a broader perspective allowing the theory to evolve from the facts available. In Horne's view, any evidence of sleep as a restorative process only applies to brain functioning. In this respect, Horne proposed that the sleep period in humans primarily serves an overall restorative role in brain functioning while most of body restitution occurs during wakefulness.

The question of whether sleep offers the only method of prolonged periods of inactivity is also central to Horne's argument. Since regulated physical activity suppresses and thus delays tissue restitution, it is important that some intervening rest period occurs at a later time in order to counteract these effects. If sleep provides the only source of rest, then it seems reasonable to assume that restitution will predominantly occur during this period. This will result, not because sleep itself stimulates this restitution, but simply because sleep is the only provider for rest.

Horne (1983, 1988, 1989) has used the notion of obligatory and facultative sleep proposed by Parmeggiani (1980) to develop a model of sleep postulating two main sleep processes that operate in parallel to fulfil brain restoration and energy conservation. The first is

thought to specifically serve a brain restitutive role, with human SWS, particularly the "deeper" stage 4 sleep, and some evidence of REM sleep, occupying the early sleep period. Horne (1983) defined this initial sleep process as "Core Sleep" that repairs the effects of waking wear and tear on the cerebrum. The second form of sleep process maintains sleep beyond the point where core sleep declines. Horne termed this process "Optional Sleep" and claimed that it occupies the hours when activity is less advantageous and more difficult to undertake. He considered that this process is governed by circadian influences and the behavioural drive to sleep. It also may serve a more facultative sleep drive that promotes energy conservation and safety (Horne, 1983). In summary, the two processes of core and optional sleep are both activated at sleep onset with core sleep subsiding after a few hours to allow optional sleep to continue. These categories of core and optional sleep are effectively subdivisions of Borbely's process S, while process C is the circadian temperature rise that normally determines the ending of optional sleep.

Horne (1977) has contended that if human sleep is effectively active in brain restitution, then SWS may be of central importance in brain restitution because of the phylogenetic correlation between the development of SWS and cerebral development. Furthermore, REM sleep is considered important in the overall cortical restitutive process, however its role does not extend beyond that of the SWS level (Horne, 1979, 1989). Horne (1983) noted that human SWS reflects a period of recovery when cerebral functioning is at a minimum. Moreover, he proposed that the SWS period may perform an energy conservation function by promoting a state of deep torpor within the brain (Horne, 1985). An obligatory function is therefore assigned to SWS based on a number of sources of available evidence. For example, Hartmann (1973) and Webb (1989) have described the immutable nature of SWS, where it appears that any attempt to alter SWS levels is difficult. Any success in initiating SWS changes has involved experimental conditions using exercise, sleep deprivation, body heating and extended attention (Horne, 1985). In addition, it is extremely difficult to selectively deprive individuals of SWS. For

example, human SWS levels appear to be retained under conditions of partial sleep deprivation, and shows rebound effects following total sleep loss. The finding that natural short sleepers maintain the same levels of human SWS as age-matched controls while showing a reduction in other sleep stages (Horne, 1988) also has been considered to reflect a preservation of their core sleep.

In addition, Horne (1981) addressed the possible effects of thermal loading on SWS based upon the findings of increased levels of SWS following daytime exercise and passive heating (Horne & Moore, 1985; Horne & Porter, 1976; Horne & Reid, 1985; Horne & Staff, 1983). From these studies, he speculated that it is an increase in cerebral temperature induced by such thermally loaded states as exercise and passive heating, which plays the key role in generating SWS increases. He equated raised cerebral temperature with increased "brain work" or cerebral MR which is believed to somehow initiate SWS increases in response to more cerebral "wear and tear", or to an accelerated accumulation of brain substances especially associated with SWS promotion. Furthermore, these processes may directly influence the control mechanisms associated with human SWS and are believed to operate concurrently, with sleep substances acting to modulate SWS without affecting any underlying function. A study by Horne and Minard (1985) demonstrated that SWS elevations occurred following exposure to prolonged increases in daytime brain work via raised sensory stimulation. This further supports the idea of increased cerebral MR being associated with SWS mechanisms.

In conclusion, Horne's view of the restorative theory of sleep at the level of the brain provides a model for the proposed roles of SWS and REM sleep. In his model, SWS has been assigned an obligatory role while REM sleep adopts a more facultative purpose during cortical restoration. The overall aim of the sleep process is then to provide a safe compensatory period during which energy conservation can be achieved to counteract the effects of prior heightened wake metabolism. Any attempt to increase metabolic processes through such physical activities as intense exercise, appears to influence the

generally fixed patterns of SWS by increasing the levels of this deep stage of sleep.

Conclusions

In support of the energy conservation hypothesis the evidence derived from phylogenetic and ontogenetic comparisons of sleep data points to an association between metabolism and sleep. Similarly, the evidence for the theories of both body and cerebral restoration propose a relationship between sleep and waking metabolism. Both theories claim that sleep fulfils a compensatory function, and thus predict that the relative amount of time devoted to sleep and to each sleep stage will change in response to an increase in wake-period energy expenditure or catabolic activity. In addition, both theories anticipate that metabolic activity will be lower during sleep, either because of the mediating effects of the change in sleep architecture, or because of independent falls in metabolic activity during sleep. However, the exact nature of the relationship between wake-period MR and sleep MR remains equivocal. For example, whether wake MR undergoes some form of compensation during the sleep period is still questionable. Direct analysis of the relationship between metabolism and EEG patterns, especially SWS, therefore presents an essential issue in need of future research.

CHAPTER 3

THE EFFECT OF EXERCISE AND PHYSICAL FITNESS ON SLEEP ARCHITECTURE AND PHYSIOLOGICAL PROCESSES

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Central to the current thesis is the hypothesis that sleep differences between aerobically fit and unfit individuals (Griffin & Trinder, 1978; Montgomery, Trinder & Paxton, 1982) are due to variations in the underlying metabolic status of these subject populations. This chapter will consider the empirical support for a facilitative effect of exercise and physical fitness on SWS and sleep duration.

The Effect of Exercise on Sleep

Since the development of the sleep restorative theories, there have been numerous studies investigating the outcome of increased catabolic activity on subsequent sleep architecture (see Tables 3.1, 3.2, 3.4). Oswald (1962) and Roffwarg, Dement and Fisher (1964) were among the first to predict that increased amounts of exercise raises SWS levels and increases total sleep duration. As can be seen from Tables 3.1, 3.2 and 3.4 the studies that have investigated the relationship between exercise and sleep obtained diverse results (Adamson, Hunter, Ogunremi, Oswald & Percy-Robb, 1974; Baekland, 1970; Bonnet, 1980; Browman & Tepas, 1976; Desjardins, Healey & Broughton, 1974; Hauri, 1968; Horne & Porter, 1975; Kupfer, Sewitch, Epstein, Bulik, McGowen & Robertson, 1985; Montgomery et al., 1982; Montgomery, Trinder, Paxton & Fraser, 1987; Montgomery, Trinder, Paxton, Fraser, Meaney & Koerbin, 1985; Montgomery, Trinder, Paxton, Harriss, Fraser & Colrain, 1988; Moses, Lubin, Naitoh & Johnson, 1977; Paxton, Montgomery, Trinder, Newman & Bowling, 1982; Paxton, Trinder & Montgomery, 1983; Torsvall, Akerstedt & Lindbeck, 1984; Trinder, Bruck, Paxton, Montgomery & Bowling, 1982; Walker, Floyd, Fein, Cavness, Lualhati & Feinberg, 1978; Zir, Smith & Parker, 1971). Such diversity

in results can most likely be accounted for by the methodological differences between the studies (Trinder, Montgomery & Paxton, 1988).

Only a few studies on exercise and sleep have found clear-cut evidence for a positive relationship between these two variables with increases in SWS following exercise (Baekland & Lasky, 1966; Horne & Moore, 1985; Horne & Staff, 1983; Shapiro et al., 1975; Shapiro, Bortz, Mitchell. Bartel & Jooste, 1981; Shapiro & Verschoor, 1979). Other studies have addressed this issue but failed to find such a clear-cut positive relationship between exercise and SWS (Bunnell et al., 1983; Hobson, 1968; Kupfer et al., 1985; Matsumoto, Nishisho, Suto, Sadahiro, & Miyoshi, 1968; Matsumoto, Saito, Abe & Furumi, 1984; Zloty, Burdick & Adamson, 1973). However, variables such as individual attributes of age, gender and level of fitness, as well as the nature of the exercise performed, especially in terms of its intensity, duration and temporal placement within a normal daily cycle (Hauri, 1968; Horne & Porter, 1975) have resulted in confounding factors in these studies that limit the value of the findings.

In regards to the type of exercise, rate of energy expenditure of exercise is considered to be more important in influencing sleep than total energy expenditure (Horne, 1981). For example, studies demanding high rates of energy expenditure have been successful in finding associated increases in SWS after exercise (Bunnell et al., 1983b; Shapiro et al., 1975; Shapiro et al., 1981). Those employing low rates of energy expenditure however, regardless of using high total energy expenditure, failed to show this result (e.g., Buguet et al., 1980). Horne (1981) suggests that the critical factor in producing SWS changes following exercise is the associated rise in T_b, and in order for this to be achieved the level of exercise needs to be intense. The notion that T_b constitutes an important factor in initiating the SWS effect is supported by studies investigating the influence of passive heating on body and sleep processes. These studies have demonstrated a SWS elevation (Horne & Staff, 1983; Horne & Reid, 1985) comparable to that associated with a combination of exercise

and body heating (Horne & Moore, 1985). It is still unclear however, what produces the SWS effect. One relevant variable is fitness. The role of fitness in producing increased SWS is considered in the next section.

Fitness and Sleep

Fitness can influence sleep via the capacity of fit subjects to undertake more exercise than unfit subjects (e.g., Driver, Rogers & Shapiro, 1987; Griffin & Trinder, 1978), or as a direct result of the fitness and/or as a function of changes in physiological or metabolic processes following prolonged training. These issues will be discussed in the following sections. A brief overview of the studies investigating the impact of fitness on sleep quality will be presented. The fitness effect will then be more closely examined. Finally, physiological or metabolic processes that are most influenced by prolonged physical training will be reviewed.

Exercise and Sleep in Fit Subjects

There is a general contention that only individuals who are "fit" and endurance athletes show increased SWS and sleep duration after exercise (e.g., Horne & Moore, 1985; Horne & Staff, 1983; Shapiro et al., 1981). More sedentary individuals who are unaccustomed to exercise, do not demonstrate such a relationship (Adamson et al., 1974). Griffin and Trinder (1978) were the first authors to report a SWS effect due to fitness confirming the speculation of Baekland and Lasky (1966). The results of Griffin and Trinder's (1978) study showed that fit subjects increased stage 3 sleep and a decrease in SWS latency after an afternoon exercise session. Other studies demonstrating this outcome include those using both men and women (Bunnell et al., 1983a); and those investigating women only (Walsh, Puig-Antich, Goetz, Gladis, Novacenko & Glassman, 1984) and after an aerobic training program (Driver, Meintjes, Rogers & Shapiro, 1988). Another study found similar results for TST (Montgomery et al., 1982). In fact, studies using fit subjects have been far more

consistent in showing an exercise effect (see Table 3.2). There are however, other studies that fail to find a significant relationship (e.g., Baekland, 1970; Bonnet, 1980; Kupfer et al., 1985; Montgomery et al., 1985; Montgomery et al., 1988; Walker et al., 1978). These inconsistencies may be attributed to body composition differences, and hence contrasting metabolic factors. Nevertheless, discrepant findings have led to increased research specificity in order to yield more consistent results by designating both the type of subject and exercise used. For example, while endurance athletes show sleep effects, other athlete groups such as power trainers with similar levels of daytime activity do not show the same changes in sleep (Trinder, Paxton, Montgomery & Fraser, 1985).

Equivocal results obtained with physically fit subjects, have led to further investigation of body composition in these individuals. It has been predicted that any correlation existing between body composition and various components of sleep, such as SWS, may be interpreted as evidence that supports a possible association between MR and sleep architecture (Trinder, Stevenson, Paxton & Montgomery, 1982).

Some of the evidence has shown lean body mass (LBM) to be positively correlated with MR (Ravussin, Lillioja, Anderson, Christin & Bogardus, 1986), NREM sleep (Shapiro, Warren, Trinder, Paxton, Oswald, Fleneley & Catterall, 1984), REM (Shapiro, Catterall, Warren, Oswald, Trinder, Paxton & East, 1987) and TST in trained athletes (Driver et al., 1988). These support the suggestion of a relationship between metabolic activity and sleep patterns. The positive TST-LBM relationship found in Driver et als. (1988) study may be partially attributed to genetic predetermination since two factors affecting maximal O_2 consumption are the individuals' heredity and body composition (McArdle, Katch & Katch, 1986).

Table 3.1

Studies using Untrained Subjects in Single Group Designs

AUTHORS	SUBJECTS	EXERCISE	SLEEP EFFECTS
Adamson et al. (1974)	12M 5 fit 7 unfit * 19-31yrs	Athletic activities	No sleep EEG Effects
Browman & Tepas (1976)	9M 19yrs	Light dynamic 20km on bicycle erg.	SOL inc. REML inc.
Browman (1980)	4M 3F 19-22yrs	Static at 40% VO_2max 2x40 min sessions	SOL dec. 1st cycle SWS inc.
Bunnell et al. (1983a)	4M 5F	dynamic to volitional exhaustion	SWS inc.
Desjardins et al. (1974)	6M 23-28yrs	high intensity and low intensity Ex.	%REM dec. st2 inc both conditions No SWS effects
Driver et al. (1988)	9F unfit 18-28yrs	1hr 70% VO_2max cycling- 12 wk aerobic training eg. graded cycling 3 times/wk, interval training, + a road run/wk	Before training- st4 latency inc., SWS + REM dec. After training- REML inc, st4 latency dec., SWS dec. smaller
Hauri (1968)	15M	6hrs strenuous	No effects
Horne & Porter (1975)	8M 18-22yrs	45% VO_2max for 85 min on bicycle erg.	st3 inc in 1st cycle

Table 3.1 cont'd

AUTHORS	SUBJECTS	EXERCISE	SLEEP EFFECTS
Matsumoto et al. (1984)	6M 20-24yrs	prolonged Ex. at low rate EE	SWS inc. SOL dec.
Montgomery et al. (1982)	14M 10F	4 groups: graded Ex. intensity- very light (50% VO_2max for 45 min) to exhausting (100% VO_2max for 60 min) on bicycle erg.	No sleep effects
Paxton et al. (1982) Exp 1:	14M 10F 4 groups	light to medium intense varied across groups	No sleep effects
Zir et al. (1971)	10M 17-23yrs	light exercise in 5, medium exercise in 5	inc. in mean TST

* subjects treated as a single group.
Abbreviations: **dec** = decrease; **erg** = ergometer; **F** = female subjects; **hrs** = hours; **wk** = week; **Ex** = exercise; **inc** = increase; **M** = male subjects; **min** = minutes; **yrs** = years; **EE** = energy expenditure; **st** = stage. These abbreviations are used in other tables. Sleep abbreviations are found in text.

Lean body mass is considered proportional to the "active tissue mass" (Miller & Blyth, 1952; Sachchidananda & Robindranath, 1958) and has been found to have the highest correlation with MR when compared with seven other relevant anthropometric measures (Halliday, Hesp, Stalley, Warwick, Altman & Garrow, 1979). This supports a relationship between these parameters. Total body mass also has been shown to be related to both total sleep and REM duration (Adam, 1977). Shapiro et al. (1987) noted that the generally reliable methods used to assess body composition, as described by Boddy, King, Tothill and Strong (1971), nevertheless have limitations. For example, the relationship between body composition and sleep often appears to depend on the subject's level of fitness (Paxton, Trinder, Montgomery, Oswald, Adam & Shapiro, 1984). It can therefore be inferred that the sleep architecture of athletes is particularly affected by body composition factors such as the percentage of LBM.

Other studies that have investigated anthropometric variables have failed to show a consistent relationship. Although SWS and height have been found to be significantly negatively related (Nakazawa, Hasusawa, Ohkawa, Sakurada & Nonako, 1978), there is however no consistent relationship between SWS and weight (Adam, 1977; Crisp et al., 1973; Ho, Fekete-Mackintosh, Resnikoff & Grinker, 1978; Olgivie & Broughton, 1977; Lacey et al., 1975) or between sleep and weight/height ratio (Hunter, Feind & Strong, 1966; Othmer, Levine, Malarkey, Corvalon, Hayden-Otto, Fishman & Daughaday, 1974).

Studies with unfit subjects have generally shown negative facilitatory effects for exercise on sleep (see Table 3.1). Only a few studies have demonstrated results similar to those found with fit subjects. For example, Zir et al. (1971) showed increased TST; Horne and Porter (1975) showed increases in stage 3 of sleep in the first cycle; Browman (1980) reported increases in SWS in the first cycle of sleep and a decrease in sleep onset latency (SOL); and Bunnell et al. (1983a) showed increases in SWS following exercise. It is interesting

to note that exercise-induced sleep changes were relegated mostly to the first sleep cycle of the night (e.g., Bunnell et al., 1983a). Most SWS architectural changes in response to prior wake-period metabolic manipulations occur during this period (see Table 3.3) with particularly marked increases in SWS within this period (e.g., Browman, 1980; Bunnell et al., 1983a; Maloletnev, Telia & Tchatchanashvili, 1977). However, in a majority of the studies using unfit subjects, the failure to find exercise-sleep facilitatory effects may be a function of factors including the type of exercise or subject gender.

It is quite prevalent for sleep disturbance to be heightened following exercise (Baekland & Lasky, 1966). This disturbance is usually manifested by increased SOL and delayed REM latency (Browman & Tepas, 1976); decreased levels of REM (Bonnet, 1980; Desjardins et al., 1974; Shapiro et al., 1975; Shapiro et al., 1981); and increased time awake (Shapiro et al., 1981). Such sleep disturbances, especially in unfit individuals, may be a product of general bodily discomfort emanating from physiological arousal and extensive muscle and body soreness. The dramatic MR changes and body dehydration occurring after exercise are additional factors that possibly contribute to these sleep disturbances. Fit individuals however, by the nature of their fitness levels, can generally endure higher levels of bodily stress and recover rapidly from the side-effects of intense physical exercise. Therefore, they would most likely avoid sustained disruption into the sleep period. This may help explain the distinctive exercise-sleep relationship seen in this group compared to their unfit counterparts. The level of physical activity that they are able to sustain elevates general metabolic activity well beyond the exercise period (Simonson, 1971; Wilmore, 1977). Passmore and Johnson (1960) have shown sustained levels of O_2 consumption for at least seven hours following prolonged periods of moderate exercise. This increase in O_2 consumption known as the "O_2 debt" or the "excess post-exercise O_2 consumption" (EPOC) (Astrand & Rodahl, 1977; Brooks & Fahey, 1984; Hill, 1922) is used to counteract the aerobic energy deficit in O_2 consumption after exercise (Berger, 1982). Two

phases are involved in the O_2 recovery process: the fast early process of "alactacid O_2 debt" that repays approximately one-half of the total recovery O_2 consumption within 30 seconds after exercise; and the slow phase known as the "lactacid O_2 debt," that may take up to several hours, or even a day, before the pre-exercise O_2 consumption level is regained, and is generally dependent upon the intensity and duration of exercise and fitness level due to its responsiveness to the considerable increases in lactic acid and T_b induced by strenuous exercise (Davies, DiPrampero & Cerretelli, 1972; Hagberg, Mullin & Nagle, 1978).

It is hypothesised that fit subjects have elevated levels of MR into the sleep period which is related to increased SWS levels. The unfit group would more likely be expected to suffer from higher levels of bodily stresses conducive to greater sleep disturbance (Buguet et al., 1980), therefore masking any potential SWS increases and TST that may otherwise occur.

In summary, exercise is associated with increased levels of SWS in fit subjects but not unfit individuals. Discrepant results between studies may be attributed to the stress effects invoked by exercise, or possibly by other confounding factors interfering with the exercise effect.

The Fitness Effect and Sleep

Some of the studies that have investigated the effects of exercise on sleep have led to consideration of fitness effects and sleep. It is this factor that will be examined next.

Only a few studies have isolated the effects of physical fitness in facilitating SWS and TST. These studies directly contrasted the sleep architecture of trained and untrained individuals (see Table 3.4) where several have reported higher overall SWS levels in young aerobically fit individuals compared to their unfit counterparts (Baekland & Lasky, 1966; Griffin & Trinder, 1978; Paxton et al., 1984; Trinder et al., 1982; Trinder et al., 1985; Walker et al., 1978),

and in exercising and sedentary women (Driver et al., 1987). This finding is consistent with the view of sleep as less sensitive to acute changes in energy expenditure responding slowly to metabolic changes (Anch, Browman, Mitler & Walsh, 1988).

Even though significantly more TST has been found to occur in fit rather than in unfit subjects following exercise (Walker et al. 1978), failure to demonstrate elevated amounts of SWS in fit subjects (e.g., Paxton et al., 1984; Walker et al., 1978) may be related to the compounding effects of other factors. These include diet (Paxton et al., 1983), body composition (Leiker, Harsh, Thompson & Burks, 1989; Paxton, Trinder, Montgomery et al., 1984; Paxton et al., 1984) or type of physical training performed (Trinder et al., 1985). Furthermore, although the findings are not consistent (Montgomery et al., 1987; Trinder et al., 1982) age is still considered to be an important factor that modifies the effect of fitness on SWS or TST (Williams et al., 1974), where studies using young subjects have demonstrated a relationship (Montgomery et al., 1982).

In summary, physical fitness affects SWS and duration of sleep, although the exact nature of this relationship is uncertain. Whether metabolic factors are involved remains unknown and hence further research is needed in this area.

Physical Training, Fitness and its Relationship to Metabolic Properties Associated with Physiological Changes

The effects of physical training are apparent at several different levels associated with gross physiological changes, cellular changes, and temperature regulation. Some of the physiological changes produced by exercise can be observed during a state of rest. A more notable training-induced adaptation is a reduction in the exercise cardiac frequency as a result of increased cardiac stroke output due to a more complete draining of the heart during systole (Cotes, 1976).

Table 3.2

Studies of Trained Subjects

AUTHORS	SUBJECTS	EXERCISE	SLEEP EFFECTS
Baekland (1970)	14M	Usual	Tendency to SWS inc.
Baekland & Lasky (1966)	10M	Usual	SWS inc.
Bonnet (1980)	12M	6.5hr march	REM dec.st4 inc
Buguet et al. (1980)	6M	6 x 34 km/d	individual differences
Horne & Moore (1985)	6F	2x40 min run (13km/hr) on treadmill at 75% VO_2max in Hot and Cool conditions	Hot run Total NREM inc. SWS inc.(esp. st4) in 1st sleep cycle for 2nd + 3rd episodes of SWS Cool run No SWS or sleep effects
Horne & Staff (1983)	6M 2F	High Level Ex (6MJ Ex. load) with 2x40 min periods of 80% VO_2max treadmill run	SWS inc., st3 inc. for High intensity Ex.
		Low level run (6MJ Ex. load) with 2x80 min periods of 40% VO_2max treadmill run	No SWS effects TST inc., SOL dec.
Kupfer et al. (1985)	10M	Usual run + a run 2x the distance at subjects' usual rate	No SWS effects REML dec.
Maloletnev et al. (1977)	15M	intense	st4 inc.

Table 3.2 cont'd

AUTHORS	SUBJECTS	EXERCISE	SLEEP EFFECTS
Maloletnev & Telia (1976)	10M	usual	st4 inc. in 1st NREM cycle
Montgomery et al. (1982) <u>Exp. 5</u>	11M	<u>3 Ex. sessions</u> 1hr walk 1hr run 6hr walk	No Ex. effect
Montgomery et al. (1985)	8M 40yrs	intense 46km marathon	No SWS effects TST + REM dec. REML inc.
Montgomery et al. (1988)	<u>Exp 1:</u> 7F 7M	afternoon Ex.	No sleep effect SWS dec. in F's REM dec. in M's
	<u>Exp 2:</u> 12M older 10M young	usual	No sleep effect st4 dec. in young
	<u>Exp 3:</u> 10M power lifters	afternoon Ex.	No SWS effect
Shapiro et al. (1975)	2M	graded to intense	SWS inc. REM dec.
Shapiro & Verschoor (1979)	1M	88km marathon run	SWS inc. REM dec.
Shapiro et al. (1981)	6M	intense/long 92km marathon	TST inc SWS inc REM SOL WAKE dec.
Torsvall et al. (1984)	6M	30-43km cross country run	No SWS effect EEG total power density inc., REM dec., REML + st2 inc.

Table 3.2 cont'd

AUTHORS	SUBJECTS	EXERCISE	SLEEP EFFECTS
Trinder et al. (1982)	**young:** 6 fit 6 unfit **older:** 6 fit 6 unfit	fit only usual	No sleep effects
Zloty et al. (1973)	16M	usual	SWS inc.

Table 3.3

First Cycle Sleep Effects or Early Sleep Period Effects

AUTHORS	CYCLE OR HOURS	SLEEP EFFECTS
Browman (1980)	1st cycle	SWS inc MT dec.
Browman & Tepas (1976)	1st 2.5 hrs	SOL inc.
Bunnell et al. (1983a)	1st cycle	SWS inc REM dec
Desjardins et al. (1974)	1st 3 hrs	%REM dec.
Hauri (1968)	1st cycle	No effects
Horne & Porter (1975)	1st cycle	ST3+2 inc after morning Ex.
Maloletnev et al. (1977)	1st cycle	SWS inc.
Paxton et al. (1982)	1st cycle	No effects
Paxton et al. (1983)	1st cycle	No effects
Zir et al. (1971)	1st 3 hrs	No effects

Note. There are many papers which have not specifically commented on the first cycle effect. However, in view of the overall results obtained in those studies, it is presumed that sleep changes have occurred in this part of the sleep period.

Table 3.4

Studies directly comparing Trained and Untrained Subjects

AUTHORS	SUBJECTS	EXERCISE	SWS EFFECTS
Driver et al. (1987)	6F sedentary 3F exercising	1hr bicycle ergometer 70% VO_{2max}	inc. SWS for exercising and sedentary subjects. dec. SOL in 4 sedentary. dec. SOL in 1 exercising.
Griffin & Trinder (1978)	8 fit: 4M 4F 8 unfit:4M 4F	7.3km run	Fitness effect st3 Fitness by exercise effect. No exercise effects in unfit subjects
Montgomery et al.(1982)	4M fit 4F fit 4M unfit	4.5 mile run for fit and unfit	TST inc., SOL dec. in fit No sleep effect in either group SWS inc. in fit
Exp 1:	4M unfit		
Exp 3:	older: 6M fit 6M unfit younger: 6M fit 6M unfit	usual fitness activity at normal intensity and moderate fatigue	TST + SWS inc., SOL dec. in fit
Exp 4:	8M fit 9M unfit	normal Ex. activity in fit with moderate fatigue. 2 mile run at mild rate for unfits	TST inc., SOL dec. in fit

Table 3.4 cont'd

AUTHORS	SUBJECTS	EXERCISE	SWS EFFECTS
Montgomery et al.(1987)	10M fit (young) 12M fit (older) 10M unfit(young) 10M unfit(older)	intense 90min continuous training run for fit. No Ex. for unfit	No SWS or TST in fit No sleep effects
Paxton et al. (1983)	8M fit 9M unfit	**Fit:** exhausting but stressful **Unfit:** 4-6kms	No exercise effects
Walker et al. (1978)	10M fit 10M unfit	10.2km run 2.4km run	No exercise effect in unfit or fit

Other physiological changes associated with aerobic training include lowered resting HR (i.e., post-training bradycardia)(deGeus, van Doornen & Orlebeke, 1990; Frick, Konttinen & Sarajas, 1963; Holmgren, Mossfeldt, Sjostrand & Strom, 1960; Knehr, Dill & Neufeld, 1942; Roussel & Buguet, 1982; Scheuer & Tipton, 1977; Taylor, Wang, Rowell & Blomqvist, 1963) due to increased vagal tone, decreased sympathetic drive and plasma catecholamines (Frick, 1977); modification in the O_2-transporting system resulting from increased heart volume due to greater maximal O_2 consumption (Cureton, 1971; Gollnick & King, 1969); capacity for greater O_2 debt; lower pulmonary ventilation (Andrew, Guzman & Becklake, 1966); an increase in bone, tendon and muscle strength (Appenzeller & Atkinson, 1983a); body composition adjustments where there tends to be a generally lower proportion of fat tissue and a higher proportion of active tissue in trained compared to untrained individuals (Ismail, 1971) (i.e., increased LBM, muscle mass and

blood volume), although some studies have shown %LBM to be lower in athletes (Paxton et al., 1983); increased thyroxine turnover (Terjung & Winder, 1975); lower serum cholesterol in trained subjects than in the general population (Karvonen, 1959); changes within the hormonal system with particular effects in the balance between sympathetic and parasympathetic activities where insulin levels tend to be higher but glucagon lower after training (Hartley, Mason, Hogan, Jones, Kotchen, Mougey, Wherry, Pennington & Ricketts, 1972; Bloom, Johnson, Park, Rennie & Sulaiman, 1976); a lowered catecholamine response to exercise (Aakvaag & Opstad, 1985; Peronnet, Cleroux, Perrault, Cousineau, de Champlain & Nadeau, 1981); less displacement of physiological function by any given level of work load and; faster recovery to baseline values after exercise completion (deVries, 1980). Additionally, some evidence suggests significantly lower systolic and diastolic blood pressure (Montoye, Metzner, Kellner, Johnson & Epstein, 1972) along with improvement in the efficiency of fat use which acts as a carbohydrate sparing source in more active individuals. Subjects who perform minimal exercising activities fail to show such a result, which supports the notion that these effects are directly attributable to physical training (Cotes, 1976). It should be emphasised however, that the type of adaptive responses resulting from chronic exercise corresponds to the type of physical training performed. For example, the change in evaporative heat loss by virtue of an increased rate of sweating occurs in all trained individuals.

At a cellular level, physical training culminates in several basic changes that result in bodily systems and organ adaptations. Endurance training, in particular, enhances muscle protein synthesis which increases the myoglobin and mitochondrial content of muscle cells (deVries, 1980). These changes in muscle mitochondria correspond to increases in metabolic activity (Gollnick & King, 1969; Holloszy, 1967). This initial response is critical in order to permit the necessary changes for overall improvement in aerobic performance. Such cellular processes are unique to endurance trained individuals and allows carbohydrate sparing to take place in addition to the

utilisation of more fat as an energy source. Unfit individuals fail to exhibit such processes (Berger, 1982). It also has been noted that the production of muscle and blood lactate and utilization of muscle and liver glycogen decrease as a result of training (Holloszy, Rennie, Hickson, Conlee & Hagberg, 1977). Thyroxine production and degradation also have been shown to increase as a result of habitual exercise (Terjung & Winder, 1975). Changes within the hormonal system with particular effects in the balance between sympathetic and parasympathetic activities, where insulin levels tend to be higher but glucagon lower after training, also have been reported (Bloom et al., 1976; Hartley et al., 1972). Direct basal MR measures in terms of O_2 consumption (Hill, 1972; Webb & Troutman, 1970) after 12 hours of fasting (McGilvery, 1970) also have generally indicated higher basal MRs in fit subjects (Cureton, 1971) in response to the chronic effects of exercise, although earlier studies (e.g., deVries & Gray, 1963; Karpovich, 1941; Steinhaus, 1933) have failed to show such effects.

Temperature regulation and control is a critical factor affecting the capacity to perform exercise, especially exercise requiring a high level of endurance (Horne, 1981). There are a number of thermosensitive receptor zones located on the body surface, the peritoneal cavity, in the brainstem and in the spinal cord, that detect changes in T_b (Noble, 1986). The area within the brainstem that has been found to be thermally responsive is located in the preoptic area, or more specifically relates to the preoptic portion of the anterior hypothalamus. The hypothalamus serves as the integrating centre for inputs concerning T_b, and thus remains a critical component involved in the process of temperature regulation. In many ways, the hypothalamus can be considered to function in a manner closely resembling a thermostat; but unlike a house thermostat, it cannot "turn off" the heat and only initiates responses to protect the body from a build-up or loss of heat. Excessive heating of the body core beyond a threshold level, initiates a process involving signals being sent to cutaneous blood vessels (causing vasodilation) or to the sweat glands (causing sweat secretion), or both. The hypothalamus therefore regulates heat loss through

vasodilation of the skin and stimulation of the sweat glands (Brooks & Fahey, 1984; Noble, 1986). In the latter case, the message is delivered to the sweat glands by sympathetic motor nerves, and acetylcholine is released (Noble, 1986). Physical endurance training improves the tolerance to high core T_b that can reach levels in excess of 40°C during extended periods of high intensity exercise (Brooks & Fahey, 1984). Endurance training effectively decreases the temperature regulatory stress allowing more efficient dissipation of heat as well as decreasing the demands of the peripheral circulation (Appenzeller & Atkinson, 1983b). Therefore, the beneficial effects afforded to trained subjects are less heat storage and thus faster return of MR to steady state. This implies that trained subjects are more effective heat dissipators, and as such would most likely demonstrate shorter durations of elevated T_b following intense exercise, and therefore be more likely to return to baseline T_b levels by the time of retirement than their unfit counterparts. The baseline level for trained individuals however, may be at a higher threshold level than that for untrained subjects culminating from possible physiological changes following prolonged physical training. It is this aspect that holds particular interest.

Summary and Conclusions

The chronically elevated SWS levels and longer sleep durations in endurance athletes (Trinder et al., 1988) in conjunction with chronically higher basal levels of MR in fit individuals (Cureton, 1971; Trinder et al., 1982) is consistent with the theories of energy conservation and body restoration (Adam, 1980; Oswald, 1980; Berger, 1984; Berger & Phillips, 1988a). Sleep, in this context, is considered to respond in a compensatory manner to variations in energy expenditure and catabolic activity during wakefulness. However, this relationship is not conclusive since other athletes have failed to show sleep architectural changes in response to similar high levels of wake-period energy expenditure and catabolism (Trinder et

al., 1985). Nevertheless, with the evidence from endurance athletes, it may also be useful to specifically assess T_b in these individuals since it has been suggested that T_b affects sleep (Horne, 1981). To find elevated T_bs in the endurance athletes for example, who have often shown higher levels of SWS and longer sleep durations (Trinder et al., 1988), would be consistent with the theories of compensation, particularly if elevated T_bs are equated with increased catabolic activity. Such an effect would therefore support the sleep differences between trained and untrained individuals in terms of the theories. However, the fact that MRs are chronically higher in athletes compared to untrained individuals (Cureton, 1970) is not enough to suggest that T_b will also be higher. Indeed, it would be unwise to suggest that T_b is on average chronically higher throughout the 24 hour period, even without exercise, in the athletes on the basis of the MR evidence. For example, while both T_b and MR share a common association with the process of heat production, this is not the case with the process of heat dissipation. Only T_b represents a net indicator of both opposing processes which are difficult, if not impossible to unconfound. Therefore, to pre-suppose that T_b is a complete indicator of MR would be misleading. Nevertheless, since it has been reported that both MR and T_b share a linear relationship (Stolwijk, Satlin & Gagge, 1968), where an increase in T_b is associated with an increase in metabolic heat production, and vice versa under conditions of minimal heat loss (Keele, Neil & Joels, 1982), then it is reasonable to observe both factors together. A more comprehensive assessment of the level of overall difference in body metabolism between athletes and sedentary individuals would therefore be obtained if both MR and T_b were measured.

Consideration of the underlying processes that differentiate these two groups of individuals has turned attention to the role of MR and T_b in influencing sleep architecture. The effect of MR, T_b and the role of circadian cycles on sleep architecture will be assessed in the following chapter.

CHAPTER 4

BODY TEMPERATURE, METABOLIC RATE, CIRCADIAN CYCLES AND SLEEP

CHAPTER 4

BODY TEMPERATURE, METABOLIC RATE, CIRCADIAN CYCLES AND SLEEP

Sleep and thermoregulatory processes are considered to be closely associated (Day, 1941; Geschichter et al., 1966; Glotzbach & Heller, 1976; Haskell et al., 1981a; Heller & Glotzbach, 1977; Henane, Buguet, Roussel & Bittel, 1977; Parmeggiani, 1980, 1987, 1990; Sakagucchi, Glotzbach & Heller, 1979; Shapiro, Moore, Mitchell & Yodaiken, 1974; Smith, 1969). For example, the time of sleep onset and its duration are usually linked with the circadian phase of T_b (Czeisler, Zimmerman, Ronda, Moore-Ede & Knauer, 1980; Monk & Moline, 1989; Wexler & Moore-Ede, 1985), where T_b and O_2 consumption fall at sleep onset (Gillberg & Akerstedt, 1982; Kreider et al., 1958; Palca et al., 1986; White, Weil & Zwillich, 1985). This has been found to occur at any point in the circadian rhythm of T_b (Gillberg & Akerstedt, 1982; Mills, Minors & Waterhouse, 1974; Wexler & Moore-Ede, 1985).

This chapter will therefore discuss the inter-relations between temperature regulation, sleep and circadian rhythms. It also will highlight the metabolic changes that occur during the sleep period. In addition, the direct effects of temperature on sleep will be reviewed.

Changes in Body Temperature and Sleep

Investigation into the relationship between sleep and T_b has been the focus of research for many years (Bardswell & Chapman, 1911; Lindhard, 1911) where an interaction between thermoregulation and the sleep-waking activity has been repeatedly reported (Parmeggiani, 1977, 1990). There appears to be a strong sleep evoked decrease in T_b (Barrett, Morris & Lack, 1987; Lack, Balfour & Kalucy, 1985; Monk & Moline, 1989; Shapiro et al., 1984; Weitzman, Czeisler, Zimmerman & Ronda, 1980) especially during the first one to two hours of sleep. This effect is largely independent

of the influences of the circadian phase of sleep onset (Gillberg & Akerstedt, 1982) in thermoneutral or cool environments (Day, 1941; Geschichter et al., 1966; Haskell et al., 1981b; Kreider et al., 1958).

Numerous studies have found a direct relationship between T_b and sleep. These studies have ranged from investigations using more specific pharmacological manipulations such as injections of pyrogens to produce fever in humans (Karacan, Wolff, Williams, Hursch & Webb, 1968) and animals (Krueger, Walter, Dinarello, Wolff & Chedid, 1984; Krueger, Kubillus, Shoham & Davenne, 1986), or using dietary methods of increasing energy metabolism by administering various dosages of the hot spice capsaicin (Cameron-Smith, Hettiarachchi, Colquhoun & Clark, 1988; Henry & Emery, 1986; Kawada, Watanabe, Takaishi, Tanaka & Iwai, 1986), to other more physical manipulations of elevating T_b, such as exercise or passive heating, that avoid the complication of effects associated with using exogenous compounds. From these investigations evidence suggests that both ambient (Bonegio, Driver, King, Laburn & Shapiro, 1988; Czeisler, Weitzman, Moore-Ede, Zimmerman & Knauer, 1980; Muzet, Libert & Candas, 1984; Parmeggiani, 1977; Palca, Walker & Berger, 1986; Sewitch, Kittrell, Kupfer & Reynolds, 1986; Shapiro et al., 1974) and core T_bs have the potential to exert significant effects on sleep parameters. Some of the evidence available that highlights the possible link between T_b and sleep is outlined.

Pharmacological Effects

Some studies have shown that exposure to pathogens produce increases in SWS and T_b (Krueger, Majde, Blatteis, Ahokas & Cady, 1988; Toth & Krueger, 1988, 1990), which are most likely mediated by such sleep factors as muramyl peptides (Krueger, Pappenheimer & Karnovsky, 1982), interleukin-1 (Krueger et al., 1984), interferon (Krueger, Dinarello, Davenne, Walter & Kubillus, 1987), lipopolysaccarides (Krueger et al., 1986), lipid A (Krueger et al., 1986) and a tumour necrosis factor (Shibata, Blatteis, Krueger, Obal

& Opp, 1989; Shoham, Davenne, Cady, Dinarello & Krueger, 1987). Although these investigations appear to yield a simple cause-effect relationship where the increased T_b caused by sleep factors are responsible for their SWS-increasing capacity, there are several diverse lines of evidence indicating a dissociation between sleep factor-induced increases in T_b and SWS (Davenne & Krueger, 1987; Inoue, Honda, Komoda, Uchizono, Ueno & Hayaishi, 1984; Krueger, 1985; Krueger et al., 1982; Krueger et al., 1984; Matsamura, Goh, Ueno, Sakai & Hayaishi, 1988; Walter, Davenne, Dinarello & Krueger, 1986). Other studies using pharmacological manipulations to induce fever states have shown marked associated increases in waking and stage 1 with significant decreases in both NREM stage 4 and REM sleep (Karacan et al., 1968). These changes in sleep parameters were considered as direct responses to the changes in T_b since such sleep responses did not occur if T_b did not change from baseline levels.

Capsaicin Effects

Research related to the effects of a dietary substance known as "capsaicin" on thermoregulation and sleep also has shown evidence to suggest a link between these two processes (Benedeck, Obal, Jansco-Gabor & Obal, 1980). For example, capsaicin which is a substance derived from red pepper has been found both functionally and morphologically to impair central and peripheral warm receptors (Jansco-Gabor, Szolcsanyi & Jansco, 1970a,b; Nakayama, Suzuki, Ishikawa & Nishio, 1978; Szolcsanyi, Joo & Jansco-Gabor, 1971). This suggests a link between thermoregulation and sleep since the evidence has shown such capsaicin-induced impairment or desensitisation of thermoregulation against heat to be accompanied by a change in sleep-waking behaviour (Benedeck et al., 1980). Notably both REM and SWS increase in a manner consistent with the hypnogenic (Roberts & Robinson, 1969) and paradoxical sleep-prolonging (Parmeggiani, Zamboni, Cianci, Agnati & Ricci, 1974) effects of preoptic heating. Such evidence supports the hypothesis that activating effects in warm environments are mediated by warm

sensors sensitive to capsaicin. These authors hence described a competitive system of behavioural activation and deactivation effects in response to heat in rats. Assessment of the evidence also provides support for the hypothesis that basal forebrain hypnogenic mechanisms may be nonspecifically activated by heat (Benedeck et al., 1980; Parmeggiani et al., 1974). Benedeck et al. (1980) results therefore suggest that behavioural effects of warm environments can be mediated through various mechanisms.

Exercise and Passive Heating Effects

Other more recent studies have investigated the effects of exercise and passive heating on subsequent sleep architecture. The results from these studies show that induced elevations of T_b above resting levels prior to the sleep period leads to changes in sleep parameters primarily concerned with NREM sleep. Under appropriate conditions, the NREM facilitatory effect associated with passive heating is similar to exercise-induced NREM facilitatory effects (Horne & Staff, 1983). However, for these effects to arise exercise (Baekland & Lasky, 1966) or passive heating (Horne & Shackell, 1987) episodes need to occur at a time not too close to the sleep period. For example, it has been reported that passively heating individuals too near to the sleep period tends to disrupt sleep (Horne & Shackell, 1987).

The hypothesis that T_b is important in controlling sleep architecture followed the observation (Horne, 1981) that SWS increases after exercise tended to occur in warm climates (Shapiro et al., 1975; Shapiro & Verschoor, 1979; Shapiro et al., 1981). It also was suggested that the effect was most likely to occur if the physical exercise was intense. This supports the proposal that a suitably high level of body heating is necessary for SWS changes to occur. Horne (1981) emphasised that it is the "rate" of energy expenditure during the day, rather than merely the total amount, that is the important underlying factor influencing sleep architecture and thus SWS levels. It is believed that this rate needs to be sufficiently elevated in order to increase T_b (Horne, 1981), or more specifically cerebral

temperature (Horne, 1983; McGinty, Szymusiak & Nakao, 1990) to a level that will affect subsequent sleep architecture. This proposal has been supported by the findings from Bunnell et al. (1983b) who used high rates of energy expenditure, without affecting total energy expenditure for the day, to produce increased SWS levels. According to Horne's proposal, the elevated T_b mediates increased brain temperature which stimulates brain metabolism, resulting in a compensatory increase in SWS.

Of the passive heating studies, Horne and Staff (1983) for example, showed that a passively induced 2°C elevation in T_b above resting levels in trained subjects seated in a warm water tank as well as after high-intensity exercise, lead to localised increases in NREM sleep stages 3 and 4 without effects associated with REM sleep. Such findings indicate that core temperature influences sleep where exercise affects sleep as a function T_b changes. Body heating during the sleep period when REM sleep propensity is high, stage 4 propensity is low, and T_b is normally at its circadian nadir, also has shown similar effects on sleep architecture despite differences in the timing and duration of heating compared to those methods manipulating T_b prior to sleep onset (Bunnell & Horvath, 1985). Bunnell and Horvath (1985) for example, have found significant increases in NREM stage 2 and delta sleep in the fourth NREM cycle with no change in REM following heating during the night. Studies investigating the hypothermic effects of reduced T_bs on sleep associated with hibernation (Walker, Glotzbach, Berger & Heller, 1977) and shallow torpor (Walker, Garber, Berger & Heller, 1979) in ground squirrels have found significant increases in NREM and decreases in REM sleep compared with euthermic sleep distributions in these species.

Conclusions

The evidence suggests that there is an association between T_b and sleep where increases in T_b have been found to be associated with increased SWS levels. In order for SWS changes to occur however, it

may be crucial for T_b, or more specifically, cerebral temperature (Horne, 1983), to be sufficiently elevated (Horne, 1981).

Changes in Cerebral Temperature and Sleep

Recently, cerebral temperature has been recognised as playing a major role in the production of SWS (Horne, 1988, 1989; Horne & Moore, 1985; McGinty & Szymusiak, 1989; McGinty et al., 1990; Obal, 1984). The recent work of Berger et al. (1988) and Berger and Phillips (1988a,b) indicates that elevated T_b at, and shortly after sleep onset has a facilitative effect on SWS during the subsequent sleep period. Since their subjects had exposure to warming of the head then it is likely that cerebral temperature increases also have facilitative effects on SWS. It has been hypothesised that SWS is regulated by a subset of warm-sensitive neurons (WSNs) located in the hypothalamic preoptic area (POA) and basal forebrain; these sites are particularly active in thermoregulation and would be affected by cerebral temperature increases (McGinty & Sterman, 1968; McGinty & Szymusiak, 1989; McGinty et al., 1990; Han & Brobeck, 1961; Horne, 1983).

The fact that homeotherms (e.g., birds and mammals) share similar evolutionary SWS states, has led to the suggestion of a relationship between SWS and endothermic thermoregulation (McGinty et al., 1990). It is possible that homeotherms have evolved maximal waking brain temperatures that cannot be sustained indefinitely without some type of neurotoxicity (McGinty et al., 1990). Sleep may therefore be functionally beneficial, acting as a protective mechanism by regulating mean brain temperature against extreme high temperatures, and providing a compensatory period of relative slight cooling (McGinty et al., 1990). Slow-wave sleep is seen to evoke total suppression of behaviour and reduction in cerebral MR regulating heat loss and cooling that is independent of activity changes or circadian temperature cycles. That is, the functional requirement for SWS is thermoregulatory in nature, where SWS serves

to cool the brain or body or both (McGinty & Szymusiak, 1989; Treiber, Phillips & Berger, 1991). This hypothesis is consistent with energy conservation models of sleep functioning (Allison & Van Twyver, 1970; Berger, 1975; Snyder, 1966) and in part, with Horne's (1988, 1989) theory of cerebral restitution where SWS is proposed as restorative at the site of the cerebrum. McGinty and Szymusiak (1989) however, point out that the hypnogenic mechanisms based on a need for brain or body cooling represent very different physiological needs than energy conservation per se. Under a wide range of conditions however, both energy conservation and heat loss are correlated.

The energy conservation theory predicts that SWS is associated with regulated cooling which is similar in nature to shallow torpor and hibernation (McGinty et al., 1990). Raised brain temperature would most likely accelerate cerebral MR due to the well-documented increase in the chemical reactions within the cerebrum due to heating of this system. Increasing the already high waking metabolism of the brain by heating, may therefore accelerate the accumulation of the proposed sleep substance within the brain during wakefulness. As suggested previously, this effect may stimulate an increase in the length of wakefulness (Horne, 1988) leading to a greater build-up of this substance. An alternative view within the framework of the cerebral restitution model, is that since nerve activity has a chemical basis, increased cerebral temperature and cerebral MR also may speed-up this activity, and thus heighten wear and tear on nerve networks (Horne, 1988). Reduced cerebral MR during SWS would therefore be viewed as beneficial in counteracting these effects. However, the extent of these benefits is undefined. Nevertheless, the general implication is that SWS duration would increase with elevated waking cerebral metabolism.

Preoptic Stimulation Effects and Cerebral Heating

It appears that stimulation of the preoptic thermodetector area is especially effective in inducing sleep, while peripheral cold and

heat neurons exert an inhibitory control over REMs (Szymusiak, Satinoff, Schallert & Whishaw, 1980). Roberts and Robinson (1969) found that localised preoptic heating in freely moving cats elicited sleep. It also has been reported that in acute immobilised animals cortical synchronisation occurs in response to preoptic heating (Benedeck, Obal, Szekeres & Obal, 1976) and to elevation of the ambient temperature (T_a) (Okuma, Fujimori & Hayashi, 1965). This evidence has led to the development of several proposals about the areas and mechanisms involved in the facilitation of SWS under conditions of cerebral heating.

Slow-wave sleep, especially the stage 4 component, has been found to increase in humans along with slight decreases in REM following exposure to increased body heating while awake prior to bedtime (Horne & Reid, 1985). Immersion in cooler water showed no associated changes in T_b or subsequent sleep parameters. Horne and Moore (1985) also have shown increases in SWS following exercise under conditions in which body heating was allowed to occur; however, this effect was prevented if the subjects' head was cooled during exercise, a finding consistent with the proposal that cerebral heating can induce SWS (Horne, 1988, 1989). An increase in SWS is thought to counteract the effects of awake heat loads; in particular, cerebral heating. Such results may reflect the progressive proportional lowering of the threshold of sleep-inducing WSNs in response to prior heat exposure. There is some speculation that the fall in T_b is a response to a regulated reduction in the hypothalamic set point for thermoregulatory control (Parmeggiani, 1980, 1987, 1990). Alternatively, this threshold is elevated during the relative cooling of sleep (McGinty et al., 1990) such that a time-integration of waking brain temperatures equals the integrated cooling achieved during perfectly restorative sleep. Thus, sleepiness at any time is determined by comparison of hypnogenic threshold and hypothalamic temperature (McGinty et al., 1990; Parmeggiani, 1980, 1987, 1990).

Summary and Implications

It is evident that T_b influences sleep architecture, although the mechanisms that increase core temperature and delta sleep are unknown. It is assumed that the state of accelerated catabolism induced by the passive heating process regulates delta sleep (Horne & Staff, 1983). Since passive heating generally involves the immersion of the body in a hot bath set at approximately 43 °C, this method promotes the uptake of heat by the immersed parts of the body, as well as reduces evaporative heat loss (i.e., sweating) from the body by minimising the number of sites from which heat loss can occur (Keele et al., 1982). The net result is a rise in T_b, which in turn increases body metabolism and heat production (i.e., MR) by accelerating the oxidative activities of the body (Benedict & Talbot, 1921; Brobeck & DuBois, 1980; Grollman, 1930b; Keele et al., 1982). In fact, it has been reported that for every rise of 0.5 °C in T_b, the basal metabolism increases by seven percent (Keele et al., 1982).

The finding that passive heating during wakefulness increases the amount of SWS in the subsequent night's sleep period (Berger et al., 1988; Horne & Moore, 1985; Horne & Reid, 1985; Horne & Shackell, 1987; Horne & Staff, 1983) therefore holds particular relevance to the current thesis in that it serves as another technique of directly manipulating wake-time metabolism which appears to affect sleep architecture. While the validity of the passive heating effect appears established, it is unclear however if it can account for all of the positive effects that have been reported. It is believed that in order to observe the passive heating effect on SWS, T_b must return to normal levels before sleep onset (Horne & Shackell, 1987). That is, after having been passively elevated during the wake period, T_b then needs to have returned to control levels at the time of sleep onset in order for SWS increases to occur.

Circadian Rhythms

Circadian rhythms, a term coined by Halberg (1959), and its relevance to the human sleep-wake cycle will be the focus of this section. Many human behavioural habits are governed by circadian rhythms that are ascribed to the environmental cues associated with the solar day (Monk, 1989). Circadian rhythms are endogenous (Morris, Dawson & Lack, 1989), being the products of an internal biological clock mechanism very much like a physical oscillator (Pittendrigh & Bruce, 1957). They also are generated by one or more master circadian oscillators, or pacemakers, one of which has been located in the suprachiasmatic nucleus of the hypothalamus (Rusak & Groos, 1982). These are then synchronised by one or more zeitgeber/s, which are environmental time cues such as light, that entrain the circadian rest-activity rhythms displayed by most organisms (Mistlberger & Rusak, 1989).

Circadian rhythms closely reflect a 24-hour regular periodicity on a variety of different processes including behaviour (e.g., drinking, feeding), thermoregulation, metabolism, hormone secretion and several neurotransmitter receptors (Rusak & Zucker, 1979; Borbely & Tobler, 1989). These daily rhythms persist under constant environmental conditions (Rusak, 1980). They are self sustaining, and in the absence of zeitgebers, are said to "free-run" (Mistlberger & Rusak, 1989). Environmental factors such as ambient light intensity (Dawson & Campbell, 1990; Dawson, Morris & Lack, 1989a,b; Eastman, 1991; Wever, 1989) may act to regulate or set the phase relation between the organism and the environment. Circadian rhythms also can be modified by selective breeding (Bunning, 1973) and gene mutations (Konopka, 1980) and they develop normally in successive generations of organisms kept in constant light or dark (Davis & Menaker, 1981).

Probably the most familiar experience of the daily cyclicity is the pattern of sleep and wakefulness (Mistlberger & Rusak, 1989). Although the sleep-wake rhythm is normally highly correlated with T_b, in the absence of zeitgebers they become dissociated or uncoupled, seemingly controlled by different clocks (Anch et al., 1988).

In the following sections a brief overview of the processes of sleep, MR and T_b that are governed by circadian cycles will be presented.

The Circadian Cycle of the Sleep Process

Unlike many other physiological parameters such as T_b, the sleep cycle is non-unitary in nature being marked by its distinction from wakefulness; its composition of different sleep stages and; the fact that sleep onset contrasts with sleep offset (Dinges, 1989). Much of the research has assumed that human adults show a monophasic (circadian) sleep-wake cycle during which sleep generally occurs at the same time each night (Dinges, 1989). This effect depends upon the individual's phase of the cycle providing the wake state is maintained throughout the day. The results to emerge from such studies as Czeisler et al. (1980) strongly point to the influence of a circadian oscillator controlling both the sleep-wake and the components of sleep structure itself. It has been suggested however, that the sleep process may be governed by two oscillators alternating at two different periodicities (Dinges, 1989). Dinges (1989) suggests that if this is correct, then there must be at least two different biological "clocks" influencing sleep. The one that controls core temperature is "circadian" at all times (Dijk, Cajochen, Tobler & Borbely, 1991), whereas the one that underlies the sleep-wake cycle appears more variable in its period (generally ranging from >25 to $<$ 60 hours, although it may be $<$ 25 hours in desynchronised subjects, especially in women). The latter is therefore "weaker" relative to the temperature oscillator, that serves to keep it coupled to the circadian period during much of the time in temporal isolation.

The architecture of the adult human nocturnal sleep is characteristically defined by a predominance of NREM SWS occurring during the first few hours of sleep, with the latter half of the sleep period being dominated by REM sleep (Williams et al., 1974). These sleep stages are extremely consistent within an individual, and alternate cyclically showing a differential distribution across the night that is highly resistant to change (Dinges, 1989). Kleitman (1963) in fact, has suggested that sleep architecture itself, in particular the NREM/REM cycle, is more a dependent function of a rhythmic process common to sleep and wakefulness rather than on sleep alone. This view of the sleep cycle as a sleep-independent ultradian (90 minutes) rhythm has marked the turning point in the discovery of differential circadian control of sleep processes (Dinges, 1989).

Both sleep onset and SWS generally occur on the descending portion of the circadian T_b cycle. SWS is particularly affected by the length of prior wakefulness (Akerstedt & Gillberg, 1986), but only shows a slight relationship to the circadian temperature phase (Dinges, 1989). REM sleep on the other hand, varies with circadian phase appearing at a time corresponding with the rising phase of the temperature cycle and is influenced by circadian processes (Dinges, 1989). Sleep efficiency (TST/TiB) also has been reported to be primarily governed by circadian processes (Weitzman et al., 1980).

The Metabolic Rate Rhythm and Metabolic Rate Changes During Sleep

Extensive data reported in recent years from both animal and human studies suggest a link between sleep and metabolic activity. In rats, MR as measured by heat production, is lower in NREM sleep than in wakefulness and lower in REM than in NREM sleep (Roussel & Bittel, 1979). Human MR measures during sleep generally show a gradual fall for approximately 5 hours over the night (Buskirk, Thompson, Moore & Whedon, 1960; Kreider & Iampietro, 1959; Robin, Whaley, Crump & Travis, 1958; Yamashiro, Fukushima,

Okudaira, Suzuku & Nishi, 1987) followed by a rise (Buskirk et al., 1960; Fraser et al., 1989). The pioneering study of Brebbia and Altschuler (1965) showed MR to be slightly higher during REM compared to stages 3 and 4 of NREM, but in general distinctly lower than during the waking state. This finding has been consistently confirmed (Colrain, Trinder, Fraser & Wilson, 1987; Fraser et al., 1989; Haskell et al., 1981a; Palca et al., 1986; Shapiro et al., 1984). The decline in MR during sleep is not simply a consequence of decreased motor and digestive activity, since it occurs in fasted subjects, in patients with total bedrest, and even in paralysed humans (Heller & Glotzbach, 1985). In addition, the daily decline of MR during sleep cannot be due entirely to sleep since it occurs in subjects deprived of sleep (Kleitman, 1963; Kreider, 1961). Clearly there are both sleep and circadian influences on MR where the reduction in MR during sleep is enhanced by underlying endogenous circadian rhythm effects (Grollman, 1930a; Kleitman, 1923). The consistent reduction of MR at sleep onset in human adults is paralleled with other thermoregulatory changes, such as T_b decreases.

Further evidence in support of MR reductions during sleep is derived from studies using infants. Infants have been found to demonstrate a pattern of MR reduction at sleep onset similar in nature to human adults. Studies of thermal balance and metabolism during sleep in both term and pre-term infants have shown relative increases in O_2 consumption during REM sleep compared with NREM sleep (Kairam, Schulze, Koenigsberger & James, 1979). The significantly higher levels of REM sleep compared to NREM sleep at thermoneutrality (31.5 to 33.5 ^{o}C) in the first week of life for term infants was found to occur provided REM followed NREM sleep (Stothers & Warner, 1978, 1984). This MR difference between REM and NREM is particularly accentuated at cool T_as (28.5 to 29.5 ^{o}C) by approximately 15%.

In overview, both adult and newborn humans show elevated MR during REM sleep compared with NREM sleep, and this difference may be heightened at T_as below thermoneutrality. The drop in MR

between Wake and NREM sleep seen in all mammals is most likely due to the alterations in the thermoregulatory system between these two states (Glotzbach & Heller, 1989).

The Body Temperature Rhythm

Many research findings have demonstrated the existence of a diurnal rise and nocturnal fall in T_b that continues to occur even in bedrested individuals (e.g., Conroy & Mills, 1970). Therefore, this satisfies the criteria for an endogenous circadian rhythm. Although this pattern is consistent within a single individual, there is nevertheless, large individual variation across subjects (Horne & Ostberg, 1977). Researchers also have differed in the temporal positioning of the maximum and minimum temperature (Conroy & Mills, 1970). Much of this variability could be accounted for by differing behavioural patterns within subjects, or a difference in the phase relationship between the T_b rhythm and the individual's sleep/wake cycle. The normal diurnal variation of T_b is between 1-2°C (Aschoff, 1970; Conroy & Mills, 1970; Schmidt-Nielsen, 1981) with a minimum being reached at 0500-0600 hours and a peak at about 1600 hours (Cabanac, Hildebrandt, Massonnet & Strempel, 1976; Colquhoun, 1971; Faria & Drummond, 1982; Glaser & Shephard, 1963; Hildebrandt, 1974; Kleitman & Kleitman, 1953). In general, it appears that T_b is low during sleep and high during the active period. Although activity levels, food intake, T_a and sleep have specific effects on temperature regulation, they do not generate the T_b rhythm, but rather act to modulate the regulated temperature (Fuller & Sulzman, 1982; Fuller, Sulzman & Moore-Ede, 1978; Fuller, Sulzman & Moore-Ede, 1980).

Recent work investigating the effects of nocturnal bright light exposure on human circadian rhythms has shown that bright environmental illumination can shift the phase of the human circadian T_b rhythm (CTR) (Edelson, Tirney, Gaddy, Stewart & Brainard, 1991). It also has been associated with an acute attenuation of the normal nocturnal drop in T_b, significant elevation in tympanic

(Badia, Culpepper, Boecker, Myers & Harsh, 1990; Badia, Culpepper, Myers, Boecker & Harsh, 1990; Edelson et al., 1991; Murphy, Myers, Badia & Harsh, 1991; Myers & Badia, 1991) and oral (Hannon, Brainard, Gibson, French, Arnall, Brugh, Littleman-Crank, Fleming & Howell, 1991) temperature, and increased sleep latency (Badia, Myers, Boecker & Murphy, 1990). It has been speculated that these effects follow bright light exposure since there is an effective suppression of the release of the photosensitive hormone melatonin that may be instrumental in processes of temperature regulation (Badia, Culpepper, Boecker, et al., 1990).

Body temperature depends on a balance between heat production and heat loss. It is therefore possible that the circadian periodicity in heat production is responsible for the T_b rhythm, especially if the corresponding circadian variations between heat production and fluctuations in the T_b rhythm are considered. For example, both share their lowest values early in the morning (Bornstein & Volker, 1926; Deighton, 1933), although specific attempts to closely correlate temperature curves with variations in energy metabolism have been unsuccessful (Metz & Andlaver, 1949). More specifically, it seems likely that the T_b rhythm is a function of either the temperature regulating mechanism as a whole, or the hypothalamic mechanisms rather than any one component of the heat production or heat losing mechanisms (Conroy & Mills, 1970). Variations in the heat-loss mechanisms, such as cutaneous blood flow, are probably more important in regulating the T_b rhythm, and it has been shown that the daily variation in skin temperature is almost a mirror image of the change in T_{re} (Halberg, Halberg, Barnum & Bittner, 1959). The "critical skin temperature", the cutaneous temperature below which if the room temperature falls heat production must be increased linearly to maintain the T_b, shows a rise at night.

Since human T_b is easily measured as a continuous physiological variable and demonstrates an extremely stable free-running circadian periodicity with minimal variability during

temporal isolation from zeitgebers, it has constituted the "touchstone" circadian parameter in humans for investigating sleep-wake cycle oscillations during isolation (Dinges, 1989).

Circadian Interactions of Temperature and Sleep

A further factor to be considered in the relationship between T_b and sleep is the relevance of the CTR. As has been reviewed earlier, much evidence has suggested that the daily T_b rhythm and thermoregulatory responses are influenced by both circadian and sleep-related properties with the circadian changes in the thermoregulatory system being independent of arousal state (Glotzbach & Heller, 1989). There has been evidence to suggest that the CTR is a consequence of a deep internal circadian controlling system rather than of the daily activity cycle (Kleitman, 1963). This is supported by the results from studies using inactive, immobile subjects undergoing minimal muscular activity who nevertheless continue to display a cyclic T_b pattern (Kleitman, 1963). In addition, the daily fluctuations in T_b appear to be more dependent upon changes in thermal conductance rather than in heat production (Smith, 1969).

Sleep-related declines in T_b which are considered to "mask" components of the circadian T_b rhythm are at a maximum (approximately 0.5°C) during the descending phase of the T_b rhythm (Gillberg & Akerstedt, 1982; Mills et al., 1974). During sleep deprivation T_b continues to show daily fluctuations (Kleitman, 1963; Kreider, 1961) and specific thermoregulatory responses in awake subjects vary with a daily rhythm (Wenger, Roberts, Stolwijk & Nadel, 1976). The T_b rhythm particularly affects the timing and duration of human sleep (Glotzbach & Heller, 1989). As mentioned earlier, without zeitgebers to provide environmental cues, the sleep-wake and T_b cycles may dissociate and free-run under different circadian periodicities (Czeisler, Weitzman et al., 1980; Czeisler, Zimmerman et al., 1980; Zulley, Wever & Aschoff, 1981). Such conditions often shift voluntary bedtime and sleep onset to a time

corresponding to the lowest point or nadir of the circadian cycle (Czeisler et al., 1980). If bed rest coincides with the rising phase of the T_b rhythm, this is generally associated with greater sleep disturbance (Czeisler, Zimmerman et al., 1980; Zulley et al., 1981). The timing and duration of napping periods also are affected by the phase of the T_b cycle in free-running subjects. Naps are generally longer if they occur at a time when T_b is at a minimum rather than half-way between minima (Campbell & Zulley, 1985). In addition, the mean duration of sleep is longer if an individual falls asleep closer to the temperature maximum rather than at the temperature minimum (Czeisler et al., 1980).

Even though it has been reported that the amount of SWS is contingent upon the duration of prior wakefulness (Akerstedt & Gillberg, 1986; Borbely et al., 1981; Feinberg, March, Floyd, Jimison, Bossom-Demitrack & Katz, 1985), it is the phase of the T_b cycle at bedtime that influences sleep parameters. The sleep variable most strongly affected by the T_b cycle is REM sleep which shows the shortest latency, the longest duration and the greatest amount at a time corresponding to the T_b minimum (Czeisler, Zimmerman et al., 1980). The effect of the T_b cycle on sleep has been demonstrated in free-running subjects with desynchronised cycles of T_b and bedrest activity. For example, Akerstedt and Gillberg (1981) demonstrated that the phase of the T_b rhythm at bedtime affected sleep architecture more than the duration of prior wakefulness in sleep deprived subjects entrained to a normal 24-hour day whose bedtime was regulated at varying times beginning at different points in the T_b cycle. Therefore, the daily T_b rhythm exerts strong influences on sleep architecture although there are many other factors that affect sleep structure (Glotzbach & Heller, 1989).

Conclusions

Much of the research directly investigating the effects of T_b on sleep properties supports a relationship between thermoregulation

and sleep. The passive heating studies (e.g., Horne & Porter, 1976) have especially shown a T_b-SWS relationship which has been suggested as being partly attributed to cerebral heating (Horne, 1988). Ultimately, this indicates that cerebral metabolism may be an important factor contributing to sleep architectural changes. In order to provide a clearer assessment of such a relationship however, there needs to be further research designed to manipulate T_b without circadian effects, allowing an insight into the level at which T_b alone affects sleep architecture. A study investigating the effects of T_b on subsequent sleep architecture is reported in Chapter 6.

CHAPTER 5

EXPERIMENT ONE:

THE EFFECTS OF EXERCISE ON METABOLIC RATE AND SLEEP ARCHITECTURE IN THE FIRST SLEEP CYCLE

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There is much evidence that points to a relationship between MR and sleep architecture (Berger, 1984; Trinder et al., 1982; Walker & Berger, 1980; Zepelin & Rechtschaffen, 1974). However, the nature of this relationship remains unclear. This is especially true given some of the previous research findings that have shown conflicting effects of daytime increases in energy expenditure on subsequent sleep architecture depending upon the fitness level of the subject population (Trinder et al, 1985). Closer assessment of the processes involved is therefore required.

The aim of this study was to explore the relationship between MR and SWS by attempting to change daytime O_2 consumption through intense exercise sessions in endurance athletes, so that subsequent effects on the first sleep cycle (Bunnell et al., 1983a) and sleep-time MR could be assessed. Endurance athletes have consistently been shown to have increased levels of SWS following exercise (Griffin & Trinder, 1978; Montgomery et al., 1982; Trinder et al., 1985), and it is this population of subjects that generally tolerate high levels of exhaustive exercise which may be necessary to facilitate these sleep changes (Shapiro & Verschoor, 1979). To maximise the possibility of an exercise-induced MR effect, endurance athletes were therefore considered as being more likely able to tolerate conditions involving exhaustive levels of exercise. Furthermore, the study attempted to test whether the increased SWS in endurance athletes following intense levels of exercise is a function of the increased wake-time energy expenditure, or more specifically a function of subsequent MR elevations during the sleep period as a result of prior exercise. To optimize the effect of increased wake-time MR into the sleep period, an evening exercise session was included since it is unknown whether

intense exercise in the late afternoon results in increased MR that would sufficiently be sustained into the first sleep cycle. This is particularly important since trained individuals approach baseline O_2 consumption levels more rapidly than their untrained counterparts, even following the same work intensity (deVries, 1980). Metabolic rate elevations however, are more likely to be sustained into the first sleep cycle following late evening exercise immediately prior to bedtime than late afternoon exercise, due to the difference in their temporal relationship to the sleep period.

Previous findings that show increased SWS levels in endurance athletes following daytime exercise (Baekland & Lasky, 1966; Horne & Moore, 1985; Horne & Staff, 1983; Shapiro et al., 1975; Shapiro & Verschoor, 1979) are consistent with energy conservation and bodily restorative theories (Adam, 1980; Oswald, 1980; Berger, 1984; Berger & Phillips, 1988a). They suggest that sleep reacts in a compensatory manner to variations in energy expenditure or MR during wakefulness. Whether it is increased MR during wakefulness, or sustained exercise-induced increases in MR into the first sleep cycle that facilitates SWS increases in endurance athletes, still remains to be assessed.

With this view, the present study attempted to manipulate wake-time MR using an exercise routine (i.e., 19 km run) in order to investigate its effect on subsequent sleep architecture and sleep MR. Energy expenditure generally shows a clear response to physical exercise of high intensity. Increased MR during intense exercise was assumed to be sustained at least into the first cycle of the sleep period as a result of "post-exercise effects" and the "O_2 debt" during the recovery period (Hill, 1922), and on the basis of previous reports that have shown basal MR increases to be sustained for 12 to 24 hours (Wilmore, 1977) or even up to 72 hours (Simonson, 1971) after strenuous work. It was hypothesised that increased wake-period MR would be associated with increased levels of SWS.

METHOD

Subjects

Ten physically fit males with a mean age of 21.8 years (SD = 3.49) were recruited from the University community. All subjects were paid for their participation in the experiment. Subject selection was dependent upon fitness level and on the subjects having a history free from respiratory and sleep pathology. In addition subjects were non-smokers and free from medication. Level of fitness was assessed by bicycle ergometer where subjects were required to show a maximal O_2 uptake (VO_2max) greater than 3.7 litres/minute (or 52 ml/kg/min. adjusting for weight). All subjects were engaged in regular exercise at the time of the experiment and were experienced distance athletes undergoing additional training sessions in rowing, cycling, football and soccer. As a result of their habitual exercise programme it was assumed that all subjects could theoretically successfully perform the required exercise. The mean VO_2max values for the experimental population were 4.37 litres/minute (SD = 0.85) and the averaged age adjusted O_2 uptake value of the subjects was 67.7 ml/kg/min. (SD = 14.95) (see Table 5.1). The project was approved by the University of Tasmania, Human Ethics Committee and all subjects completed a consent form.

Design

In a repeated measures design four experimental conditions were conducted in counterbalanced form. The experimental conditions were preceded by a single adaptation night. The four conditions included:

1. Late evening exercise before bedtime (LE).
2. Late afternoon exercise followed by a meal (LAM).
3. Late afternoon exercise without intervening meal (LA).
4. No exercise control day (CN).

Table 5.1

Relevant Subject Characteristics

SUBJECT (Initials)	AGE (Years)	WEIGHT (kg)	VO_2max (l/min)	Adj.VO_2max (ml/kg/min)
D.C.	19	71.0	65	4.0
J.P.	21	60.0	65	4.0
L.N.	26	75.5	85	6.0
M.L.	18	62.5	74	4.0
N.M.	20	70.6	59	4.0
P.M.	21	78.2	103	6.0
P.N.	29	81.1	54	4.2
P.W.	21	63.5	58	3.4
R.G.	25	75.9	62	4.2
S.R.	18	59.0	52	3.7
Mean	21.8	69.7	69.3	4.4
SD	(3.7)	(8.0)	(14.8)	(0.90)

The independent variable was the exercise condition designed to increase MR, while the dependent variable was sleep architecture with particular focus on the amount of SWS in the first cycle of sleep.

Condition 3 (LA) was designed to test for any compounding effects of consuming an evening meal on MR. This was considered a necessary precaution since it was assumed that a more accurate assessment of post-training run increases in O_2 consumption that may be sustained into the sleep period would be achieved if possible confounding thermic effects of food were removed. The thermic effect of food is a well established phenomenon (Rothwell & Stock, 1981). Even though it has been reported that endurance athletes have efficient thermoregulation and a reduced thermic effect of food (LeBlanc, 1986; LeBlanc, Mercier & Samson, 1984), the magnitude of the stimulating effect on metabolism depends upon the constituents of foodstuffs. For example, a protein meal increases metabolism by 10-35% within three to five hours after food consumption, with a total increase in metabolism averaging approximately 20% over a period of four to six hours (Keele et al., 1982). On the other hand, consumption of an equivalent calorific amount of carbohydrate and fat increases metabolism to a lesser degree being within the order of only 5-10%. Normal mixed diets have the effect of increasing metabolism by 50-150 Kcal daily (Keele et al., 1982). As there appears to be little evidence to indicate whether or not a meal consumed several hours before bedtime will exert a metabolic effect during the sleep period, it is considered appropriate to assess this effect in the study.

A comparison over conditions 1, 2 and 4 provided a means of testing the effects of exercise on MR as measured by indirect calorimetry (i.e., O_2 consumption) and any following chronic effects on subsequent quality of the first cycle of sleep. A comparison over conditions 2 and 3 allowed a test of the effects of consuming a meal on MR. Since the range of variation in basal MR has been found to be large for any one individual (Ryan, Mlynczak, Erickson, Man & Man, 1989), repeated measures were undertaken .

Procedure

Measurement of Fitness

The subjects were tested for fitness using a bicycle ergometer. Physical fitness was defined in terms of predicted VO_2max using the submaximal exercise nomogram method described by Astrand and Rodahl (1970).

The 19 km running course was considered to be sufficiently effective in producing MR increases in fit subjects. Whether these increases would be sustained into and during the sleep period was open to investigation. Many studies using such exercise loadings however, have reported first cycle effects (see Table 3.1). The run was considered not to be more stressful than any other serious training run that each subject included within their normal weekly training schedule.

Measurement of Metabolic Rate

Metabolic rate was measured and expressed in the form of O_2 consumption using a method of indirect calorimetry. The associated CO_2 production was not measured in this study. The collection of O_2 consumption was achieved using a C.I.G antistatic anaesthetic mask attached to a Rudolph #2600 or #2700 two-way breathing valve. The mask was used in preference to a hood since its use enabled a more accurate breath by breath analysis of ventilation and O_2 consumption. Each subject was provided with a size mask most suited to the shape and configuration of the face to minimise potential problems of leakage during data collection and to maximise subject comfort. The mask was fitted with an inflatable cuff and held securely in place with a three-sided head strap. Prior to experimental sessions, each subject wore the mask on a few adaptation sessions to allow familiarisation as well as a training period aimed at increasing subjects' awareness and ability to detect mask leakage. Since recordings were to be only taken across the first sleep cycle, it was considered that this time period would be short enough to avoid any possible measurable

disruption to sleep as a result of the constraints exerted by wearing the mask. Subjects also were encouraged to sleep on their back to avoid possible mask dislodgement which would otherwise lead to substantial air leakage. While the detection of leakages during the sleep period may appear difficult and potentially disruptive, subjects nevertheless were able to acquire through practice, an almost automatic and subconscious ability to detect potential leakages, and were on the whole able to rectify such problems by readjusting the position of the mask, when necessary. Further precautions included close scrutiny of the ventilation record during data collection. This was an effective measure for alerting the experimenter to those times when respiratory patterns suggested air leakage. Only a small quantity of data needed to be excluded from the final analysis as a result of such effects.

The deadspace values of the valves were 33.3 cc and 118.8 cc respectively. Since the mask deadspace was dependent on facial configuration, the total deadspace of the mask and valve varied from 115.3 cc to 233.8 cc. Measurement of mask deadspace was achieved using a technique of water displacement with the valve removed. Air was collected for O_2 consumption analysis from an outlet in the breathing valve located where the mask connected with the valve. The sample was transported via 110 cm of 1 mm internal diameter tubing to an Applied Electrochemistry S-3A Oxygen Analyser. The O_2 analyser was calibrated to fresh air at the beginning of each session and periodically to tank air of known concentration. The subject's bedroom was ventilated with fresh air throughout the period of each experimental session and the room temperature was maintained within thermoneutral range (i.e., 20-24 ^{o}C) using a thermostatically controlled oil-filled electric heater. The rate of expiratory air flow was measured via a Fleisch pneumotacograph (connected to the major outlet of the breathing valve) and a Gould PM 15E pressure transducer.

The output signals of both the O_2 analyser and pressure transducer were amplified using a Beckman R411 polygraph recorder

and input to a D.E.C. PDP-11/23 computer via an eight-bit analogue to digital converter. The collected data was stored on a thirty megabyte Winchester hard disk. The sampling rate was 20 Hz for both variables. Software running under RT-11 Pascal was developed within the laboratory to enable online computerised display of incoming data using a high resolution graphics screen. Additionally, the integrity of the data being collected was checked throughout the experimental session via paper chart, a variety of digital voltmeters and computer alarms. Consequently, these safety devices ensured that the experimenter was suitably equipped to identify and locate the source of any data collection problem. In response to any aberrant signals, the equipment was checked and adjusted or recalibrated as necessary.

The accurate measurement of MR by indirect calorimetry may be affected by ventilatory instability. Rapid shifts in ventilation, unrelated to metabolic stimuli, are associated with less rapid shifts in arterial O_2 levels and thus expired O_2. The delay in shift in O_2 levels is approximately 30 seconds. In these instances O_2 consumption, when measured on a breath by breath basis, will be underestimated when ventilation falls, and overestimated when ventilation rises. Due to a documented reduction in ventilation at sleep onset (Colrain et al., 1987), O_2 consumption during this transition period would be underestimated. Under these conditions of continuous ventilatory fall, actual O_2 consumption values would be expected to lag approximately 30 seconds behind the measured values.

Oxygen consumption was determined for each breath by integration of the cross product of O_2 level and rate of expired air flow allowing O_2 consumption values to be obtained for each breath, each minute, or minute values extrapolated from individual breaths. Minute O_2 consumption (VO_2) was used for this study, and subsequent studies reported in the current thesis. In addition to VO_2, respiratory cycle duration (CD), the tidal volume of expired air corrected to STP (Vt), Minute ventilation of expired air (V_E) and fractional end tidal O_2 level ($FetO_2$) were available for analysis.

Measurement of Wakefulness and Sleep

The four experimental conditions were conducted over non-consecutive nights. Sleep stages were identified using EEG, Electrooculogram (EOG) and Electromyogram (EMG) signals. The EEG was traced using copper disk electrodes applied to positions C3/A2 (International 10-20 system), and a single EOG was recorded using electrodes vertically displaced on the outer canthi of the eyes. EMG was recorded using a bilateral submentalis placement of two electrodes. All recordings were amplified and collected on paper chart using a Beckman R411 Dynograph. EEG amplification was set at 100 u/cm with a time constant of 1 second. Paper speed was set at 10 mm/sec. This procedure also was used in Experiments 2 and 3.

Since the laboratory was not equipped with digital recording methods in order to subject EEG-data to power spectral analysis through a Fast Fourier Transformation (FFT) routine, the recording and scoring of the records complied with the more arbitrary and limited standardised procedures instituted by Rechtschaffen and Kales (1968). The sleep records were scored in 30 second epochs by two blind scorers. The scorers had an interrater agreement over 90%. The data was later combined into 60 second epochs in order to equate the duration of the sleep-wake scoring unit with that of the metabolic and respiratory variables.

During the 30 minute resting MR measurements, prior to lights out on all conditions, the associated EEG recordings allowed careful monitoring of subjects' waking state, since it has previously been found that the loss of alpha activity is directly associated with a substantial reduction in ventilation (Colrain et al., 1987). As mentioned previously, the subsequent adjustment in arterial O_2 level is less rapid and consequently O_2 consumption values are underestimated. Additionally, it has been suggested that the early period of sleep is associated with an effective reduction in MR (Colrain et al., 1987). Hence, waking MR is likely to be underestimated if subjects become drowsy and enter stage 1 sleep.

This demonstrates the importance of careful monitoring of the subject's EEG during assessment of resting MR. In this study, audio-visual stimulation using television, videos, or radio/cassettes were used to assist subjects in maintaining a wake state, a provision commonly used as standard procedure in the measurement of resting MR.

For a number of reasons, data collection was restricted to the first sleep cycle. This period relates to the first 2-3 hours of the sleep cycle when most SWS occurs. Table 3.3 showed those studies that have successfully used the first sleep cycle to document changes in sleep architecture. By deduction then, confining recordings to this period of the night should allow possible effects of prior MR manipulations on SWS architecture to be captured within this period. Only a few recent studies show the effects of pre-sleep activity to be most likely revealed during the first cycle of the sleep period. Many of these studies show the first sleep cycle is sufficient time for demonstrating any sleep architectural changes within this period. Further investigations later in the sleep cycle would therefore be considered unnecessary when SWS constitutes the major dependent variable.

Tilley, Donohoe and Hensby (1987) emphasised that restriction to the first cycle of sleep only increases the likelihood of losing more information pertaining to the stages of REM and stage 2 sleep (approximately 60% in both cases) than SWS data (approximately 20%). Also, confining sleep recordings to the first cycle in this study would reduce the time period during which the subject may experience discomfort due to wearing the anaesthetic mask and minimise the chances of mask displacement during the night.

As Shapiro (1981) noted many of the studies investigating the changes after low levels of exercise have been confined to the first sleep cycle. In these situations it has been suggested that the recovery process has occurred in this period and therefore the remainder of the sleep is unaltered. Horne (1988) also suggested that core sleep (i.e., the first part of the sleep) is most important to the restoration

process whereas optional sleep (i.e., occurring later in the sleep cycle) is more flexible in terms of its necessity in the overall restorative process.

On the basis of these previous research findings then, sleep recordings for the current study were confined to the first cycle. This was considered sufficient time to reveal any effects on sleep architecture as a consequence of previous manipulations.

General Laboratory Procedure

All conditions differed only in respect to the temporal placement of the exercise programme which was performed largely during the winter months. The resting MR of subjects was measured across 30 minute intervals between the hours of 1530-1600; 2000-2030 and prior to normal lights out (i.e., usually 2230-2300 hours). These resting MR sessions effectively allowed a record of the magnitude of MR elevation immediately before and after the exercise session. Thus, a pre- and post-exercise O_2 measurement could be documented. Starting times for conditions 2 and 3 of the late afternoon run was 1600 hours, and the late evening exercise condition at 2030 hours (see Table 5.2). The specified time schedule was adhered to by all subjects.

On all occasions subjects arrived at the laboratory at 1500 hours to be fitted with the mask and electrodes. The laboratory comprised a sound attenuated room and an adjacent monitoring equipment room. At 1530 hours subjects were required to rest in a supine position on the bed for 30 minutes while resting MR and EEG data were recorded. As stated previously, it was important to monitor EEG to establish whether the subject remained awake throughout this recording period. Also during the awake recording sessions, the lights were dimmed while subjects watched television or listened to music whilst in a supine position. Since both upright posture (Kleitman & Doktorsky, 1933) and activity (Gander, Graeber &

Table 5.2

Diary of events during each experimental session

TIME		EVENT
Start	**Finish**	
1530	1600	**RMR** Measurement
1600	1730	**LAM** 19 km training run
		LA " " "
1800	1900	Evening Meal
		[except condition **LA**]
2000	2030	**RMR** Measurement
2030	2200	**LE** 19 km training run
2200	2300	Preparation for bed
2300	2330	**RMR** Measurement
2330	0200	Sleep period

Abbreviations:

LAM = Late Afternoon Exercise with Meal.

LA = Late Afternoon Exercise without Meal.

LE = Late Evening Exercise.

RMR = Resting metabolic rate.

Note. The Control condition required subjects to be sedentary. On experimental days the subjects only performed the specific exercise (i.e., 19 km run).

Connell, 1985) affect MR, supine positioning was used to control for any masking by upright posture or activity of the hypothesised effects on MR.

At 1600 hours on the late afternoon exercise conditions (LAM & LA) subjects were required to commence their specified 19 km run and instructed to aim for a 1.5 hour time in order to maximise the workload ensuring maximal MR increases. The average completion time for the run was 1 hour 35 minutes, with a range of 1 hour 10 minutes to 2 hours. On completing the run, subjects were refitted with the mask and post-exercise MR recorded. This allowed a measure of the degree of increase in MR as a function of the exercise condition.

The late afternoon exercise conditions differed on one aspect: an evening meal followed one late afternoon exercise session (LAM) and not the other (LA). This provided a means of documenting any confounding effects of the evening meal on MR.

For the condition LE, subjects arrived at the laboratory at 2000 hours to be fitted with the mask and electrodes. A 30 minute resting MR measurement was recorded between 2030 and 2100 hours. Following this session subjects commenced the 19 km run at 2100 hours. Immediately on return from the run a further post-exercise measurement was recorded.

Subjects returned to the laboratory one hour before the scheduled 2300 hours bedtime to allow for preparation and physiological adjustment to the laboratory environment. Prior to retirement, electrodes were attached and the mask secured. Subjects wore light night attire and slept under bed-clothes which could be adjusted for optimal comfort. Sleep recordings commenced at 2330 hours following the 30 minute resting MR session. Metabolic rate recordings also were continued throughout the first cycle sleep period which was terminated three hours after lights out for all subjects.

The first night in the laboratory constituted an adaptation night allowing dissipation of possible "first night effects" (Agnew, Webb & Williams, 1966) as well as providing an effective opportunity to screen for potential periodic breathers. Detection of periodic breathing patterns in subjects led to their exclusion from the study. In the present study, no periodic breathing patterns were detected in any of the individuals screened.

All subjects were instructed to refrain from any extracurricular exercise on the experimental days and from drinking alcohol and caffeinated substances such as coffee, tea, or chocolate. They also were required to closely adhere to a diet that was calorifically controlled across experimental sessions. A chart of the food intake for the evening meal was recorded and the calorific content documented.

Data Preparation and Statistical Analysis

The stored digitised data collected across the first cycle of sleep was itemised into several respiratory variables by laboratory developed software. These data were analysed using a breath by breath procedure that calculates minute by minute values. The software-based determination of what constituted each breath was subject to verification by the experimenter. The formulated program was essentially 100% accurate as determined by the experimenter, provided there were no marked artifactual data produced by gross body movements. Data generated by periods of smaller body movements were usually permissible in their inclusion within the MR averaging. It only was within periods of gross body movements that the available program failed to accurately identify distinct breaths. Each one minute epoch was stored and ultimately included in the subsequent analyses providing there was accurate identification of breaths within at least 30 seconds of each epoch. Minute values for the time recorded for each condition were then calculated.

Oxygen consumption was measured over 30 minutes and averaged across the last 20 minutes to exclude the effects of possible

heightened arousal that could occur early in recording sessions. Oxygen consumption was averaged across all subjects starting at 1530, 2000 and 2230 hours (see Table 5.3). Table 5.4 shows pre- and post-exercise MR measurements and includes the averaged O_2 consumption data monitored over 30 minutes from 1530 and 2000 hours, and immediately after the running session in only eight subjects. Data from two subjects during the post-exercise recording session was unavailable due to equipment failure, and was excluded from this analysis. This table therefore effectively shows the immediate effects of exercise on O_2 consumption by presenting pre- and post-exercise measurements. Thus the apparent discrepancy in the O_2 consumption data at times 1530 and 2000 hours on experimental days between tables 5.3 and 5.4 is attributed to the differences in sample size used in each set of analyses. In addition, since the analyses using both unadjusted and adjusted O_2 consumption for body weight produced essentially the same statistical results, only those ANOVAs using the unadjusted O_2 consumption data will be reported. Raw data and ANOVA summary tables are presented in Table B-1 in Appendix B.

A power analysis was performed upon the results from a number of studies that have assessed the effect of intense exercise upon subsequent sleep in order to predict the probability of finding a significant sleep effect following such levels of exercise. The sample of studies selected to be used in the analysis were taken from research laboratories outside of the present laboratory in order to remove possible bias (see Appendix Table B-2). Significantly more of these studies confirmed a SWS effect following intense exercise than not, where the power of predicting a SWS effect following exercise was a high 0.74 at $p<.05$ level for a two-tailed test.

RESULTS

To assess the direct post-effects of exercise on O_2 consumption during the three exercise conditions, a 3 x 2 Condition (LAM, LA, LE) by Time (i.e., pre- and post-exercise; 1530hrs-postrun for LAM + LA

conditions; 2000hrs-postrun for LE condition) ANOVA was performed (see Table 5.4). The results from this analysis showed a significant main effect for Time [$F(1,7) = 9.09$, $p < .05$]. The main effect for Condition [$F(2,14) = 0.48$, $p > .05$] and the Condition by Time interaction [$F(2,14) = 1.82$, $p > .05$] differences were not significant. Dependent groups t-tests revealed that both the late afternoon exercise sessions including, and excluding, the evening meal produced significant elevations in O_2 consumption [$t(7) = 2.411$ $p < .05$, and $t(7) = 2.585$, $p < .05$ respectively]. The direct increase in O_2 consumption following the LE condition however, was not significant [$t(7) = 2.101$, $p = .07$].

To compare the overall effect of exercise on O_2 consumption to control conditions at selected times, a 4 x 3 Condition (CN, LAM, LA, LE) by Time ANOVA for the times 1530, 2000 and 2230 hours was performed (see Table 5.3). The results revealed a significant Time effect [$F(2,18) = 5.04$, $p < .05$]. There was no significant main effect for Condition [$F(3,27) = 1.15$, $p > .05$] suggesting that there was no significant difference in O_2 consumption across experimental conditions following exercise, and no Condition by Time interaction [$F(6,54) = 1.39$, $p > .05$]. Post-hoc analyses using dependent groups t-tests however showed no significant effect for the experimental conditions LAM [$t(9) = 1.87$, $p = .09$], LA [$t(9) = -0.09$, $p > .05$] and LE [$t(9) = -1.14$, $p > .05$] between time intervals 1530 to 2230 hours. These results suggest that although there were significant changes in O_2 consumption at the time intervals following the experimental 19km run, the changes in O_2 consumption returned to a level that was not significantly different from baseline at the time of retirement (see Table 5.3).

The sleep data was averaged across all subjects with the exclusion of data from one subject [D.C.](see Table 5.6). This subject found the anaesthetic mask to be particularly uncomfortable during the sleep period, and as a result extreme sleep disturbance was noted. A repeated measures ANOVA showed no significant main effects of Condition on any of the selected sleep variables or any interaction

effects between the sleep variables and Condition (See Appendices). That is, exercise did not appear to affect sleep architecture, and SWS levels did not significantly change across experimental conditions.

To assess the relationship between sleep and MR a correlation analysis was performed between sleep variables taken from the first sleep cycle and O_2 consumption data taken from the specific times outlined earlier. Sleep variables were entered as minutes during the first sleep cycle. For the correlation analyses, data were entered for each subject on each condition (i.e., CN, LAM, LA and LE). The sleep and MR data for one subject were however excluded from the correlation analyses for the reasons defined above. The results shown in the correlation matrix (Table 5.5) indicated significant positive correlations between O_2 consumption at 2000 and 2230 hours and both sleep and SWS onsets. This suggested that increased MR at these times appeared to be associated with delayed sleep and SWS onset. There were no other significant correlations between O_2 consumption at specific times and Stages 3 and 4 sleep or SWS.

Of interest was the reasonably large variation in O_2 consumption data between subjects, although adjusting for body weight reduced this effect. Error variability is an issue with these recordings but every precaution was taken to ensure the integrity of the data. The degree of variation in the VO_{2max} capacity within the sample of athletes used in this study may also have accounted for some of the variability in O_2 consumption between subjects. For example, subject PM appeared to have a much higher VO_{2max} compared to subject SR (see Table 5.1), even though both were of above average fitness on the basis of testing criteria of Astrand and Rodahl (1977). This difference in the level of VO_{2max} would mean that the subjects could have worked at different capacities of VO_{2max} during the 19km run, thus contributing to increased deviations in O_2 consumption between subjects. On reflection, to overcome this possible effect there may have needed to be an equalisation of energy expenditure by prescribing graded exercise loads according to individual subjects' maximum capacities.

Table 5.3

Mean Oxygen Consumption during each 30 minute resting MR session at selected times for the four experimental sessions ***(n = 10)***

	MEAN OXYGEN CONSUMPTION (ml/min.) (SDs in Parentheses) [O_2 Consumption adjusted for body weight (ml/kg/min.)]			
TIME	**CN**	**LAM**	**LA**	**LE**
1530hrs				
O_2 consumption:				
ml/min	278(73.2)	275(123.9)	303(98.6)	301(98.6)
ml/kg/min	[4.02(1.14)]	[4.25(1.46)]	[4.41(1.60)]	[4.41(1.69)]
2000hrs				
O_2 consumption:				
ml/min	286(96.7)	332(154.7)	319(113.4)	319(106.9)
ml/kg/min	[4.11(1.43)]	[4.89(2.63)]	[4.66(1.89)]	[4.65(1.74)]
2230hrs				
O_2 consumption:				
ml/min	275(82.6)	319(134.6)	302(128.7)	291(102.2)
ml/kg/min	[3.96(1.19)]	[4.67(2.16)]	[4.41(2.17)	[4.26(1.69)]

Abbreviations:

LAM = Late Afternoon Exercise With Meal

LA = Late Afternoon Exercise Without Meal

LE = Late Evening Exercise

CN = Control

Table 5.4

Mean Oxygen Consumption for Pre- and Post- Exercise sessions during all Exercise conditions
(n = 8)

	MEAN OXYGEN CONSUMPTION (ml/min.) (SDs in Parentheses) [O_2 Consumption adjusted for body weight (ml/kg/min.)]		
TIME	**LAM**	**LA**	**LE**
1530hrs			
O_2 Consumption:			
ml/min	258 (72.4)	262 (61.6)	
ml/kg/min	[3.73(1.06)]	[3.78(0.97)]	
POSTRUN			
O_2 consumption:			
ml/min	290 (96.3) *	320 (105.9) *	
ml/kg/min	[4.19(1.45)]	[4.61(1.59)]	
2000hrs			
O_2 consumption:			
ml/min			275 (68.1)
ml/kg/min			[3.99(1.15)]
POSTRUN			
O_2 consumption:			
ml/min			300 (92.7)
ml/kg/min			[4.39(1.55)]

* Significant ($p < .05$) increases in O_2 consumption immediately following the 19 km run.

Note. The statistical analyses are described in the text. The data from two subjects (J.P. and P.M.) for O_2 measurements directly taken after each exercise session was excluded from the analyses due to equipment failure, and this accounts for the difference in *n* between tables 5.3 and 5.4.

Table 5.5

Correlation matrix between selected sleep variables and O_2 consumption (mls/min.) values at specific times.
(n = 9)

	RMR1530	RMR2000	RMR2230	SOL	SWSL	Stage3	Stage4	SWS
RMR1530	1							
RMR2000	.90***	1						
RMR2230	.92***	.93***	1					
SOL	.26	.47**	.39**	1				
SWSL	.17	.41**	.30**	.79***	1			
Stage3	.08	.03	-.02	-.15	-.24	1		
Stage4	.09	-.15	-.06	-.28	-.50***	.22	1	
SWS	.11	-.11	-.05	-.29	-.51***	.59***	.92***	1

Significance levels are indicated by the following* p<.05, ** p<.01, *** p<.001.

Abbreviations: RMR1530= RMR at 1530 hours; RMR2000= RMR at 2000 hours; RMR2230= RMR at 2230 hours. Sleep variables are defined in other tables described in text.

Table 5.6

Mean time for selected sleep variables during the first cycle of sleep in the control and the three experimental conditions (n = 9)

SLEEP VARIABLES	MEAN TIMES (min) (SDs in Parentheses) CN	LAM	LA	LE
SWS	51.6 (21.1)	39.8 (16.3)	46.2 (21.1)	36.3 (19.7)
Stage 3	16.1 (8.5)	15.6 (8.3)	13.7 (6.4)	13.2 (9.4)
Stage 4	35.4 (18.5)	24.2 (12.5)	32.4 (18.1)	23.1 (14.9)
REM	2.8 (5.7)	1.7 (3.6)	1.3 (3.3)	1.8 (3.8)
TIB	157.8 (2.8)	158.5 (3.7)	157.2 (3.9)	152.1 (12.7)
TST	136.9 (25.4)	131.2 (25.0)	126.9 (27.2)	131.4 (21.3)
Awk before sleep	5.7 (6.1)	16.0 (22.4)	14.8 (11.5)	9.2 (5.7)
Stage 2	66.3 (15.6)	68.5 (17.3)	61.2 (21.5)	74.4 (8.5)
SOL	9.7 (7.9)	20.9 (24.8)	20.6 (13.4)	14.3 (6.5)
SWSL	20.2 (8.1)	34.7 (23.8)	33.0 (14.6)	34.2 (16.7)
Stage 1	15.3 (4.8)	20.9 (11.9)	17.7 (5.1)	18.6 (6.7)
MTST1wk	37.0 (27.3)	51.0 (30.4)	48.3 (28.5)	39.5 (19.4)
TTA (1st cycle)	15.1 (21.9)	11.2 (13.5)	15.5 (21.5)	11.5 (19.1)
TTA in bed	20.8 (23.4)	27.2 (23.5)	30.3 (25.1)	20.7 (17.6)

Abbreviations: **TIB** = time in bed; **TST** = total sleep time; **SOL** = sleep onset latency; **TTA** = total time awake; **MT** = movement time; **REM** = Rapid eye movement; **SWS** = slow wave sleep; **SWSL** = slow wave sleep latency.

Note. The sleep data from subject D.C. was excluded from the analysis due to extreme mask discomfort and interference.

DISCUSSION

Manipulation of the independent variable (i.e., the 19 km run) failed to produce the necessary conditions to test the direct relationship between sleep period MR and SWS, since a sustained elevation of MR into the nocturnal sleep period was unable to be obtained. Any systematic relationship between sleep period energy expenditure and SWS within athletes thus could not be assessed effectively in this study. Neither of the afternoon exercise conditions showed significant differences in MR measurements from that of the control measures at the time of retirement. Furthermore, the LE condition did not sustain increased MR into the sleep period. That is, even though O_2 consumption was significantly increased immediately following exercise and this elevation was significantly sustained into the early part of the evening, it returned to basal levels by late evening or prior to lights out. In addition, neither a sleep-facilitation nor a sleep disturbance effect within the first sleep cycle was found following the exercise sessions. Limited evidence from the correlation analyses between selected sleep variables and O_2 consumption at specific times however, indicated that increased wake-time O_2 consumption was associated with increased sleep onset and SWS latencies. This finding would in part, contradict the theories of compensation and restoration. Since the evidence was limited however, such a relationship remains open to speculation.

Failure to find increased SWS levels in the endurance athletes following experimental sessions containing exercise, was surprising given the results from other studies investigating the effects of similar exercise intensities upon sleep. As mentioned previously, significantly more of these studies confirmed a SWS effect following intense exercise than not. The finding that exercise very clearly did not increase such sleep variables as SWS in the present study may therefore question the validity of the theories of compensation. This result supports a number of other studies that have failed to show exercise-related SWS effects. For example, Kupfer et al. (1985) reported an unexpectedly greater automated delta wave count for the

second NREM/REM sleep cycle following their no-exercise condition compared to their double-exercise condition which was designed to increase exercise load by doubling the distance of each subjects' regular training run while maintaining a normal pace. One possible explanation for such a paradoxical finding was that the double-exercise condition acted as an emotional stressor by suppressing SWS increases in the trained subjects (Baekland & Lasky, 1966). However, minimal clinical or polygraphic support was present from their study to substantiate a stress effect interpretation to the decreased amount of delta activity in the second NREM\REM sleep cycle following the double-exercise condition when compared to the exercise condition. For example, no other sleep architectural changes and continuity (e.g., increased sleep latency, decreased time spent asleep, decreased sleep efficiency) were found in the trained subjects following the double-exercise condition. Nevertheless, the possibility of a stress effect existing in the present study remains open for consideration.

An alternative explanation for the unchanged SWS levels found in the present study is that O_2 consumption may have been insufficiently elevated into the sleep period in order to facilitate the expected sleep architectural changes. This view assumes the theories of restoration and conservation to be correct so that heightened metabolic activity needs to be present during initial stages of the sleep period in order to act as a precursor to SWS onset.

The use of a facial mask during the sleep period may have further inhibited the production of nocturnal levels of stages 3 and 4 in this study. This is particularly feasible given the findings of White et al. (1985), and Ryan et al. (1989) who found that the level of stages 3 and 4 and REM sleep in proportion to TST were reduced as a result of wearing either a facial mask or ventilated hood during the course of MR measurement. These studies also reported a higher proportion of stage 2 emerging as a possible replacement of the deeper stage 3 and 4 levels. Such devices used in ventilatory measurement also have been shown to alter respiration (Askanazi, Silverberg, Foster, Hyman, Milic-Emili & Kinney, 1980; Gilbert, Auchincloss, Brodsky &

Boden, 1972; Hirsch & Bishop, 1982). Alternatively, it is suggested that since sleep architecture during the second sleep cycle was not recorded in this study, it was possible that sleep effects may have been delayed until this time due to sleep disturbance associated with the wearing of the anaesthetic mask. Any sleep effects that may have been likely to occur may therefore have gone undetected. The relationship between wake-time MR and sleep architecture thus remains unclear and inconclusive. Furthermore, using computerised analyses of the delta wave activity rather than purely assessing sleep architectural changes as a function of exercise, on the basis gross procedures such as the manual scoring by 60-second epochs, may have provided a more effective measure of discerning finer changes in SWS activity. Such techniques however, were unavailable during the course of this study, and thus the possibility of finer changes occurring in SWS as a result of exercise is open to speculation.

Another plausible explanation for the current findings is that T_b may not have been elevated to a sufficiently high level during the exercise session to elicit sleep architectural changes (Horne, 1981). This view is particularly viable since the current study was performed predominantly within the winter months. It is therefore likely that while MR was elevated immediately following exercise sessions, increases in T_b on the other hand may have been counteracted by the effects of cold T_as. The rate of energy expenditure during exercise sessions also may not have been high enough to elicit critical T_b elevations (Horne, 1981), and thus SWS effects. Since T_b was not measured in this study however, due to the unavailability of equipment suitable for accurate assessment of such indices, its status remains unknown. The aim of this study however was to assess MR and sleep, and not T_b. The evidence showed that MR was elevated and thus not affected, or perhaps not as much, as what T_b may have been by the colder T_as.

In summary, the results showed that increased wake-period energy expenditure does not clearly affect sleep architecture in the first cycle if MR levels return to control levels by the time of sleep

onset. There was only minimal evidence to suggest a positive relationship between wake-time MR and such sleep variables as sleep onset and SWS latencies, a finding inconsistent with the compensatory theories. In addition, there were only transient elevations of wake-period MR following exercise so that the direct effect of increased sleep-period MR on sleep architecture, especially in terms of SWS structure, could not be ascertained. Such transient elevations in MR following high intensity exercise contrasts with those studies showing sustained MR increases (Simonson, 1971; Wilmore, 1977). This discrepancy may have been attributed to differences in the degree of subjects fitness levels, where the current study used endurance athletes. It was more likely that MR returned more rapidly to baseline levels in these athletes as a function of their more highly trained state (deVries, 1980). It is therefore recommended that prescribed exercise loads according to individual subjects' maximum capacities necessary to facilitate maximal O_2 changes in all subjects as a result of the exercise session, be implemented in future studies.

Finally, the findings from this study can be added to the growing body of work that has failed to support the SWS-exercise hypothesis (Kupfer et al., 1985) and as such places the theories of compensation under some scrutiny. It is possible that the positive SWS effects following exercise that have been previously reported are due to the influences of other factors such as temperature. The ability for homeotherms to effectively regulate T_b regardless of large changes in metabolic heat production and exchange of thermal energy between the body and the environment, has been the focus of much research. The next chapter reports a study investigating the direct effect of T_b on sleep architecture.

CHAPTER 6

EXPERIMENT TWO:

THE EFFECT OF AFTERNOON BODY HEATING ON BODY TEMPERATURE AND SLOW WAVE SLEEP

CHAPTER 6

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THE EFFECT OF AFTERNOON BODY HEATING ON BODY TEMPERATURE AND SLOW WAVE SLEEP

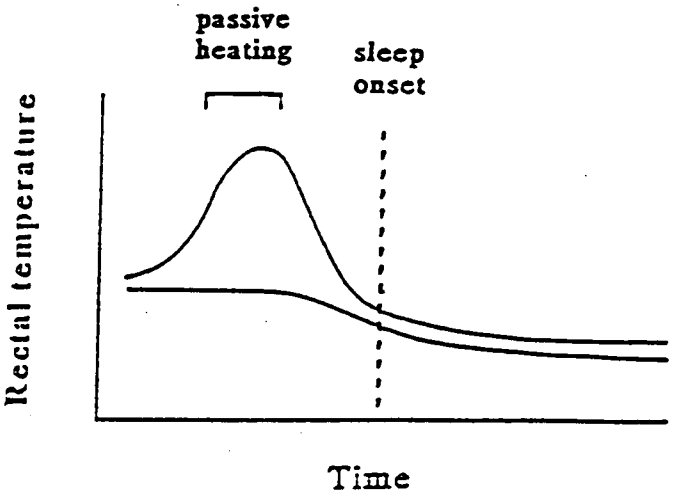
There has been considerable interest in recent years in the relationship between T_b and sleep. A reduction in T_b occurs over the first one to two hours of sleep (Day, 1941; Geschichter et al., 1966; Gillberg & Akerstedt, 1982) and is largely independent of the circadian phase of sleep onset (Gillberg & Akerstedt, 1982). Falls in T_b are related to a regulated reduction in the hypothalamic set point for thermoregulatory control (Parmeggiani, 1980, 1987, 1990) where T_b has been suggested to affect sleep. In particular, sleep duration and the distribution of REM sleep are influenced by the circadian temperature rhythm (CTR) (Czeisler et al., 1980; Gillberg & Akerstedt, 1982; Zulley et al., 1981). It also has been suggested that T_b affects the amount of SWS.

There have been two hypotheses regarding the relationship between T_b and the level of SWS (See Figure 6.1). One proposes that the regulated reduction of T_b at sleep onset is a necessary prerequisite for the occurrence of SWS. Alternatively, the amount of SWS on any particular night will be a positive function of the rate of fall of T_b following sleep onset (Sewitch, 1987). The other hypothesis is that the amount of SWS is a positive function of T_b at, and shortly after, sleep onset (Berger et al., 1988; Berger & Phillips, 1988a,b). The observation that passive heating during wakefulness increases the amount of SWS in the subsequent sleep period (Bunnell et al., 1988; Bunnell & Horvath, 1985; Horne & Moore, 1985; Horne & Reid, 1985; Horne & Shackell, 1987; Horne & Staff, 1983) is quoted by both authors as evidence for their respective positions (Berger & Phillips, 1988a,b; Sewitch, 1987). However, in view of their different hypotheses about the relationship between T_b and SWS, the two theories necessarily predict a different relationship between passive

BERGER et al. (1980)

Hypothesis:

"The amount of SWS is a positive function of T_b at and shortly after sleep onset."



SEWITCH (1987)

Hypothesis:

"The regulated reduction in T_b at sleep onset promotes SWS onset."

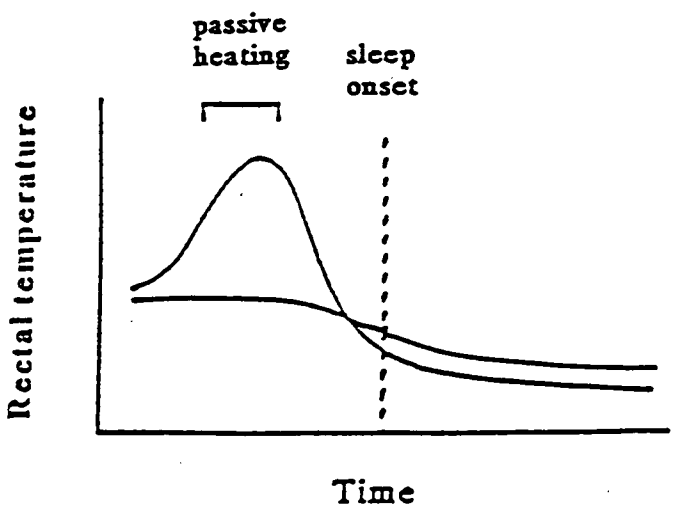


Figure 6.1: *Hypotheses of Sewitch (1987) and Berger et al. (1980)*

heating and T_b. Sewitch (1987) thus argues that passive heating results in a faster decline in T_b following sleep onset and a lower minimum temperature in comparison with a control condition. This effect is viewed as critical in the facilitation of increased SWS levels after passive heating. In contrast, Berger and Phillips (1988a,b) require that passive heating produce elevated T_bs during the early sleep period. The latter hypothesis would be consistent with findings from exercise studies conducted in warmer climates (e.g., South Africa) where the level of exercise was such that T_b would most likely have remained elevated during the night (Shapiro & Verschoor, 1979; Shapiro et al., 1981). Indeed, elevated T_bs have been shown to persist throughout the night following extreme exercise (Montgomery et al., 1985).

The issue is unresolved because, with one exception, studies investigating the passive heating effect have not measured T_b beyond the passive heating period. Horne and Staff (1983) measured the effect of passive heating on T_b during the subsequent sleep period in a single subject with one night's recording for each condition. They found a compensatory fall during sleep, as predicted by Sewitch (1987). Berger and Phillips (1988b) however, have reported a positive relationship between both T_{re} and tympanic (T_{ty}) temperature and the amount of SWS (Berger et al., 1988) and an increase in SWS as a result of increasing T_{ty} by facial heating (Berger & Phillips, 1988b; Moriarty, Phillips & Berger, 1988).

The aim of the present study was to examine the two hypotheses of Berger and Phillips (1988a,b) and Sewitch (1987) by measuring the effect of passive heating on T_{re} and SWS during the subsequent sleep period.

METHOD

Subjects and Design

Five male subjects with a mean age of 20.4 years (SD= 1.52) participated in the study. All were healthy at the time of testing, they did not report a history of respiratory or sleep disorders, were non-smokers and were not receiving medications. Fitness was not an issue as the passive heating effect has been previously reported in both fit (Horne & Staff, 1983) and unfit (Horne & Reid, 1985) subjects. The project was approved by the University of Tasmania, Human Ethics Committee and all subjects completed a consent form.

The design of the study involved two conditions. In the first, subjects were exposed to passive heating (PH) in the late afternoon, while the second was a non bath control condition (CC). A temperate bath control procedure was not considered necessary because differences between this and a non-bath control have not been found (Bunnell & Horvath, 1985). Each subject participated in the two conditions on three separate occasions. The presentation for conditions was CC, PH, PH, CC, CC, PH for three subjects and PH, CC, CC, PH, PH, CC for two subjects. The experimental strategy of running a relatively small number of subjects on several nights in each condition (rather than a larger number of subjects on a single night each) was selected in view of the importance of having subjects well adapted to the unusual sleeping circumstances. From a statistical perspective the strategy was designed to reduce the within subject error variance component. In the statistical analysis, data was thus averaged over within subject replications.

In order to further facilitate subjects familiarisation with the equipment, the six experimental sessions were preceded by two adaptation nights and a number of afternoon adaptation sessions. During the latter sessions subjects accustomed themselves to wearing the various pieces of equipment, particularly the face mask. The experimental sessions were over non-consecutive days.

Procedures

Physiological Measurements

The physiological variables measured were T_b, O_2 consumption, EEG, EOG, and EMG recordings.

Body Temperature

In this study, T_{re} was employed as a measure of core T_b. It was considered an appropriate technique to minimise T_a fluctuations and for simultaneously recording physiological measurements. The fact that MR was monitored using a head mask precipitated a need for a temperature measurement that could be taken from a non-intrusive, painless site. A general purpose Yellow Springs, YSI Series, 401 Temperature probe with a diameter of 4 mm, a width of 5 mm and a thermistor length of 8 mm was inserted 10 cms into the rectum and maintained in place by surgical tape. The system was calibrated against known resistances and temperatures to an accuracy of .01 °C at the beginning of each recording session. The time constant of this series of rectal thermometer was 7.0 seconds. The probe was connected directly to the data recording system via a cable long enough to allow the subject freedom to move around the laboratory. Temperature was measured continuously during the entire experimental sessions where the temperature signal was amplified using the Beckman R411 Dynograph recorder and then input to a D.E.C. PDP-11/23 computer via an eight-bit analogue to digital converter and sampled at 1-minute intervals. The collected data was stored on a thirty megabyte Winchester hard disk. The sampling rate was 1 Hz for the temperature variable. Software running under RT-11 Pascal was developed within the laboratory to enable the online display, via a high resolution graphics screen of data entering the computer. This continuous display of temperature data allowed sudden aberrant values such as in the form of a dramatic drop in temperature to be detected immediately during which the subject was asked to check the positioning of the rectal thermometer. A computer alarm system was installed within the temperature data

collection programme which was designed to alert the experimenter to possible artifacts in data values. This highlighted the importance of ensuring that the positioning in terms of the depth of the rectal probe remained constant, since changes in positioning along the rectum leads to slight changes in the absolute values of recorded temperature.

The available programme included the facility of averaging temperature data points that were collected once a second. For the purposes of this experiment the T_{re} was averaged across 15 minute intervals.

Oxygen Consumption

The indirect calorimetric method of measuring O_2 consumption was used to assess overall MR. The procedures and equipment used were the same as that used in Experiment One (i.e., expired air was collected by a free mask attached to a two-way breathing valve (Hans Rudolph #2600). The valve deadspace was 53 cc with the total deadspace of the mask and valve varying from 127-135 cc depending on facial configurations. Ventilation was calculated by the computed integration of air flow as measured by a Fleisch Pneumotachograph and Gould PM 15E Pressure Transducer. The O_2 level in the expired air was measured by an Applied Electrochemistry S-3A Oxygen Analyser. Signals were fed via a Beckman R411 Recorder to the computer for breath by breath computation of O_2 consumption. Greater details of the laboratory procedures for measuring O_2 consumption are given in Experiment One and are documented in Colrain et al. (1987). Due to technical problems, the O_2 consumption data for one subject were incomplete and the subject was dropped from the analysis of this variable.

Sleep Recordings

The identification of the stages of sleep was achieved by simultaneous recordings of EEG (C3/A2), EMG and EOG signals as in Experiment One, where collection and scoring complied to

standardised procedures (Rechtschaffen & Kales, 1968). Each night's sleep record was scored in 30 second epochs by two blind scorers and disagreements were resolved by discussion (the initial level of agreement was 91% of all epochs). As noted later in this section, lights out was at 2300 hours and recording was ended at 0700 hours.

During the 30 minute waking resting MR session recorded before lights out on all experimental sessions, an EEG recording was monitored to ensure that the subject did not enter stage 1 sleep. This stage of sleep is associated with marked reduction in O_2 consumption (Fraser et al., 1989). Since there has been some evidence suggesting that audio-visual stimulation can elevate MR to a certain degree (Shea, Walter, Pelley, Murphy & Guz, 1987), the use of these devices to assist in the maintenance of the wake state was excluded during resting MR recording in this study.

Experimental Routines

Subjects reported to the laboratory at 1330 hours. Rectal temperature recordings began at 1400 hours and, with the exception of the time the subjects spent in the bath in the passive heating condition and two other brief periods in both conditions, continued until the end of the sleep period at approximately 0700 hours the next morning. Passive heating was administered in two 40 minute blocks with a 25 minute inter-block interval beginning at 1715 hours and ending at 1900 hours. In addition to T_{re}, O_2 consumption was measured at various times during the afternoon and evening. In the passive heating condition measurements were taken for 15 minutes before the passive heating, 20 minutes between the two heating sessions, 20 minutes following the second heating session and 30 minutes before lights out. Oxygen consumption measurements were then continued throughout the night. In the control condition measurements were taken during the 30 minutes before sleep onset and then throughout the sleep period. Some O_2 consumption measurement periods were less than the specified duration because

of the need to maintain a strict time schedule throughout the sessions.

In addition to the periods in the bath, T_{re} recordings were interrupted on two other occasions. The first was during the evening meal when some subjects left the laboratory. The second was immediately before the pre-sleep O_2 consumption measurements and was necessary to begin a new data acquisition file incorporating the O_2 consumption measurements. Also on some nights the probe was temporarily removed at this time. Thirty minutes of data were lost in association with the evening meal and 15 minutes before the pre-sleep O_2 consumption measurement.

Sleep was assessed during the night in each condition. Subjects ate their evening meal between 1530 and 1630 hours, while lights out was at 2300 hours. The timetable of events for the laboratory sessions is shown in Table 6.1.

During the afternoon and evening subjects were confined to the laboratory and were free to engage in a range of sedentary activities. Tea and coffee were not permitted and no food or drink other than water could be consumed following the end of the evening meal at 1630 hours. Caloric intake at the evening meal was held constant at a level of each subject's choosing.

The passive heating procedures were essentially the same as those developed by Horne and Staff (1983). Subjects sat on a chair in the bath with the water level at neck height. Water temperature was maintained between 42 °C and 43 °C by a thermostatically controlled valve in the attached hot water system. Subjects remained in the bath for two 40-minute sessions. Although they were told that they could leave the bath at any sign of ill effects, all subjects adapted well to the procedures and this precaution was not utilised. The average elevation in T_{re} from the last 15 minutes before heating to the first minute following the second passive heating session was 1.64 °C (SD = 0.36).

Table 6.1

Diary of events during each experimental session

TIME		EVENT
Start	Finish	
1330		Arrive at laboratory
1400		Begin T_b recording
1530	1630	Evening meal
1645*	1700	O_2 consumption recording
1715*	1755	Passive heating 1
1800*	1820	O_2 consumption recording
1820*	1900	Passive heating 2
1910*	1930	O_2 consumption recording
2230	2300	O_2 consumption recording
2300	0700	Sleep period

* Passive heating sessions only.

Note. The unaccounted time was spent in sedentary activities within the laboratory.

During the pre-sleep sessions O_2 consumption was measured with the subject lying on, or in, a bed in the supine position with eyes open and the room light on. Subjects also were instructed to remain in the supine position during the night. This was achievable because the mask made it difficult to sleep in any other position. The laboratory was maintained within a temperature range of 23 °C to 25 °C and subjects could adjust their clothing or bedcovers to maintain thermal comfort.

RESULTS

The data was first averaged over replications within conditions and subjects. The resulting values were used in all subsequent analyses, tables and figures. Replications were not formally analysed as their inclusion was designed to reduce error due to within subject variability, rather than to be of specific interest. Further, preliminary data analyses had indicated that, although within a subject there was an expected level of variability in both initial temperature and sleep architecture, from replication to replication, the experimental manipulation had highly consistent effects over replications on both variables.

The effect of passive heating on T_{rc} is illustrated in Figure 6.2. As can be seen, passive heating caused a substantial elevation in T_{re}, an effect which persisted throughout the sleep period. In addition to the effect of passive heating, there was a reduction in T_{re} associated with sleep. The fall was first apparent following the non-recording period associated with the setting up of new data files and continued during the first 1 to 1 1/2 hours of sleep. The marked fall observed following the non-recording period was most likely associated with the attainment of a lateral position (Kleitman, 1963). There was no perceptible change in the rate of fall of T_{re} immediately following sleep onset. The rates for the intervals immediately before lights out, between lights out and sleep onset, and sleep onset to SWS onset were .0090, .0069, .0060 and .0103, .0078, .0069 °C/min. for the control and passive heating conditions respectively. Tests of significance showed

the rate of fall over the interval, sleep onset to the onset of SWS, did not differ from either of the other two intervals immediately before lights out, and between lights out and sleep onset (the *t* values for the before lights out comparison were $t(4) = 1.58$, $p > .05$; and $t(4) = 1.48$, $p > .05$ for the control and passive heating respectively, and for the lights out to sleep onset comparison were $t(4) = 0.43$, $p > .05$; and $t(4) = 0.82$, $p > .05$ for the control and passive heating respectively).

The mean temperature values for critical periods over the experimental sessions are shown in Table 6.2. These periods were the 15 minutes before passive heating, the 15 minutes following passive heating, the 5 minutes before lights out and following sleep onset, and minutes 61-65 following the onset of SWS. The analysis of these values was carried out using three separate ANOVAs. The first variable in each of these analyses was the experimental condition (Passive Heating or Control). The other variable was Time, the levels of which were: before versus after passive heating in the first analysis; after passive heating versus before lights out in the second; and before lights out, following sleep onset, before SWS and 60 minutes after SWS onset in the third. Significance levels for the effect of time were assessed using $1/n$-1 degrees of freedom, the most conservative approach to dealing with the problem of sphericity.

The first analysis indicated a significant increase in T_{re} as a result of passive heating ($F(1,4) = 96.96$, $p < .01$, for the Condition by Time interaction for the periods before and after passive heating); the second, a significantly greater reduction in T_{re} in the passive heating condition during the 4 hours following passive heating ($F(1,4) = 49.28$, $p < .01$, for the Condition by Time interaction for the periods after passive heating and before lights out); and the third, a significant fall in T_{re} in association with sleep ($F(1,4) = 96.42$, $p < .01$, for the main effect of time from lights out to 60 minutes following SWS onset); and a significantly higher T_{re} in the passive heating condition ($F(1,4) = 62.49$, $p < .01$, for the main effect of heating). The Condition by Time interaction for the period from lights out to 60

minutes following the onset of SWS was not significant [$F(1,4) = 1.50$, $p > .05$]. ANOVA summary tables are presented in Appendix C.

In addition to the changes in temperature that were of interest to the hypotheses under consideration, there was a small and brief increase in T_{re} following the evening meal. The increase was most likely due to a thermogenic effect of the meal, although because subjects had to leave the laboratory area to obtain their meal, there also was a small increase in activity at this time.

Passive heating was not associated with a significant increase in O_2 consumption (see Figure 6.3). Of the 4 subjects for whom O_2 consumption data was available, 2 increased, and 2 decreased O_2 consumption levels following passive heating. The mean change was an increase of 14 ml/min., from 311 ml/min. to 325 ml/min., which was not a significant difference [$t(3) = 1.50$, $p > .05$]. Further, there was no difference in O_2 consumption between the two conditions during measurements before lights out (255 ml/min. & 261 ml/min. for the control and passive heating conditions, respectively), or for the 15 minutes immediately following stage 1 sleep onset (227 ml/min. & 227 ml/min.). An analysis using a 2 x 2 ANOVA with repeated measures on both variables indicated that although neither the main effect of condition [$F(1,3) = 0.09$, $p > .05$], nor the interaction effect [$F(1,3) = 0.55$, $p > .05$] were significant, the effect of sleep onset was significant [$F(1,3) = 138.34$, $p < .01$]. ANOVA summary tables are presented in Appendix C.

Inspection of Table 6.3 indicates that, despite considerable adaptation to the laboratory, subjects' sleep was disturbed by the experimental conditions. The amount of disturbed sleep was higher and SWS and REM sleep lower than is usually found in this laboratory (e.g., Trinder et al., 1985). However, the levels were consistent with studies, from this and other laboratories, in which respiratory variables were measured (e.g., White et al., 1985).

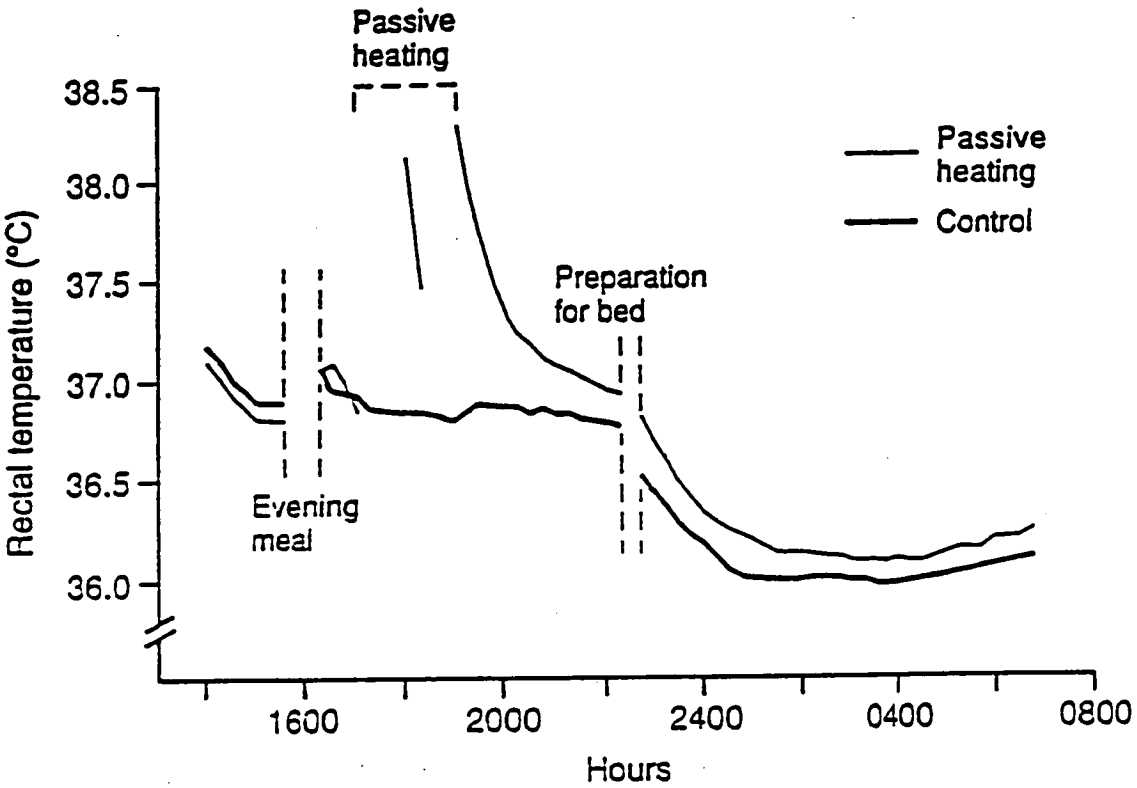


Figure 6.2: *Rectal temperature as a function of experimental condition over the recording period. Data points were mean values averaged over 15 minutes, the three replications within each condition, and the five subjects. Temperatures were not available from all subjects during the evening meal and thus data from 1545 to 1645 hours have not been presented. Data collection also was discontinued during passive heating and for a 15 minute interval before the evening O_2 consumption measurement. Lights out was at 2300 hours.*

Table 6.2

Mean rectal temperatures for selected time intervals during the two experimental conditions

TIME	MEAN TEMPERATURES (°C) (SDs in Parentheses) PASSIVE HEAT	CONTROL	
1645-1700 before heating	36.86 (.11)	36.91 (.09)	C,T,CxT
1845-1900 after heating	38.28 (.35)	36.81 (.09)	C,T,CxT
5 min before lights out	36.76 (.14)	36.57 (.14)	
5 min after sleep onset	36.63 (.12)	36.45 (.13)	C,T
5 min before SWS	36.58 (.13)	36.39 (.15)	
5 min after 60 min of SWS	36.28 (.10)	36.14 (.17)	

Note. The statistical analyses are described in the text. Significant comparisons ($p < .01$) are shown on the right of the table.
C = Conditions, T = Time, CxT = Interaction.

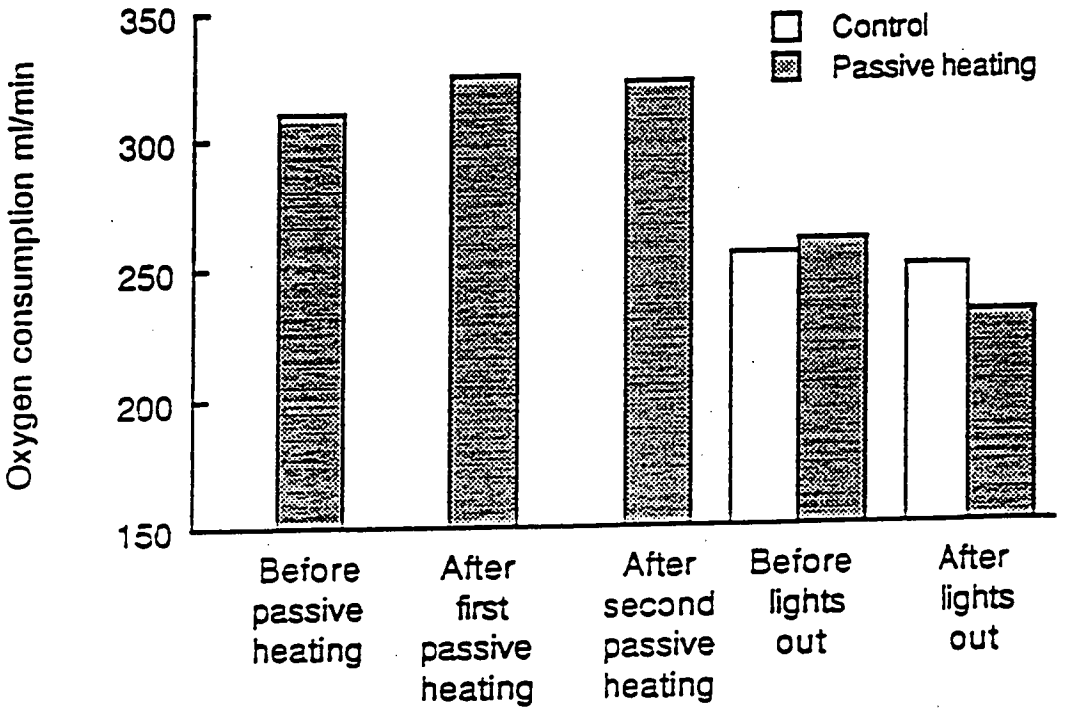


Figure 6.3: *Mean O_2 consumption before and after passive heating sessions, and before and after lights out compared to the control condition.*

Table 6.3

Mean time for selected sleep variables in the passive heating and control conditions

	MEAN TIMES (min) (SDs in Parentheses)	
SLEEP VARIABLES	**PASSIVE HEAT**	**CONTROL**
SWS (150min)	43 (12.9)	35 (10.9) *
Stage 3	17 (2.7)	15 (5.5)
Stage 4	26 (12.1)	20 (13.4)
SWS (total night)	65 (25.5)	57 (21.5)
Stage 3	30 (9.9)	27 (12.1)
Stage 4	35 (19.1)	30 (22.7)
REM (150min)	14 (8.4)	8 (8.1) *
REM (total night)	86 (15.4)	79 (19.6)
REM Latency[a]	101 (27.8)	154 (99.6)
TST	446 (24.3)	441 (29.6)
Stage 2	256 (12.0)	250 (19.8)
Sleep Latency[b]	10 (4.1)	10 (4.5)
Disturbed Sleep (1+Wake)	73 (33.3)	95 (39.4) *
Stage 1	40 (13.9)	53 (23.4)
Time Awake	33 (21.5)	42 (25.5)
Sleep Efficiency[c]	.93 (0.04)	.91 (0.06)

* Conditions significantly different at $p < .05$.

[a] Time from the first stage 2 to the first REM epoch.

[b] Time from lights out to the first stage 2 epoch.

[c] Ratio of total sleep time to time in bed.

Passive heating resulted in an increase in SWS during the early part of the night. Thus, SWS was significantly higher following passive heating in the first 150 minutes of sleep [$t(4) = 3.42$, $p<.05$]. (The first 150 minutes of sleep is the interval used in this laboratory to assess early night effects). The increase in SWS was largely the consequence of a change in stage 4 [$t(4) = 2.74$, $.05<p<.10$] with a smaller increase in stage 3 [$t(4) = 1.26$, $p>.05$]. The magnitude of the effect on SWS was small, though consistent over subjects. While the amounts of SWS and of stages 3 and 4 for the whole night were each higher following passive heating, the differences were not significant [$t(4) = 1.54$, $p>.05$; $t(4) = 0.71$, $p>.05$; $t(4) = 1.26$, $p>.05$], for SWS and stages 3 and 4, respectively.

REM sleep was higher during the first 150 minutes of sleep following passive heating [$t(4) = 3.36$, $p<.05$]. Also REM latency was reduced and total REM increased, though neither significantly [$t(4) = 1.53$, $p>.05$; $t(4) = 1.29$, $p>.05$], for latency and total amount, respectively. The pattern of results for sleep indicates that the changes in SWS in the first 150 minutes were not due to a suppression of REM sleep. On the contrary, both REM and SWS increased in this period.

Sleep duration, and sleep onset latency were unaffected by the experimental manipulation [$t(4) = 0.80$, $p>.05$; $t(4)= 1.05$, $p>.05$, respectively]. However, there was some evidence that sleep was less disturbed following passive heating. The amount of disturbed sleep (stage 1, MT and Time awake) was reduced [$t(4) = 3.07$, $p<.05$] and sleep efficiency was increased, although the latter difference was not significant [$t(4) = 1.16$, $p>.05$].

The correlation matrix of absolute T_b at specific times (i.e., lights out, sleep onset, SWS onset) and selected sleep variables presented in Table 6.4 consisted of the values for each subject on each separate night. Data was not averaged across night or condition. The correlation analysis between the sleep variables and absolute T_bs at specific times revealed very highly significant positive correlations between all temperatures, which in turn were highly correlated with

the average T_b across the night. The rates of T_b fall at particular times were neither significantly correlated with any of the absolute T_bs at the selected times, nor with any of the sleep variables. The sleep variables that were highly correlated with absolute T_bs at the specific times included SWS, particularly the component Stage 4, during the first sleep cycle, and across the entire sleep period (see Table 6.4). As a result of such high correlations between sleep variables and temperatures at all time points, stepwise multiple regressions could not be informatively used to predict those temperature changes that would have the greatest effect on SWS increases.

DISCUSSION

The effect of passive heating on SWS was similar, though smaller in magnitude, to that which has previously been reported (e.g., Horne & Staff, 1983). The effect was observed despite the sleep disturbance usually associated with wearing a face mask and sleeping supine. It might be argued however, that the difference in SWS between the two conditions was an artifact of the recording environment; perhaps because of a suppression of the control values. This is considered unlikely, because there appears to be no reason why the recording situation would have differentially disadvantaged either of the two conditions and because the sleep values obtained are consistent with other studies where the subjects were restricted by respiratory apparatus. If anything, the sleep disturbance associated with the recording conditions might be expected to minimise the effect of the independent variable. The present results therefore testify to the robustness of the effect. Most critically, the observation of an increase in SWS, allowed the temperature conditions which produced the effect to be assessed.

The temperature data support the relationship between T_b and SWS proposed by Berger (Berger et al., 1988; Berger & Phillips, 1988a,b). Rectal temperature was significantly elevated at sleep onset and at the beginning of SWS in the passive heating condition.

Table 6.4

Correlation matrix between body temperature at selected times, MR and sleep variables

	LOT_b	$SOLT_b$	$SWSLT_b$	$AvNightT_b$	Before LO rateT_b	Between LO-SOL rateT_b	Between SOL-SWSL rateT_b	Stage3	Stage4	SWS	1st S3	1st S4	1st SWS
LOT_b	1												
$SOLT_b$	.90***	1											
$SWSLT_b$	.81***	.92***	1										
$AvNightT_b$	.70***	.76***	.72***	1									
Before LO rateT_b	.25	.15	.14	.51**	1								
Between LO-SOL rateT_b	.13	-.13	-.23	-.19	.19	1							
Between SOL-SWSL rateT_b	-.01	-.14	-.31	-.11	-.02	.43**	1						
Stage3	.12	-.22	.27	.29	.31	-.05	-.03	1					
Stage4	.61***	.43**	.41*	.36*	-.05	.07	.06	-.05	1				
SWS	.59***	.49**	.50**	.47**	.13	.08	.04	.52**	.83***	1			
1st S3	.00	.10	.08	.12	-.01	.08	.18	.56***	-.22	.13	1		
1st S4	.64***	.46**	.43**	.50**	.06	.07	.06	-.04	.88***	.73***	-.07	1	
1st SWS	.61***	.46**	.44**	.50**	.01	.11	.10	.24	.71***	.74***	.28	.84***	1

Significance levels are indicated by the following * p<.05, ** p<.01, *** p<.001

Abbreviations: LOT_b= T_b at lights out; $SOLT_b$= T_b at sleep onset; $SWSLT_b$= T_b at SWS onset; $AvNightT_b$= T_b averaged across the sleep period; Before$LOrateT_b$= Rate of T_b fall before lights out; Between LO-SOL$rateT_b$= Rate of T_b fall between lights out and sleep onset; Between SOL-SWSLrate T_b= Rate of T_b fall between sleep onset and SWS onset; 1stST3, 1st ST4, 1stSWS= Stage 3, Stage 4, and SWS during the first sleep cycle.

Indeed, after an initial fall following the termination of passive heating, T_{re} remained approximately 0.19 °C above the control condition. There was no evidence to support Sewitch's (1987) hypothesis that the elevation of SWS following passive heating is due to a faster rate of decline in T_b after sleep onset, as the rates of decline were essentially identical in the two conditions. Nor, as has been described, was there any indication of a compensatory fall in T_b.

The present data is not however entirely consistent with results from Berger's laboratory. Berger et al. (1988) reported that the rate of fall in both T_{re} and T_{ty} to the end of the end of the last SWS period, was a positive function of the temperature at sleep onset, whereas in this study these two variables were unrelated. It is likely that this discrepancy is a consequence of procedural differences between the two studies. Subjects in the Berger et al. (1988) experiment were exposed to thermoneutral or cold T_as during sleep, whereas in this study subjects were exposed to thermoneutral, or hot temperatures before sleep, and thermoneutral conditions during sleep. Despite this discrepancy, neither study reported a relationship between the rate of fall in temperature at sleep onset and the amount of SWS. The studies are therefore consistent in showing that T_b is the critical factor determining SWS. Since the correlation analyses however, revealed high correlations between temperature at different points and sleep variables it could not be distinguished from the data whether it was T_b during the passive heating, or at sleep onset, which was the critical variable in determining the SWS effect. Furthermore, it was not the intent of this study to distinguish between these two hypotheses. Nevertheless, the data demonstrated that the rate of fall is not the relevant variable and that, in contradiction of Horne, T_b is elevated at sleep onset following the passive heating procedure. Observation of the data from this study in respect to Berger's procedure suggests that temperature at sleep onset is the critical factor.

It is of interest to speculate as to what factors may have sustained the elevation in T_b throughout the sleep period. One

possibility is that it was due to a difference in heat production. This hypothesis is however contradicted by the observation that O_2 consumption values were not different immediately before or after sleep onset. Nor is it likely that the effect was due to limitations in heat exchange, as heat loss appeared to proceed normally following the evening meal, immediately following the passive heating and during sleep. Previously it has been speculated that passive heating may produce a delayed shift in the T_b rhythm (Trinder et al., 1988). However, the data is not consistent with this hypothesis, because T_{re} remained elevated throughout the night. It remains possible that the passive heating produced a shift in T_b set point, though the evidence is more that of exclusion, rather than being direct.

The pattern of fall in T_b during sleep was as described previously (Gillberg & Akerstedt, 1982). Rectal temperature fell approximately 0.75 ^oC, asymptoting 1 to 1 1/2 hours after sleep onset in both conditions. Rectal temperature began to fall before lights out, during the measurement of resting O_2 consumption. A similar result has been reported in rats by Obal (1984). Further, the rate of fall was not affected by sleep onset. This contrasts with other reports which have linked the rapid fall in T_b during the early sleep period with sleep onset (Gillberg & Akerstedt, 1982; Parmeggiani, 1980, 1987). The most likely explanation of the present data is that the effect of sleep onset has been masked by what appears to be a relatively large effect associated with the change in body position on going to bed (Kleitman, 1963). Parmeggiani (1980) also has noted that body position may contribute to temperature fall in cats. However, the contribution of body position to the fall in T_b at sleep onset in human subjects remains uncertain.

Passive heating did not have a consistent effect on O_2 consumption. In two subjects heating was associated with moderate increases, while in two others with slight decreases. In all subjects O_2 consumption was the same for the two conditions by lights out, despite differences in T_b. The failure to find an increase in O_2 consumption in association with an increase in core temperature was

unexpected since it has been previously reported that increases in T_b are associated with increased MR as a function of the increased oxidative activity that accompanies the passive heating process (Keele et al., 1982). It is unlikely to be due to insensitivity in the recording equipment because other changes, such as the fall associated with sleep onset (Fraser et al., 1989), were identified. A more likely explanation is that the effect of the bath was masked by a thermogenic effect associated with the evening meal. In this context, the failure to measure O_2 consumption in the control condition at this time, can be seen as an error.

REM sleep was significantly higher during the first 150 minutes of sleep in the passive heating condition. The effect was largely due to a tendency for subjects to miss the first REM period in the control condition. Bunnell et al. (1988), have reported that REM was suppressed by late evening, but not morning, afternoon or early evening heating, while Horne and Reid (1984) found afternoon heating reduced REM sleep. Other studies have reported REM sleep to be unaffected by passive heating (Bunnell & Horvath, 1985; Horne & Staff, 1983). In general, the data indicate that passive heating does not have a consistent effect on REM sleep. Further, the present results show that the facilitative effects of passive heating on SWS do not depend on a reduction in REM sleep.

The results of this study, in agreement with other recent studies, show that T_b influences the SWS component of sleep. The nature of the interaction is consistent with the hypothesis that the amount of SWS is a positive function of T_b at sleep onset (Berger et al., 1988; Berger & Phillips, 1988a,b), but not with the notion that it is a function of the rate of T_b fall following sleep onset (Sewitch, 1987). An alternative view is that an homeostatic thermoregulatory reaction to changes in T_b may be primarily responsible for the changes in SWS. With this view, T_b may therefore present an index of this mechanism rather than reflecting the primary factor to determine SWS levels.

Since aerobically fit and unfit populations have different sleep architectures (Baekland & Lasky, 1966; Montgomery et al., 1987; Trinder et al., 1985), and that changes in T_b have been shown to affect SWS, it seems feasible that there could be T_b differences between the aerobically fit and unfit. The following chapter will report a study that assesses the extent to which T_b may contribute to these sleep architectural differences, and compares the status of T_b and MR, and sleep architecture in endurance athletes and sedentary individuals.

CHAPTER 7

EXPERIMENT THREE:

THE RELATIONSHIP BETWEEN BODY TEMPERATURE AND SLEEP ARCHITECTURE IN ENDURANCE ATHLETES

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The passive heating study demonstrated that T_b influences SWS. It also has been reported that physical exercise in humans results in elevated levels of SWS and longer sleep durations, both acutely on nights following daytime exercise (exercise effect), and chronically in individuals who habitually exercise (fitness effect)(Trinder et al., 1988). These observations suggest that human sleep reacts in a compensatory manner to variations in energy expenditure and catabolic activity during wakefulness; a relationship which is consistent with energy conservation and bodily restorative theories of sleep (Adam, 1980; Berger, 1984; Berger & Phillips, 1988a; Oswald, 1980).

There is however, a major difficulty with concluding, on the basis of the exercise studies, that sleep compensates for variations in waking activity. It is, that exercise only affects sleep under limited circumstances. Thus, a review of this literature found that only eight of 33 studies reported an unambiguous exercise effect on SWS and only four of approximately 26 studies reported an effect on sleep duration (Trinder et al., 1988). Further, while endurance athletes have been reported to have higher levels of SWS, shorter sleep onset latencies (SOL) and longer sleep durations (Montgomery et al., 1982; Montgomery et al., 1987; Trinder et al., 1985), these effects have not been found in athletes who engage in other forms of training, such as power training (Trinder et al., 1985). It therefore seems unlikely that the effects of exercise on sleep reflect a mechanism by which sleep architecture responds in a compensatory manner to variations in activity levels during wakefulness.

Alternatively, it has been argued that when an increase in SWS is observed following exercise, it is a consequence of body heating during the exercise (Horne, 1981). Thus, while exercise in a moderately warm environment increases subsequent SWS levels, the effect does not occur if the exercise is accompanied by facial cooling (Horne & Moore, 1985). Similarly, as was shown by the study reported in Chapter 6 and other previous studies, SWS is elevated by passive body (Bunnell et al., 1988; Bunnell & Horvath, 1985; Horne & Moore, 1985; Horne & Reid, 1985; Horne & Shackell, 1987; Horne & Staff, 1983) and facial (Berger et al., 1988) heating. The data from Experiment Two in association with other more recent data suggests that it is T_b at sleep onset and during the early sleep period which is critical in producing an increase in SWS (Berger & Phillips, 1988a,b), rather than the temperature attained during the passive heating, or exercise per se (Horne, 1981), or the rate of fall of T_b during sleep onset Sewitch (1987).

One of the most consistent findings with respect to the effects of exercise on sleep has been the chronically higher levels of SWS and longer sleep durations observed in endurance athletes. The aim of the study was to determine if these effects are due to the effect of T_b on sleep. The experiment was designed to assess the role of T_b on sleep by comparing the laboratory sleep of endurance athletes and sedentary individuals with T_b at sleep onset held constant between the two groups. The decision to hold sleep onset constant was based upon evidence from another study (Hedges, 1989:

Jordan, Montgomery, Trinder & Hedges, paper submitted for publication) that showed higher average T_res and earlier sleep onset times in athletes compared to sedentary individuals. It was therefore considered that the higher T_res reported for the athletes in this study may have been a consequence of their earlier sleep onset times. Furthermore, the athletes were run on non-exercise days to remove the possibility of exercise-induced T_b effects.

METHOD

Subjects and Design

The sleep and T_{re}s of eight endurance athletes and eight age-matched unfit sedentary males were compared. Two non-consecutive experimental sessions were run on each subject. The mean ages of the fit and unfit populations were 21.5 years (SD = 3.28) and 22.6 years (SD = 3.28) respectively. Fit subjects were selected if they engaged in regular aerobic training programmes, such as long distance running, and reached an age-adjusted VO_2max of not less than 57 ml/kg/min. The mean age-adjusted VO_2max for the fit group was 71.4 ml/kg/min. (SD = 10.37). The unfit subjects were selected if they did not participate in regular exercise and had an age-adjusted VO_2max of less than 43 ml/kg/min. The mean value was 37.1 ml/kg/min. (SD = 4.55). Sixteen subjects were then allocated to each of the two experimental groups with subjects matched for age. The mean weight and height for the fit and unfit groups was 68.6 kg (SD = 4.87) and 180.2 cm (SD = 3.45) and 65.6 kg (SD = 7.15) and 177.9 cm (SD = 4.78) respectively. All subjects were non-smokers and free from respiratory and sleep pathology.

The research project was approved by the University of Tasmania Human Ethics Committee. Informed consent was required from each subject all of whom were paid for their participation in the study.

Procedures

Each subject was run on an adaptation and two non-consecutive 24-hour experimental sessions. On each testing occasion T_{re} was measured throughout the session, beginning at approximately 0900 hours and finishing at the same time the following morning. Subjects were, with some restrictions (as indicated below), free to engage in their normal daily activities during the day and they returned to the laboratory at approximately 2100 hours. While lights out was specified as 2300 hours for both groups of subjects there was some variation in the sedentary group, with the average time of lights

out coinciding with 2323 hours. The common time of lights out was selected to be approximately that of the athletes. This procedure was followed as previous studies have shown that an advance of two hours in the time of sleep onset has little effect on SWS and sleep duration, while similar delays are associated with increases in SWS (Taub & Berger, 1973a,b; 1976). Resting O_2 consumption was measured during the half hour before lights out. Polysomnographic recordings were then collected throughout the sleep period.

Subjects usual time of sleep onset was assessed by questionnaire. The aerobic fitness of each subject was assessed by testing submaximal VO_{2max} using a bicycle ergometer (Astrand & Rodahl, 1977).

Continuous ambulatory T_{re}s were obtained from all subjects using a data logger and Yellow Springs, YSI series, 401 rectal probes. The system was calibrated against known resistances and temperatures to an accuracy of .01 °C. The data logger was the size of a personal stereo unit and was fitted with six rechargeable batteries. It was required that a minimum of two analysable periods of 24 hours be available for analysis from each subject. If due to equipment failure, probe displacement, or some other cause, less data was available, further recording sessions were conducted.

The probe was inserted 10 cm into the rectum and taped into place by the subjects, who were given detailed verbal and written instructions at the beginning of the first data collection session. Subjects were instructed to periodically check the position of the probe during the experimental sessions reducing any risk of probe dislodgement and inaccurate temperature recordings. The probe was removed during defecation. Either the subjects came to the laboratory to attach the equipment, or the initialised monitor was taken to the subjects home. Subjects were given a kit which included instructions, tape, scissors, lubricating paste and rectal probe.

Rectal temperature was collected at a sampling rate of 30 second intervals as analogue input via the data logging unit. The

logger was later connected to a 386K IBM computer for data extraction. Once transferred to an IBM computer the data was converted into "°C" and averaged over epochs varying from one minute to the total recording period.

Oxygen consumption was measured with the subject lying in a supine position with eyes open and a room light on. The laboratory was maintained within a range of 22 °C to 25 °C and subjects could adjust their clothing or bedcovers to maintain thermal comfort. Polysomnographic recordings were collected throughout the measurement period to ensure that subjects remained awake. Expired air was collected by a face mask attached to a two-way breathing valve (Hans Rudolph #2600) as described in Experiments One and Two. The mean deadspace of the anaesthetic mask and valve was 136 cc. Expiration airflow was measured by a Fleisch pneumotachograph and a Gould PM 15E pressure transducer and O_2 levels by an Amatek S-3A O_2 analyser. Airflow and O_2 values were collected on hard disk for subsequent software computation values (Digital Equipment PDP-11/23 computer). Ventilation was calculated according to the methods described in Experiments One and Two.

The sleep of subjects was assessed by EEG (C3/A2), EMG (submental) and EOG recordings using standard procedures (Rechtschaffen & Kales, 1968). Sleep records were scored using procedures described in Experiments One and Two. The initial percent agreement was above 90% for all records.

During the day subjects engaged in their normal daily routine, with the exception that they were prohibited from participating in activities, such as physical exercise, naps, and hot baths, which may have unduly influenced T_{re}. In addition, they were instructed to maintain food intake at their normal levels and to consume their evening meal between 1800 and 1900 hours. Finally, subjects were instructed not to consume alcohol, spicy foods, medication, or caffeinated beverages during experimental sessions. No additional food or drink other than the quota specified at a set time across experimental sessions was allowed.

The sleep and temperature data on one session from one subject who complained of discomfort during the night was discarded, while one resting O_2 consumption measurement for each of the two subjects was lost due to technical problems.

There sometimes appeared to be discrepancies in subject's T_b data across experimental sessions in the early part of the recording period. Closer inspection coupled with subjective reports showed that these anomalies were most likely due to rectal probe dislodgement associated with daytime ambulatory movement. The duration of these periods were however minimal since subjects were quickly able to detect such conditions and correct them. For example, inspection of graphically presented averaged 24-hour individual T_b profiles across two experimental sessions (see Appendix D) revealed very few subjects showing such discrepancies. On those occasions where a subject's T_b markedly differed between experimental sessions, it generally coincided with the morning T_b measurements (e.g., subject C.C.). Once subjects arrived in the laboratory approximately two hours before retiring, T_{re}s were carefully monitored and scrutinised. During this wake-period subjects were non-ambulatory, and thus the risk of rectal probe dislodgement was reduced to a minimum.

Data Preparation

For all variables values were averaged over testing sessions before being entered into statistical analyses.

The usual sleep onset time of subjects was assessed by a questionnaire and the two groups compared by a *t*-test with respect to both time and the phase angle on the 24-hour temperature component. The 24-hour sine wave component of the daily temperature values was calculated from fourier transform of the original temperature data. This representation of 24-hour T_b data is often used to identify the CTR, and a description of the temperature data in terms of the 24-hour component remains useful. The

identification of the 24-hour sine wave component allowed the times of sleep onset and awakening to be expressed in terms of phase angle on the 24-hour temperature rhythm (cosine value) for both the usual and actual sleep data. The position of the 24-hour component, as identified by the minimum value (Fookson, Kronauer, Weitzman, Monk & Moline, 1982; Fookson & Moline, 1985), was compared between the two groups by a *t*-test.

In order to determine if holding time of sleep onset constant successfully held T_b constant between the two groups the T_{re}s of the two groups were compared at five occasions during the pre-sleep and early sleep period. The temperatures compared were the average values over the five minutes following: going to bed (during the O_2 consumption measurements); lights out; sleep onset; SWS onset; and REM onset. The analysis was a 2x5 ANOVA with repeated measures on the second variable. In addition, T_{re}s were compared at subjects usual sleep onsets. Finally, the mean, maximum and minimum values were compared between the two groups. The results from analyses of both the original temperatures and the 24-hour component were essentially identical. Thus, in most instances, only the results of the analyses of the original data will be reported.

Mean O_2 consumption values were calculated over the last 10 minutes of the half hour recording period. Values for each group of subjects were compared by a *t*-test.

The laboratory sleep recordings were scored as described above and a number of sleep variables were then abstracted from the data (see Table 7.2). The two groups were compared on each variable using *t*-tests.

RESULTS

The usual mean sleep onset times for the two groups were significantly different [$t(14) = 5.34$, $p < .001$], being 2237 hours (SD = 44.4 minutes) for the athletes and 0107 hours (SD = 61.4 minutes) for the sedentary subjects.

The average daily temperature rhythms and the 24-hour components (referenced to clock time) for the two groups of subjects are illustrated in Figure 7.1. The phase position of the 24-hour component was slightly, though not significantly [$t(14) = 1.53$, $p > .05$], earlier in the athletes, the nadir being 0431 hours for the athletes and 0512 hours for the sedentary subjects. The phase position at sleep onset in the laboratory also was not significantly different [$t(14) = 0.02$, $p > .05$]. The values were 98° for both groups. However, phase angle at the subjects usual time of sleep onset was different [$t(14) = 3.50$, $p < .01$], being significantly earlier in the athletes (92° versus 120°).

The temperature profiles of the two groups were initially compared on five occasions (bedtime, lights out, sleep onset, SWS onset and REM onset) in a Groups by Time, 2 x 5 ANOVA, with repeated measures on the second variable. ANOVA summary tables are presented in Appendix D. The analysis indicated a non-significant effect of groups [$F(1,14) = 0.78$, $p > .05$], a significant effect of occasions [$F(4,56) = 127.22$, $p < .001$], and a non-significant interaction effect [$F(4,56) = 2.16$, $p > .05$]. The T_{re}s for the athletes and sedentary subjects over the five occasions were 37.03°C and 37.22°C; 36.78°C and 36.95°C; 36.63°C and 36.69°C; 36.57°C and 36.61°C; and 36.41°C and 36.47°C respectively. Thus, as predicted, with time of sleep onset held constant T_b during the sleep onset period did not differ between the two groups. However, T_{re} at the time of usual sleep onset did show a significant difference [$t(14) = 2.68$, $p < .05$] with T_{re} being higher in the athlete (36.95°C) than sedentary group (36.47°C). It should be noted however, that under

the experimental conditions of this study the sedentary subjects were asleep at their usual sleep onset time, a condition which would be expected to affect T_{re}s. A comparison of the 24-hour component T_{re}s at the usual time of sleep onset showed values similar to those reported by Hedges (1989) for both athletes (36.83°C) and sedentary subjects (36.61°C), though the differences were only marginally significant [$t(14) = 1.84$, $.10 > p > .05$].

The maximum [$t(14) = 0.36$, $p > .05$], minimum [$t(14) = 0.13$, $p > .05$] and mean [$t(14) = 0.08$, $p > .05$] daily T_{re}s did not differ between the two groups. The T_{re} values for the athletes and sedentary subjects were 37.50°C and 37.45°C; 36.22°C and 36.2°C; and 36.84°C and 37.83°C respectively.

Oxygen consumption was significantly higher [$t(14) = 2.71$, $p < .05$] in the endurance athletes (256 ml/min. [SD = 47.1], or adjusted for body weight, 3.77 ml/kg/min. [SD = 0.8]) than in sedentary individuals (205 ml/min. [SD = 24.6], or adjusted for body weight, 3.13 ml/kg/min. [SD = 0.4]). As can be seen from these data, the RMR values for the endurance athletes in this study were largely consistent across subjects in contrast to those found in Experiment 1. This is most likely due to a more consistent VO_{2max} within the group of athletes used in this study compared to those in Experiment 1. Correlations between MR and selected sleep variables are presented in the correlation matrix in Table 7.1 which consisted of values for each subject from the fit and unfit group on each separate session. Data from 31 individual nights were therefore included in the correlation analysis since the data from the second session for one subject (BR) was excluded due to particular sleep disturbance on this night. The correlation analysis performed on the MR data and sleep variables showed significant positive correlations between RMR and SWS across the first sleep cycle ($r = .38$, $p < .05$), and across the entire recording period ($r = .35$, $p < .05$), particularly when the component stage 4 was correlated with RMR ($r = .39$, $p < .05$ and $r = .32$, $p < .05$, respectively).

Mean values for the sleep variables and a summary of the statistical analyses are shown in Table 7.2. As can be seen SWS levels

were generally elevated in the athletes, the difference being statistically significant for SWS and stage 3 over the whole night, and for SWS during the first 150 minutes of sleep. Sleep duration also was higher as was the number of minutes in REM sleep. To determine if the higher levels of REM sleep were entirely a reflection of the larger TST of the athletes, REM was expressed as a percentage of TST. The difference remained significant ($t(14) = 2.95$, $p < .05$; 25% REM (SD = 1.4) and 21% REM (SD = 3.1) for fit and unfit groups respectively). Finally the athletes spent less time awake during the period in bed.

DISCUSSION

The present study investigated whether sleep differences between athletes and sedentary individuals were due to T_b differences by comparing these two groups under conditions of no exercise and constant sleep onset times. The results showed that T_b at sleep onset was virtually the same for the two groups when they had similar times of sleep onset. At the subjects' usual time of sleep onset, T_b was higher in endurance athletes compared to the sedentary individuals which was not dependent on the athletes having exercised on that day.

Another study which compliments this one was conducted by Hedges (1989) which directly compared T_b between endurance athletes and sedentary controls at their usual time of retirement although laboratory sleep data was not collected. In Hedges (1989) study the results showed higher temperatures at sleep onset in endurance athletes, a finding consistent with the hypothesis that higher levels of SWS observed in endurance athletes are mediated by T_b (Horne, 1981; Horne & Moore, 1985). Average T_{re}s were higher in athletes compared to sedentary subjects although this difference had not directly caused higher T_{re}s at sleep onset since at sleep onset the temperatures of the two groups were found to be essentially the same at any particular clock time. Furthermore, in that study a major methodological problem was that the athletes continued to train

throughout the data collection period which would clearly have affected their T_{re}s with the apparent higher average temperatures most likely being exercise-dependent and thus in contradiction to the T_b hypothesis considering that the characteristic sleep effects of endurance athletes are not exercise-dependent (Trinder et al., 1988). Furthermore, the effect of exercise on T_b in athletes assessed by Hedges (1989) may have masked group differences in the phase position of the temperature cycle.

Given the results of the current study and the support from the Hedges (1989) study, it is concluded that the higher temperatures at usual sleep onset were most likely due to the earlier time of going to bed, and consequently earlier sleep onset of the athletes. In effect, since the clock position of the daily temperature rhythm was similar in the two groups, earlier sleep onsets would phase delay the temperature rhythm of the athletes with respect to the sleep-wake cycle (Hedges, 1989). A consequence of this relationship was the non-significant difference in T_b at sleep onset between the two groups when sleep onset times are similar. It should be noted that the relative position of the daily temperature rhythm reported in Hedges (1989) study was not due to exercise masking as this study achieved the same effect and furthermore the results of the present study are not due to the common time of sleep onset as, again, the results are the same as Hedges in which sleep onset varied.

The possibility that holding Lights Out time constant may have affected the sleep of the unfit subjects was unlikely based upon the following considerations. Firstly, there was no evidence to suggest that the unfit subjects had been pushed into a wakefulness zone as SOL did not differ between the groups and, perhaps more significantly, the variance did not differ between the groups. Furthermore, the values were consistent with other studies for the two groups. Secondly, the earlier sleep onset would have reduced prior wakefulness. Studies that have assessed the effect of small (up to 2 hours) advances in LO have not reported significant sleep effects (Croft, Montgomery & Trinder, 1992, unpublished observations).

This is in contrast to small delays that do elevate SWS. Nevertheless, it could be argued that the effect of varying LO in this context could be more thoroughly studied, where initial studies support the view that temperature at sleep onset does not mediate the sleep effects in athletes.

Despite similar T_bs at sleep onset the endurance athletes had higher levels of SWS and longer sleep durations than the sedentary subjects. This result replicates earlier studies (Trinder et al., 1988) and suggests that the differences in sleep characteristics between endurance athletes and other individuals were not dependent on differences in T_b at sleep onset. However, the present results do not dispute the effect of T_b on sleep in other experimental situations. Indeed, it is possible that larger sleep differences between the groups would be obtained if T_b at sleep onset were higher in the athletes.

It is of interest to note that the exercise performed by the athletes in the Hedges (1989) study, though affecting average T_bs, did not appear to alter the estimated position of the 24-hour component of the daily temperature rhythm, a variable which is often used as a measure of the CTR. Thus, exercise, at least in athletes would not appear to have a masking effect on the CTR. Similarly, T_b at the time of usual sleep onset was not affected by exercise in the athletes.

The manipulation used in the present study was to change the time of sleep onset in the sedentary subjects, rather than that of the endurance athletes. The acute shift in time of sleep onset in this group may have disrupted sleep, either because of a reduction in the duration of prior wakefulness, or because of a disruption in sleep routine. While this remains a plausible interpretation, studies which have advanced sleep onset time by amounts slightly larger than in the present study, have generally not observed effects on SWS and sleep duration (Taub & Berger, 1973a,b; 1976).

Table 7.1
Correlation matrix showing correlations between MR and sleep variables

	RMR	SOL	TTA	TST	REM	SWS	Stage4	Stage3	1stSWS	1stS4	1stS3
RMR	1										
SOL	-40*	1									
TTA	-28	.73***	1								
TST	.45**	-39*	-63***	1							
REM	.45**	-24	-47**	.82***	1						
SWS	35*	-19	-42**	.55***	.43**	1					
Stage4	32*	-14	-33*	.47**	29	.93***	1				
Stage3	23	-20	-42*	.43**	.50**	.63***	31*	1			
1stSWS	38*	-47**	-62***	.54**	.42*	.84***	.75***	.60***	1		
1stS4	39*	-24	-40*	.44**	31*	.85***	.91***	.27	.87***	1	
1stS3	-01	-45**	-43**	.19	.21	-01	-30	.63***	.27	-25	1

Significance levels are indicated by the following * p<.05, ** p<.01, *** p<.001.

Abbreviations : Variables are defined in previous tables.

Table 7.2

Mean time for sleep variables for endurance athletes and sedentary individuals across two experimental sessions

SLEEP VARIABLES	MEAN TIMES (min) (SDs in Parentheses) FIT	UNFIT
SWS (150min)	55 (9.8)	39 (12.2)**
Stage 3	25 (6.5)	21 (7.2)
Stage 4	30 (13.0)	18 (12.0)
SWS (After 150 mins)	21 (17.1)	9 (9.6)
Stage 3	14 (8.9)	7 (5.8)
Stage 4	7 (9.7)	2 (4.3)
SWS (Total Night)	76 (24.5)	48 (15.5)*
Stage 3	39 (8.1)	28 (7.3)**
Stage 4	37 (21.3)	20 (14.2)
REM (150min)	10 (5.7)	10 (4.2)
REM (After 150mins)	115 (8.6)	79 (17.5)***
REM (total night)	125 (12.1)	89 (20.5)***
REM Latency[a]	89 (41.4)	101 (43.7)
REM%	25 (1.4)	21 (3.1)*
TST	510 (26.7)	422 (49.7)***
Time in Bed	535 (37.4)	493 (12.9)**
Stage 2 (150mins)	80 (11.1)	86 (16.8)
Stage 2 (After 150mins)	199 (24.1)	170 (28.1)*
Stage 2 (Total Night)	279 (27.9)	256 (41.5)
Sleep Latency[b]	14 (12.3)	27 (12.4)
SWS Latency	13 (4.0)	19 (7.6)
Disturbed Sleep (1+wake)	55 (28.6)	100 (52.3)*
Stage 1 (Total Night)	30 (12.1)	29 (9.6)
Total Time Awake	25 (23.6)	71 (49.9)*
Awake during the Night	14 (19.0)	47 (40.8)*
Awake before Sleep	11 (10.7)	24 (12.6)*
Sleep Efficiency[c]	.96(.04)	.86(.09)*

* Groups significantly different at $p < .05$.
** Groups significantly different at $p < .01$.
*** Groups significantly different at $p < .001$.
Note. The sleep data for subject [B.R] on the second experimental night was excluded from the overall mean.
[a]Time from the first stage 2 to the first REM epoch.
[b]Time from lights out to the first stage 2 epoch.
[c]Ratio of total sleep time to time in bed.

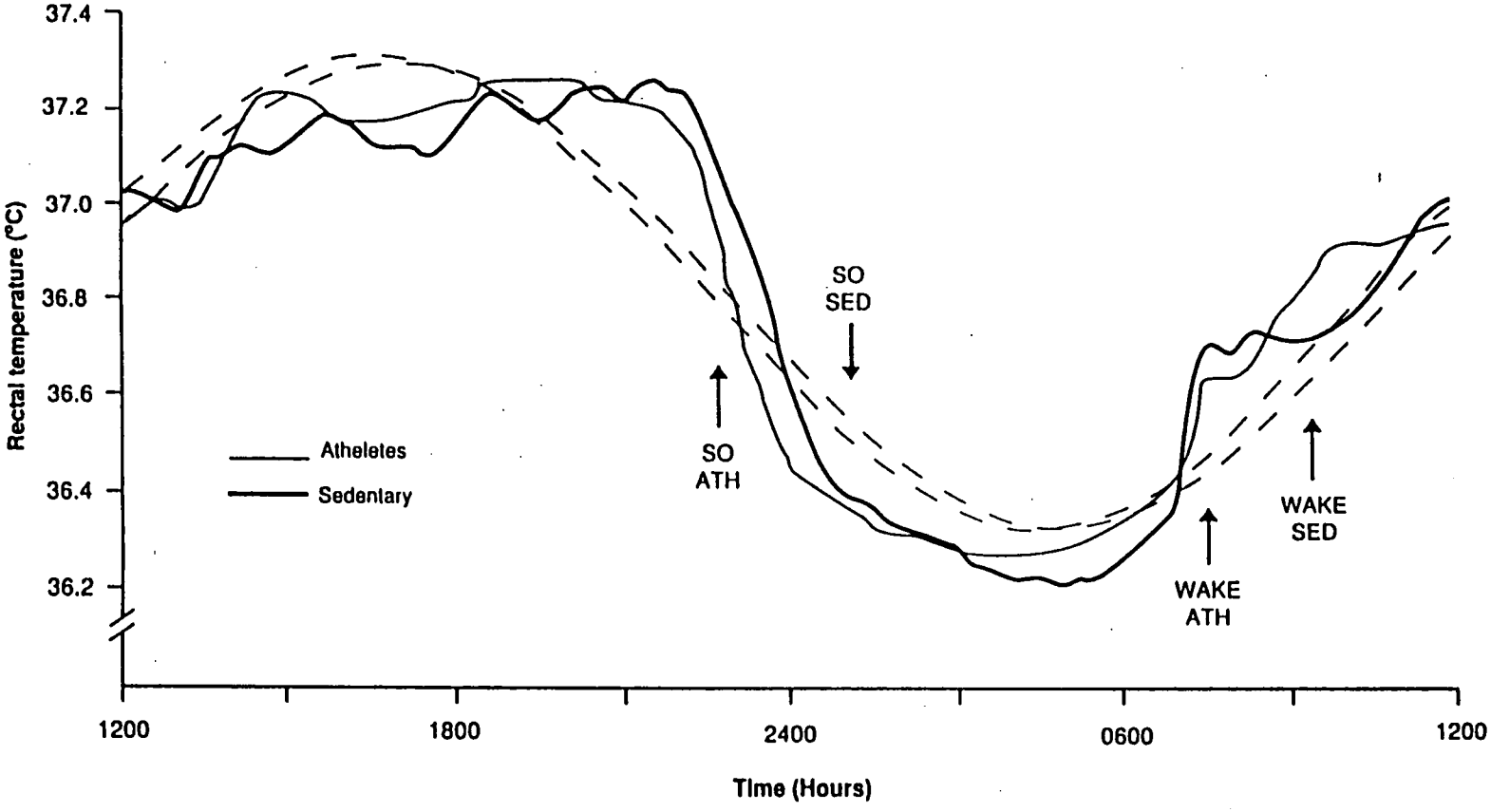

Figure 7.1: *Average T_{re}s where the values shown are the original values and the 24-hour components for endurance athletes and sedentary individuals referenced to clock time. Average usual weekday time of sleep onset (SO) and waking (WAKE) are indicated by arrows. The earlier of the two cosine functions is the 24-hour component for the endurance athletes.*

One interpretation of the present results is that they demonstrate an energy conserving or bodily restorative effect of endurance training independent of a T_b control mechanism. It is believed that this view is unlikely to be correct as the effects of physical exercise on sleep have only been shown to occur in endurance athletes (Trinder et al., 1988). It seems unlikely that, as virtuous as jogging may be, only these athletes would be the recipients of beneficial sleep changes.

A related hypothesis is that the sleep changes may be a compensatory response to the elevated MRs observed in the athletes in this study. The generalisation of a MR difference between groups however, was based upon a limited period of MR measurement that may not have provided a comprehensive assessment of the MR difference between groups at all circadian time points within a 24-hour period. It is therefore proposed that a more complete assessment of the MR status of each group would have been attained had MR been measured at more time points during the 24-hour period as was the case for temperature recording. Nevertheless, while the compensatory hypothesis remains possible, it should also be noted that the passive heating manipulation used in Experiment Two that resulted in increased SWS levels, did not elevate pre-sleep MR.

It is suggested that the sleep properties characteristic of endurance athletes are due to a particular phase relationship between the athletes sleep-wake cycle, as identified by their usual time of sleep onset, and their major circadian oscillator, as identified in this study by the 24-hour component of the raw temperature data. Specifically, it is proposed that the particular sleep properties of endurance athletes are to a phase delay of the circadian oscillator which, in this group, is achieved by an advance of the sleep-wake cycle (earlier usual sleep onset time). The hypothesis is supported by evidence that sleep onset higher on the falling phase of the major circadian oscillator is associated with higher levels of SWS (Campbell & Zulley, 1989) and longer sleep durations (Strogartz, Kronauer & Czeisler, 1986).

CHAPTER 8

GENERAL DISCUSSION AND CONCLUSIONS

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The studies presented have attempted to investigate the nature of the relationship between metabolic processes and sleep architecture. More specifically, the relationship between MR, T_b and sleep processes were assessed by means of direct or indirect measures. The major rationale for conducting the three studies was based upon those findings that have previously been used to highlight the possible function of the sleep process, particularly the component SWS.

From previous research there has been development of a number of theories for sleep function, but it is those theories of restoration and energy conservation that hold particular relevance to the studies reported. These theories largely advocate a compensatory role to the sleep process, where it is the stages of SWS, particularly stage 4, that allow increased wake-period metabolism to be counteracted during sleep. The notion of SWS being the major vehicle for compensation has been derived from direct attempts to increase catabolic processes so as to observe effects upon sleep properties. Other indirect evidence to support the theories has been derived from phylogenetic and ontogenetic comparisons, as well as from the assessment of changes more directly associated with metabolism, such as in the case of weight loss and human growth studies, and the subsequent effects on sleep properties. The general contention is that increased levels of metabolism facilitate a greater need for conservation and restoration as part of an overall compensatory process. It is thought that SWS particularly serves to restore bodily processes from increased catabolic processes (Adam & Oswald, 1977; Horne, 1981, 1988, 1989).

While there is much evidence to indirectly support the notion of compensatory properties of sleep, there are however, findings to be found in the literature that contradict the theories of restoration

and conservation. This is particularly noticeable from a review of the literature on the effects of physical exercise on sleep (Trinder et al., 1988). For example, not all of the research studies have reported an exercise-induced SWS effect. It appears that exercise only affects sleep under limited circumstances. However, obvious methodological deficiencies in some of the studies reported may have contributed to the contradictory sleep findings in this research area. For example, failure to control for time of day effects; absence of adequate control groups or conditions; the use of too small a sample size; and the fact that a vast majority of studies have exclusively examined whole-night effects of exercise on sleep, thus failing to dissect the night into smaller segments to examine the short-term effects that exercise may have on sleep, are just a few of the research deficiencies that must be acknowledged as possible contributors to the contradictory evidence. Nevertheless, there is no study to date that has directly measured MR and T_b during the sleep period following increases in wake-period metabolism. Moreover, it has recently been suggested that T_b maybe the primary initiator of SWS changes. In fact, it has been suggested that the inconsistencies in the exercise-sleep literature is a result of insufficient elevation of T_b (Horne, 1981). Since the most consistent finding, with respect to the effect of exercise on sleep, is that chronically higher levels of SWS and longer sleep durations appears in endurance athletes, it is of interest to consider whether this effect is T_b-related. Such consideration underlies the rationale for the final study reported in this thesis.

Rationales, Aims and Experimental Methodology Revisited

The first and second studies attempted to directly alter the levels of waking activity in order to investigate its relationship to subsequent sleep properties and sleep-period MR and/or T_b. Techniques used to increase wake-period metabolism were either by active (i.e., physical exercise) or passive (i.e., body heating) means. The final study assessed T_b in endurance athletes as compared to a sedentary control group in order to determine if the consistent sleep effects found in the endurance athletes were due to the effect of T_b on

sleep. The rationale, aims and experimental methodology related to the three studies are briefly discussed. Experimental findings and implications are discussed later.

Experiment One: The Effects of Exercise on Metabolic rate and Sleep Architecture in the First Sleep Cycle

The first study was designed to assess directly the theories of compensation by inducing heightened levels of wake-period metabolism, and then assessing its relationship to subsequent sleep-period metabolism and architecture. Since there is contradictory findings among the exercise-sleep literature, as well as a lack of evidence to show the direct relationship between MR and sleep properties, this study measured both MR and EEG sleep properties during the first sleep cycle. An intense exercise routine consisting of a 19 km run was used to induce MR elevations in endurance athletes. It also was assumed that these MR elevations would be compensated for by increased SWS levels in the subsequent sleep period. Such an effect would support theories of restoration and conservation. Furthermore, monitoring MR into the early sleep period allowed a direct assessment of the nature of MR following such an intense exercise routine. Metabolic rate was assumed to be sustained at least into the early sleep period (Simonson, 1971). Any changes in SWS would therefore be interpreted as a direct response to the increased MR, or in more general terms, metabolism.

Experiment Two: The Effect of Afternoon Body Heating on Body Temperature, and Slow Wave Sleep

The second study was designed to assess the effects of changes in the level of wake-period activity on sleep. Rather than using an active technique such as exercise to induce these changes however, a passive method of increasing metabolism was used to maximize T_b elevations. Since recent data has pointed to the importance of T_b in facilitating SWS, especially cerebral heating (Berger et al., 1988; Horne, 1981; Sewitch 1987), its measurement was included in this

study. As mentioned previously, although T_b was not monitored in the first study, it is possible that the failure to find sleep changes was due to insufficient elevation in T_b. It is therefore apparent that, to provide a more complete assessment of bodily changes in response to increased activity levels, T_b should be monitored in conjunction with MR measures.

The method used in the second study was passive body heating. Body heating by submersion of the subject in a warm bath decreased heat loss through sweating while increasing T_b, which in turn increased bodily oxidative activity and thus MR (Keele et al., 1982). The aim was therefore to investigate the effect of passive heating on MR, T_{re} and SWS during the subsequent sleep period. It also assessed whether it was absolute T_b at sleep onset (Berger & Phillips, 1988a,b), or the rate of fall of T_b following sleep onset (Sewitch, 1987) which determined the level of SWS in the subsequent sleep period.

Experiment Three: The Relationship between Body Temperature and Sleep Architecture in Endurance Athletes

A review of the exercise-sleep literature revealed that the only consistent finding to support the view of sleep serving a compensatory function was that endurance athletes showed chronically higher levels of SWS, and longer TST than their sedentary counterparts (e.g., Griffin & Trinder, 1978). It was this evidence that provided the rationale behind the final study. In principle, these sleep differences may have been a consequence of underlying metabolic differences between the two groups. Whether T_b initiated these sleep differences remained an important research issue, and therefore provided the basis for a comparison of T_b, MR and sleep in endurance athletes and sedentary individuals.

Changes in Metabolic Rate and Body Temperature and their Effect on Sleep Architecture

It was surprising to find that the exercise used in the first study failed to produce subsequent changes in sleep properties, especially SWS, given the number of studies that have shown exercise-induced SWS effects in endurance athletes. It was noted that while exercise successfully increased MR, these increases were only short-lived and had returned to baseline levels by the time of retirement. Based upon previous evidence (Simonson, 1971; Wilmore, 1977), it was expected that these increases may have been sustained for longer. In retrospect however, the increased fitness levels of the subjects may have accelerated the return of MR to baseline levels (deVries, 1980).

Given the results of this study, it is difficult to openly accept the theories of compensation since there was no evidence to suggest that increased wake-time metabolism facilitated greater SWS as a compensatory response. Indeed, there was some evidence from this study to suggest that perhaps increased wake-period metabolism was positively related to such sleep properties as SOL and SWS onset.

Alternatively, the negative SWS results may have been a consequence of methodological influences. For example, the cold T_a conditions under which subjects ran the 19 km may have counteracted effects of increased T_b, or more specifically cerebral temperature. However, since T_b was not monitored throughout the recording session its status remains unknown and thus open to speculation. Furthermore, the use of a facial mask may have inhibited or delayed sleep effects in this study until the second sleep cycle (Ryan et al., 1989; White et al., 1985), but since only the first sleep cycle was recorded, this too remains open to conjecture. It also is possible that exercise or fitness may have had a more pronounced effect on SWS amplitude or power than on the time spent in stages 3 and 4 as conventionally scored. The possibility of some variation of amplitude of delta activity within stage 4 would have to be considered an important variation of the sleep parameter related to the depth of

sleep and relevant to the theories of sleep in terms of compensation or energy conservation. The quality of the data would have been improved had techniques of digital recording been available allowing a more sensitive power spectral analysis through FFT routines to be calculated. These methods of sleep analysis would therefore be preferable to the older, more arbitrary and crude methods of sleep scoring systems (Dijk, Beersma & Bloem, 1989; Dijk, Brunner, Beersma & Borbely, 1990) that were used in the assessment of sleep architecture in the reported studies (Rechtschaffen & Kales, 1968). As such, the method of sleep analysis should be considered in future research when assessing the effect of variations in waking activity on sleep.

The experimental findings from the second study, clearly showed T_b to contribute to the sleep effect. It was found that while passive heating significantly elevated T_b, and MR only in two of the four subjects from which O_2 consumption data was available, it was only T_b that showed continued elevation into, and across the sleep period. This elevation appeared to be associated with increased SWS levels, particularly stage 4, in the early sleep period. The non-significant increase in MR following passive heating was surprising considering the general effect of increased T_b on oxidative activity and basal metabolism (Keele et al., 1982), and as such would not support Horne's (1981) theory. This unexpected finding was most likely due to a thermogenic effect associated with the evening meal which masked the effect of the bath. It was therefore acknowledged that O_2 consumption should have been measured in the control condition. Nevertheless, SWS effects were found in this study following wake-period variations in metabolism, whereas this was not the case for Experiment One. It is therefore proposed that the T_b-SWS effects found in Experiment Two provide support for the view that absolute T_b is the critical factor facilitating SWS changes (Berger & Phillips, 1988a,b; Horne, 1981). The failure to find a SWS effect in Experiment One may have been a consequence of unchanged T_bs, although this is still uncertain since T_b was not recorded. The implications of the findings from both studies is that the SWS effects

that have been previously reported in response to increased waking activity maybe more dependent upon T_b changes than MR elevations. Furthermore, it is proposed that the theories of body restoration and conservation are questionable given these results. It is more likely that cerebral heating is responsible for SWS effects, rather than increased SWS levels representing a compensatory vehicle for general increases in total body wear and tear. That is, SWS responds more to increased cerebral temperature than general body metabolism. The extent to which the underlying circadian rhythm influenced these temperature changes remains uncertain, and is thus of research interest.'

Individual Differences, Metabolic Rate, Body Temperature and Sleep Architecture

Endurance athletes have previously been found to retire at an earlier sleep onset time than sedentary groups (Hedges, 1989) which most likely has the effect of phase delaying the temperature rhythm of the athletes with respect to the sleep-wake cycle. The research findings from the final study further showed that T_b at sleep onset is essentially the same for endurance athletes and sedentary individuals if sleep onset times are similar. It was interesting to find that regardless of the similar T_bs at sleep onset the endurance athletes had higher SWS levels and longer sleep durations than their sedentary counterparts, a result that supports earlier research findings (Trinder et al., 1988). These findings further suggest that the unique sleep characteristics of endurance athletes may be distinct from the effects of T_b at sleep onset. The present results however, do not dispute the effect of T_b on sleep in other experimental situations, such as in the case of body heating. It may be found that greater sleep differences occur between the groups if T_b at sleep onset is higher in the athletes.

It was considered that an energy conservation or restorative effect of endurance training independent of a T_b control mechanism was unlikely since these exercise-related sleep effects have only been shown to occur in endurance athletes (Trinder et al., 1988).

Furthermore, the apparent increases in MR in the endurance athletes found in this study was based upon only one half hour period prior to sleep onset and thus may not be considered robust enough as evidence to support the theories of compensation. A more informative approach would have been to measure MR at the same circadian time points as the temperature data.

It is therefore proposed that the phase relationship between the sleep-wake cycle and the major circadian oscillator is an important determinant of sleep properties. This particularly seems apparent when the results from the final study are considered in association with other available evidence investigating sleep architecture in endurance athletes (Hedges, 1989). For example, it is possible that the unique sleep characteristics of endurance athletes is a function of a phase delay of the circadian oscillator precipitated by an advance of the sleep-wake cycle. This view is particularly feasible given that sleep onset higher on the falling phase of the major circadian oscillator is associated with higher SWS levels (Campbell & Zulley, 1989) and extended periods of sleep (Strogartz et al., 1986).

An Overview of the Experimental Findings

The results from the studies cast some doubt as to the validity of the theories of general body restoration and energy conservation (Horne, 1988). The fact that there was no evidence from the first study to suggest that sleep architecture responded to changes in the level of physical activity and transient increases in wake-period metabolism as predicted by the functional theories of energy conservation and bodily restoration questions their credibility. Furthermore, this finding in association with a review of the exercise and physical fitness literature (Trinder, Montgomery, Jordan & Croft, 1992, submitted for publication) suggest that sleep does not show immediate compensatory changes indicative of an increase in functional activity following increases in physical activity and metabolism. The fact that athlete groups have consistently shown

higher levels of SWS and longer sleep durations as supported by the final study may suggest that the compensatory sleep response has a degree of inertia, so that while sleep structure does not change in response to short term variations in physical activity, it is responsive to long term trends (Trinder et al., 1992, unpublished manuscript). The data are not readily accommodated by the compensatory theories however, since the general finding that only endurance athletes and not other athlete groups show structural changes in sleep suggest that the effect in endurance athletes is more likely to be due to an alternative mechanism.

The role of T_b or more specifically, brain or cerebral temperature, has been considered as an important factor contributing to the inconsistencies in exercise findings where physical exercise only affects SWS under those circumstances in which it elevates cerebral temperature (Horne & Moore, 1985; Horne & Staff, 1983; Shapiro et al., 1975). This is made even more likely since under conditions when exercise is performed out-of-doors, no SWS effects have been reported (Montgomery et al., 1987; Montgomery et al., 1985; Torsvall et al., 1984). It has been suggested that this is due to increased cerebral cooling via evaporative and convective cooling of the head due to increased airflow around the head associated with outdoor activity (Cabanac, 1986). However, this does not appear to be a general process by which exercise elicits a compensatory sleep response as specific mechanisms exist to prevent increases in cerebral temperature during physical exercise (Trinder et al., 1992, unpublished manuscript).

The results from the passive heating study suggest that a more likely temperature mechanism is that SWS is potentiated by a small increase in cerebral temperature during the early sleep period. This view is supported by other studies (e.g., Berger et al., 1988) that suggest an increase in SWS is indicative of a thermoregulatory (Obal, 1984), or energy conserving (Berger et al., 1988), function. It could therefore be considered that cerebral temperature may not have been sufficiently elevated at sleep onset in the first study to produce the

SWS effect, however, this remains speculative. Furthermore, only one study has reported T_{re} (Montgomery et al., 1985), but no study has reported tympanic temperature at sleep onset following physical exercise, although it has been considered that cerebral and rectal temperatures would most likely equilibrate in a normal sleep environment (Trinder et al., 1992, unpublished manuscript).

The results from the final study assessing whether temperature characteristics of endurance athletes mediated their sleep effects of higher SWS levels and longer sleep durations, showed that the higher temperatures at sleep onset in these athletes reported in another study (Trinder & Hedges, 1989) were due to an earlier sleep onset, rather than exercise. The small temperature differences observed in these studies were similar to the difference produced at sleep onset by afternoon passive heating, a finding which appears to confirm that sleep differences in endurance athletes are due to elevated temperatures at sleep onset. The finding that the athletes still produced more SWS and longer sleep durations however, even when temperatures at sleep onset were held constant between the endurance athletes and sedentary controls by holding time of sleep onset constant, questions the above interpretation. Therefore, the mechanism producing higher SWS levels and longer sleep durations in endurance athletes remains to be identified. They are not related to energy expenditure, protein catabolism, or T_b. Furthermore, there is no other evidence to suggest that these changes in sleep architecture illustrate compensatory changes in response to functional requirements (Trinder et al., 1992, unpublished manuscript).

Future Research

From the evidence available, it is believed that temperature rather than MR plays a critical role in influencing sleep changes. Furthermore, it is suggested that SWS effects are more related to changes in brain activity, particularly following to brain temperature changes, and as such are worthy of future research. The next logical step to take in the research area would therefore be to concentrate

more upon brain temperature changes rather than general body changes. Furthermore, T_{re} may not be an accurate measure of brain temperature, but rather T_{ty} may be more appropriate as a measure of brain temperature (Treiber et al., 1991). Such methods of temperature measurement should therefore be carefully considered in future research.

Proposals for future research could include the effects on sleep of manipulated T_b using body cooling a well as heating; investigations of phase shifts of the sleep/wake rhythm; and long-term studies of the effect of T_b and MR shifts on sleep.

Summary and Conclusions

The findings from these studies particularly highlight the importance of such factors as T_b and perhaps the phase relationship between the sleep-wake cycle and circadian oscillators, in affecting sleep architecture. They also help to explain of some of the previously discrepant findings within the literature, especially in relation to the exercise-sleep research area, by demonstrating the relationship between T_b and sleep. From the findings it appears that elevated T_b at sleep onset is not the mechanism causing the characteristic sleep patterns of endurance athletes, but rather they are caused by the phase relationship between the sleep-wake cycle and major circadian oscillator. The findings do not however clearly demonstrate the relationship between MR and sleep. Nevertheless, the findings along with other available evidence suggest that the theories proposing general bodily conservation and restoration in response to increased levels of wake-period metabolism, are unlikely to be correct. It is more likely that T_b, or more specifically, cerebral temperature, may be important determinants of SWS under conditions of direct variations in waking activity. That is, changes in SWS may follow changes in cerebral temperature. For example, the T_b increases associated with passive heating may trigger a cerebral or hypothalamic response which then alters SWS. The nature of this relationship however remains unclear.

Finally, the currently reported research was conducted within a framework provided by the energy conservation and restorative theories. It is clear from the findings that this theoretical background is in a general sense heuristic. However, the details within the theoretical infrastructure require clarification. It is argued that the facilitation of SWS is promoted by either (a) high T_b during sleep onset and the early part of sleep; or (b) the phase angle in the CTR at sleep onset, or both. Further elucidation of these propositions requires still more research in the area of sleep and metabolism.

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APPENDIX A

APPENDIX A

Publications from the Thesis.

Jordan, J.E., Montgomery, I.M. and Trinder, J.A. (1990). The effect of afternoon body heating on body temperature, and slow wave sleep. *Psychophysiology, 27(5)*, 560-566.

Jordan, J.E., Montgomery, I.M., Trinder, J.A. and Hedges, P. The relationship between body temperature and sleep in endurance athletes. Paper submitted for publication in the journal of *Sleep.*

Conferences at which this Publication was Presented.

(1) Paper presented at the Australian Experimental Psychology Conference (June, 1989), Adelaide, South Australia.

(2) Paper presented at the 25th meeting of the Australian Psychological Society (October, 1989), Hobart, Tasmania.

APPENDIX B

APPENDIX B

EXPERIMENT ONE

3x2 Condition (LAM, LA. LE) x Time (pre- and post-exercise) ANOVA summary table

CSS/pc: GENERAL	Summary of all Effects; design: 1-Condition x 2-Time (pre- and post-Ex.)					
Effect	df Effect	MS Effect	df Error	MS Error	F	p
1	2	1323.45	14	2646.90	0.4757	0.6359
2	1	17290.0	7	1901.78	9.0915	0.0190
12	2	1205.90	14	662.37	1.8206	0.1972

4x3 Condition (CN, LAM, LA. LE) x Time (1530, 2000 and 2230 hours) ANOVA summary table

CSS/pc: GENERAL	Summary of all Effects; design: 1-Condition x 2-Time (1530-2000-2230hrs)					
Effect	df Effect	MS Effect	df Error	MS Error	F	p
1	3	5745.42	27	5017.62	1.1451	0.3491
2	2	6454.30	18	1279.75	5.0434	0.0179
12	6	1938.98	54	1390.98	1.3938	0.2333

Repeated Measures ANOVA

CSS/pc: GENERAL — Summary of all Effects

Source	df	Sum of Squares	Mean Square	F	p
Subject	8	6215.57	776.94		
Sleep Variable	4	17516.59	4379.15	14.19	.0001
Sleep Var. * Subject	32	9875.11	308.60		
Condition	3	226.38	75.46	.271	.85
Condition * Subject	24	6674.23	278.09		
Sleep Var. * Condition	12	4953.17	412.76	1.55	.12
Sleep Var. * Condition * Subject	96	25578.53	266.44		

Table B-1

Oxygen Consumption during each resting MR session at selected time intervals for the four experimental sessions.

	OXYGEN CONSUMPTION (ml/min.)			
TIME/SUBJECT	CN	LAM	LA	LE
1530hrs				
PN	293	251	247	268
RG	264	201	240	210
LN	256	334	308	223
DC	267	175	236	205
JP	373	426	483	484
PM	341	441	449	407
PW	177	194	172	178
SR	283	285	320	338
NM	385	400	372	407
ML	144	226	204	290
POST-MR				
PN	-	275	303	-
RG	-	220	338	-
LN	-	324	280	-
DC	-	184	275	-
JP	-	-	-	-
PM	-	-	-	-
PW	-	233	176	-
SR	-	339	390	-
NM	-	509	553	-
ML	-	234	245	-

Table B-1 (cont'd)

	OXYGEN CONSUMPTION (ml/min.)			
2000hrs				
PN	315	263	259	303
RG	225	229	237	242
LN	227	334	251	266
DC	181	151	278	207
JP	400	691	538	501
PM	442	439	453	489
PW	185	188	159	157
SR	272	379	353	349
NM	419	443	428	377
ML	191	207	233	302
POST-MR				
PN	-	-	-	321
RG	-	-	-	215
LN	-	-	-	311
DC	-	-	-	223
JP	-	-	-	-
PM	-	-	-	-
PW	-	-	-	151
SR	-	-	-	430
NM	-	-	-	423
ML	-	-	-	327
2230hrs				
PN	308	234	303	229
RG	215	213	208	180
LN	196	309	237	250
DC	196	137	234	187
JP	353	546	589	438
PM	420	476	420	439
PW	186	186	139	148
SR	261	355	339	350
NM	389	489	379	381
ML	221	245	168	304

Abbreviations:
LAM = Late Afternoon Exercise With Meal
LA = Late Afternoon Exercise Without Meal
LE = Late Evening Exercise
CN = Control

Table B-2

Power analysis for sleep data reported from laboratories outside of the Hobart research unit

STUDY	CONTROL SWS %	EXERCISE SWS %	DIFFERENCE SWS %
Baekland (1970)	13.3	17.1	3.8
Baekland & Lasky (1966) [Evening Ex]	32.5	35.4	2.9
Baekland & Lasky (1966) [Afternoon Ex]	32.5	40.1	7.6
Bonnet (1980)	14.1	14.9	0.8
Horne & Moore (1985)	29.3	35.1	5.8
Horne & Staff (1983) [HI Ex]	16.8	19.8	3.0
Kupfer et al. (1985)	17.1	16.9	-0.2
Shapiro,Bortz, Mitchell,Bartel, & Jooste (1981)	22.0	42.0	20.0
Shapiro, Griesel,Bartel, & Jooste (1975)	20.5	24.5	4.0
Torsvall, Akerstedt & Lindbeck (1984)	20.2	20.6	0.4

APPENDIX C

APPENDIX C

EXPERIMENT TWO

2 x 2 Condition x Time ANOVA summary table for T_{re}s over the interval before and after passive heating

CSS/pc: GENERAL		Summary of all Effects; design: 1-Condition x 2-Time				
Effect	df Effect	MS Effect	df Error	MS Error	F	p
1	1	2.5348	4	0.0059	42.3344	0.0042
2	1	2.1650	4	0.0372	58.2463	0.0027
12	1	2.8881	4	0.0599	96.9631	0.0015

2 x 2 Condition x Time ANOVA summary table for T_{re}s over the interval after passive heating and before lights out

CSS/pc: GENERAL		Summary of all Effects; design: 1-Condition x 2-Time				
Effect	df Effect	MS Effect	df Error	MS Error	F	p
1	1	3.4528	4	0.0408	84.4358	0.0018
2	1	3.8457	4	0.0526	73.1800	0.0021
12	1	2.0547	4	0.0417	49.2884	0.0034

2 x 4 Condition x Time ANOVA summary table for T_{re}s over the interval from lights out to 60 minutes following SWS onset

CSS/pc: GENERAL		Summary of all Effects; design: 1-Condition x 2-Time				
Effect	df Effect	MS Effect	df Error	MS Error	F	p
1	1	0.3118	4	0.0049	62.4851	0.0025
2	3	0.3654	12	0.0038	96.4218	0.0000
12	3	0.0018	12	0.0012	1.4969	0.2649

2 x 2 ANOVA summary table for the Condition x Sleep onset time interaction for O_2 consumption 15 minutes immediately following Stage 1 sleep onset for Control and Passive Heating conditions

CSS/pc: GENERAL		Summary of all Effects; design: 1-Condition x 2-Sleep onset time				
Effect	df Effect	MS Effect	df Error	MS Error	F	p
1	1	22.5625	3	234.063	9.6395	0.7656
2	1	3813.0625	3	27.5625	138.3424	0.0037
12	1	22.5625	3	40.7292	0.5539	0.5131

APPENDIX D

APPENDIX D

EXPERIMENT THREE

2 x 5 Group x Time (time in bed; lights out; sleep onset; SWS onset; REM onset) ANOVA summary table for absolute T_{re}s between time intervals starting from the first five minutes following each time interval

CSS/pc: GENERAL		Summary of all Effects; design: 1-Group x 2-Time				
Effect	df Effect	MS Effect	df Error	MS Error	F	p
1	1	0.2205	14	0.2811	0.7843	0.3946
2	4	1.1305	56	0.0089	127.2170	0.0000
12	4	0.0002	56	0.0089	2.1567	0.0849

Table D-1:

Absolute Rectal Temperatures (in °C) across the two experimental sessions during the five minute epoch starting at habitual retirement time for athletes and sedentary subjects.

	Subject (Initials)	Experimental Session 1 T_{re}	Experimental Session 2 T_{re}	Mean T_{re}
S	JB	36.55	36.26	36.41
E	TD	36.52	36.19	36.36
D	ND	36.34	35.88	36.11
E	BG	36.97	36.90	36.94
N	SM	36.55	36.33	36.44
T	DM	36.55	36.40	36.48
A	AP	36.95	36.95	36.95
R	SR	36.32	36.33	36.32
Y				
A	MA	37.02	37.21	37.12
T	CC	36.50	36.34	36.42
H	GH	36.85	36.41	36.63
L	RM	37.45	37.49	37.47
E	AP	36.96	36.79	36.88
T	BR	36.88	-	36.88
E	DS	37.49	37.73	37.61
S	PT	36.98	36.62	36.80

Table D-2

Mean absolute rectal temperatures (in °C) for selected time intervals relative to experimental retirement time for athletes and sedentary groups. (SDs are shown in parentheses)

TIME	GROUP		
	Athletes	Sedentary	Mean Temperatures (oC)
5 min following:			
bedtime	37.03 (0.28)	37.22 (0.29)	37.12 (0.29)
lights out	36.78 (0.23)	36.95 (0.33)	36.86 (0.29)
sleep onset	36.63 (0.20)	36.69 (0.26)	36.66 (0.23)
SWS onset	36.57 (0.21)	36.61 (0.25)	36.59 (0.23)
REM onset	36.41 (0.19)	36.47 (0.24)	36.44 (0.21)

Note. The statistical analyses are described in the text. The absolute T_{re}s did not differ significantly between groups at any time, but did differ between time intervals [$F(4,56) = 127.22$, $p < .001$].

Table D-3

Mean resting Oxygen Consumption for athletes and sedentary subjects prior to lights out

GROUP	MEAN OXYGEN CONSUMPTION (ml/min) (SDs in Parentheses)
Athletes	256 (47.1)
Sedentary	205 (24.6)

Note. Analysis of mean O_2 consumption using an independent groups *t*-test showed the difference to be significant, [$t(14) = 2.71$, $p < .02$].

Averaged Individual 24-hour T_{re} recordings across two experimental sessions.

Figure D-1: Athlete group
Figure D-2: Sedentary group
(Note: each data point represents a 15 minute average)

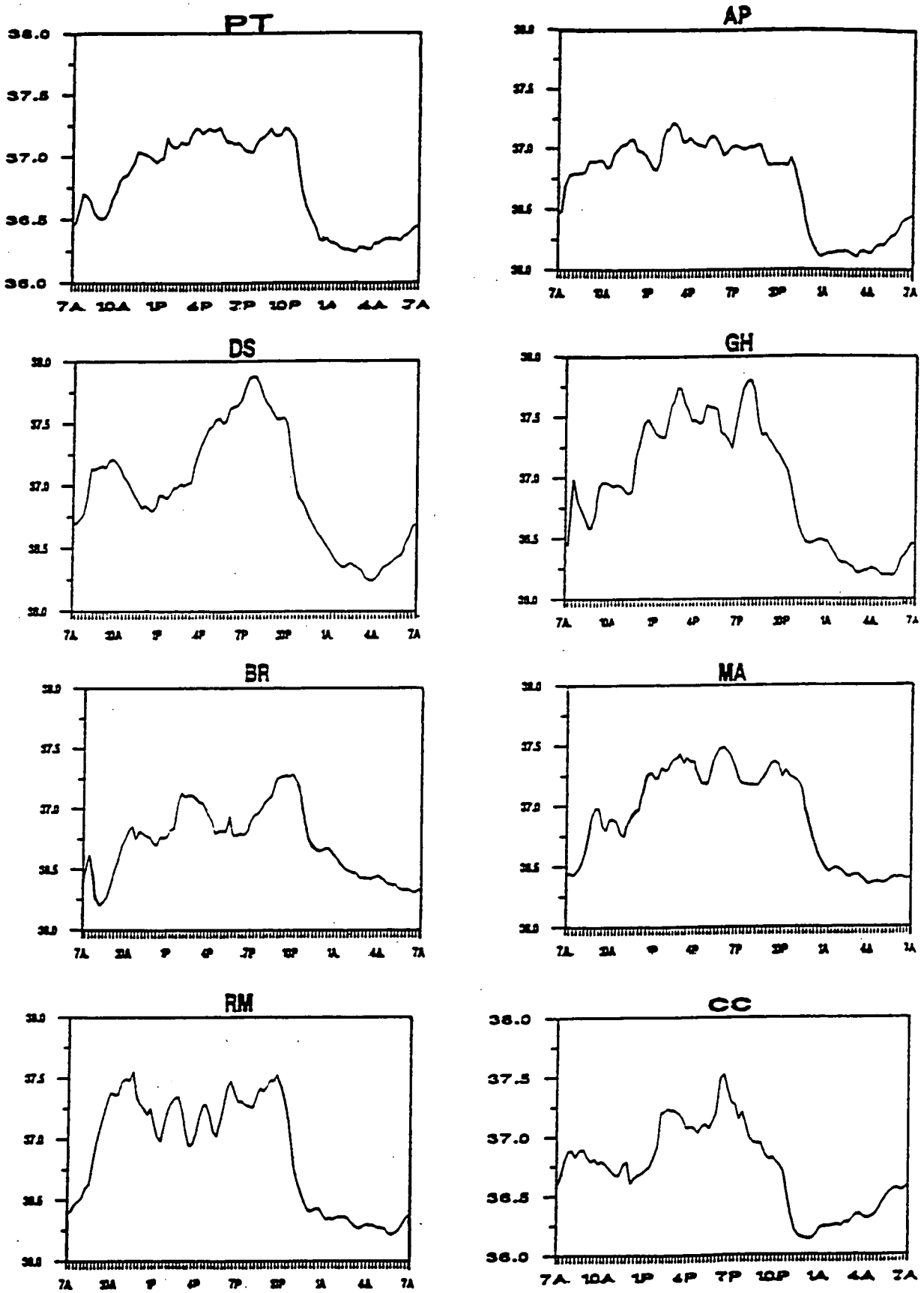

Figure D-1: Athlete Group

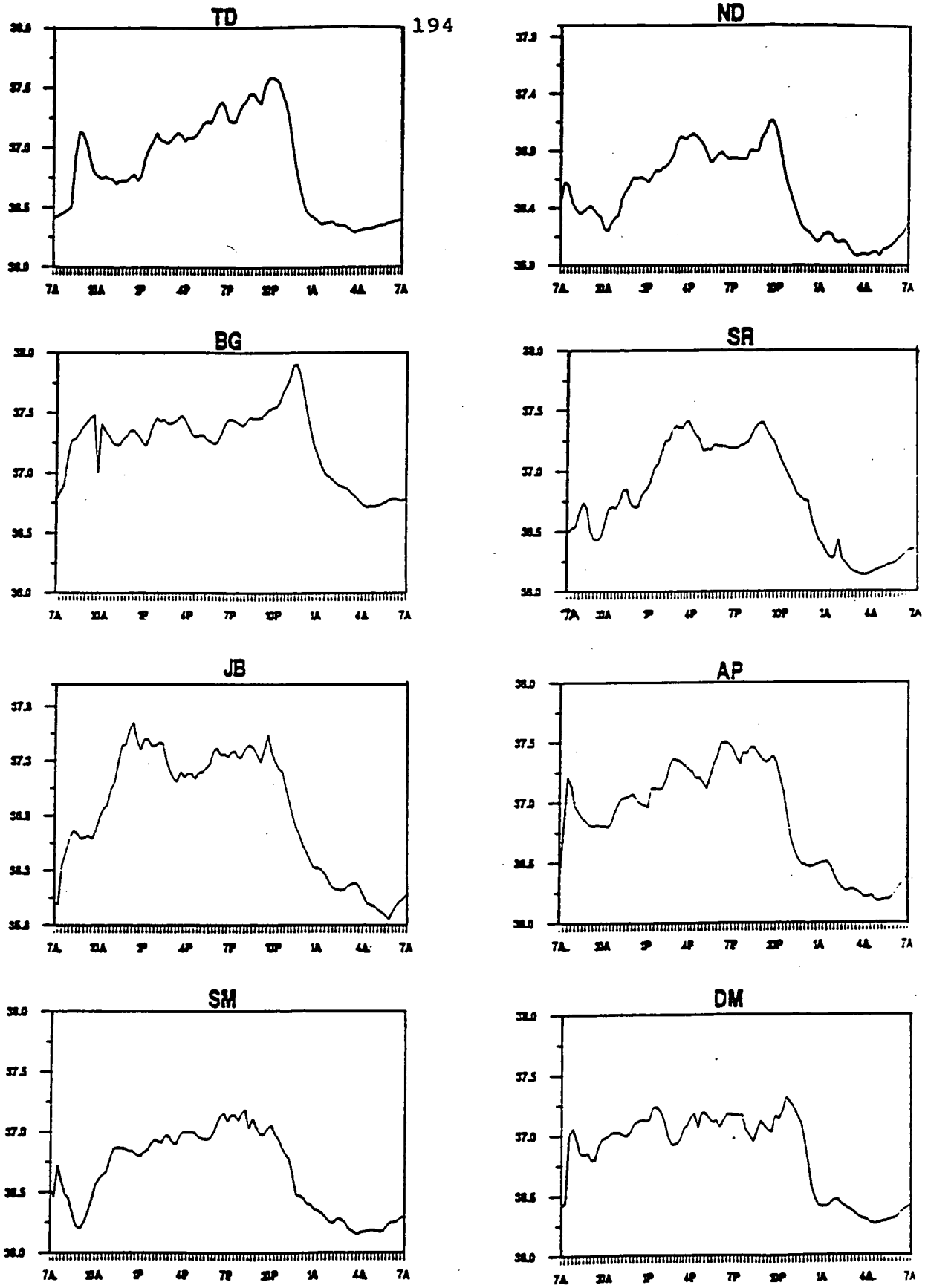

Figure D-2: Sedentary Group