Satisfying a need for sleep

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Satisfying A Need For Sleep

by

YVONNE HARRISON

A Doctoral Thesis

Submitted in partial fulfilment of the requirements for the award of Doctor of Philosophy of the Loughborough University of Technology

April 1995

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Dedication

This thesis is dedicated to my husband, Chris.
Recent studies have suggested that the average individual has a sleep requirement in excess of 8h sleep each night. These concerns stem from reports of relatively increased propensity for sleep throughout the day for otherwise apparently healthy, young adults. It has been claimed that a substantial proportion of these individuals are suffering from a chronic loss of sleep. The work presented in this thesis focuses on two key issues: (i) the adequacy of current social norms of sleep behaviour, and (ii) the assumption that an increased propensity for sleep throughout the day is the single most reliable consequence of a failure to satisfy a physiological need for sleep.

Throughout the first experiment the potential benefits of sleeping for as much as 10h per night were explored. During 26 consecutive nights EEG recordings and/or actigraphs were used to monitor the night-time sleep of 10 asymptomatic regular sleepers (mean 23.6y). The schedule comprised: 7 nights of BASELINE sleep, 14 nights of EXTENDED sleep (up to 10h/night), and 5 nights of RECOVERY sleep. During EXTENDED sleep subjects slept significantly longer (approx. 1h) but sleep latency and interim wakefulness deteriorated. EXTENDED sleep produced no improvements to self-rated mood or subjective sleepiness. Vigilance tests showed a small but significant reduction in reaction time following EXTENDED sleep compared with both BASELINE and RECOVERY nights. Ability to detect target tones did not change significantly. An objective measure of daytime sleepiness - the Multiple Sleep Latency Test (MSLT) - showed small (approx 1 min) reductions during EXTENDED sleep. These findings give little support to the view of chronic sleep deprivation in the average 7.5 h sleeper.

The second section includes evidence of a number of circumstances in which sleep can occur in alert subjects who otherwise showed no indication of sleepiness related impairment: (i) by using non-conventional scoring criteria throughout MSLT trials, sleep was found to occur as short bursts, or microsleeps, in non-arousing situations (ii) in response to a motivational incentive, and (iii) as MSLT defined pathological sleepiness unrelated to sleep-sensitive performance tasks, subjective sleepiness, or prior sleep behaviour. It is concluded that the capacity for more sleep at night, and the ability to fall asleep quickly during the day, are not systematically related to a physiological need for sleep for many healthy, regular sleeping young adults.
Acknowledgements

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The 8 Hour Sleep
SATISFYING A NEED FOR SLEEP

1.1 INTRODUCTION AND OVERALL THEMES

The question of "how much sleep do we need?" has attracted the attention of sleep researchers for at least six decades, perhaps because of the implicit suggestion that in being able to answer it some insight might be gained into the primary causes and functions of sleep itself. For early researchers, quantifying a need for sleep was approached by asking "how little sleep do we need?" and is reflected in many, now famous studies of long-term sleep loss, in which well-motivated individuals deprived themselves of all sleep over extended periods. The most impressive of these, and perhaps the most frequently quoted, refers to a successful attempt in 1965 by a healthy, young man known as Randy Gardner to remain awake for a continuous period of over 11 days (264 hours). In describing this event, Dement (1972) pointed to the apparent lack of physical or psychological distress throughout, speculating that further, perhaps 'indefinite' sleep loss would be possible for this particular well motivated and physically healthy individual.

There have also been many attempts to determine the boundaries of human sleep need in terms of tolerance to extreme deviations from normal sleep patterns. Rather than eradicate all sleep, this approach has highlighted the complex interactions between endogenous and exogenous factors influential in the placement and duration of sleep, and in particular, in determining its recuperative value. Early methodological techniques, such as the forced entrainment to artificial sleep/wake schedules throughout temporal isolation used by Kleitman & Richardson in the 1930s, remain popular today in their contemporary formats, e.g free-running environments, constant routines, and polyphasic sleep/wake schedules.

Much of this work was principally concerned to show how little sleep an individual could endure without serious risk, whether over the long- or short-term, and has led to a number of competing ideas which appear to be at odds with a more culturally determined view of sleep need as somewhere in the region of 8 hours per night for the average individual. However, throughout these earlier studies, being able to maintain adequate levels of functioning comparable with pre-existing levels did not
appear to be anticipated in the event of such extreme changes to sleep behaviour -
the emphasis was more firmly placed on whether or not such changes could be
maintained without serious or permanent injury to the individual.

Recent attempts to quantify the effects of sleep have incorporated a more pragmatic
approach to a need for sleep such that they have been primarily concerned to show
functional consequences during wakefulness of relatively small changes to a routine
sleeping pattern. This has necessitated a clearer distinction between the subjective,
physiological and functional effects of sleep during waking activity. It might be
argued that the reasons for a shift in focus away from the effects of extreme sleep
patterns towards relatively minor change reflects an increased awareness of, and
need to predict, everyday consequences of modern living. These might include an
increased reliance on artificial lighting, shift-work, trans-global travel and
communications, or in effect, any feature of industrialised societies likely to act as
an imperative to avoid sleep.

Throughout this study I have approached the problem of how to satisfy a need for
sleep, and in particular, recent suggestions that many individuals fail to achieve this
on a regular daily 8 hour schedule of sleep. There are two related themes : (i) the
first focuses on the question of a sleep need and the adequacy of generalisable
guidelines for the satiation of that need (ii) the second explores the assumption that
an increased level of sleepiness throughout the day is the single most reliable
consequence of a failure to satisfy a need for sleep. I have approached these issues
by asking whether more sleep at night will lead to measurable improvements in the
ability to maintain alertness throughout the following day, and by questioning the
assumption that reduced alertness throughout the day is necessarily related to recent
inadequacies in sleep behaviour. For this purpose I have evaluated my findings with
reference to contemporary conceptual and methodological approaches to the
measurement of sleepiness.

1.2 Why measure sleep need ?

There has been increasing concern over recent years that insufficient sleep at night
will result in considerable risk to the individual in terms of personal safety and
individual fulfilment ( Dement & Mitler, 1993) . Without such concerns the amount
of sleep an individual takes would be largely left to personal choice. However,
interest in these matters has accumulated at both a popular and scientific level to the
point where many occupational, industrial, government and health agencies now all
claim to have a legitimate interest in trends of sleep behaviour throughout the
general population.

Underlying this interest is the belief that less sleep at night will lead to a reduction in
our ability to perform tasks which are both physically and cognitively demanding.
This includes the ability to operate machinery, to drive vehicles, to concentrate over
long periods, to learn and to remember, and to interact socially etc throughout the
subsequent periods of wakefulness. Furthermore, that this is true for differences in
sleep length over time within the same individual and for differences in sleep length
between individuals. On an intuitive level, many individuals have personal
experience of sleep disturbance in the short-term, perhaps a series of late-nights, in
association with some or all of the above deficits, and this might seem to lend
support to a general view that more sleep is better than less. However, more
systematic approaches to the consequences of reduced sleep have not been
supportive of such 'common-sense' type beliefs. In order to examine the evidence for
such assumptions two basic issues can be identified (i) whether or not individuals
who regularly sleep for relatively shorter periods are more vulnerable to the
characteristics related with insufficient sleep, i.e. increased sleepiness throughout the
day, reduced capacity to perform etc. and (ii) the ability to tolerate reductions of
sleep and its effects on subsequent waking functions.

1.3 Differences between 'long' and 'short' sleepers

Studies of regular 'long' and 'short' sleepers have failed to provide evidence of
consistent differences between the two groups along the lines of functional capacity
throughout waking activity. Webb & Friel (1971) compared groups of self-defined
' short' sleepers (<5.5h per night) and 'long' sleepers (>9.5h per night). For a selected
number of subjects, overnight EEG recordings were similar for both long and short
sleepers in all but the length of time spent in 'light' sleep, i.e. stages 1 and 2 sleep. In
this respect, short sleepers were described as having more 'efficient' sleep due to the
reduced amount of time spent either awake or in light sleep throughout the total
sleep period. No differences were found between long and short sleepers in terms of
personality, depressive state, educational record, or reported illness. These authors
concluded that, for their subjects, there was no apparent detrimental effect of either
extremely short or long sleep on a regular basis. However, the question of
personality differences and regular nightly sleep length remains unresolved.
Hartmann, Baekeland, Zwilling et al (1972) reported findings of increased depression among people who slept longer than average out of personal choice. They argued that long sleepers showed signs of increased neuroticism and anxiety compared with short sleepers and described long sleepers as "worriers". These authors emphasised the volitional aspect of differences in sleep duration and suggest that for the long sleeper, this additional sleep may occur as a response to the above psychological factors. In this sense, the personality differences are thought to lead to the extreme sleep patterns, rather than the converse in which the characteristics of excessive worrying and anxiety are consequences of a prolonged period of sleep. However, prior to this, Hicks and Pelligrini (1971) found more anxiety amongst short, rather than long, sleepers. Whilst reports from even more studies were in agreement with Webb & Friel (1971), in that they failed to find significant differences between short and long sleepers, e.g. Buela-Casal (1984) and Buela-Casal & Valle-Inclan (1984), and Buela-Casal, Carlos-Sierra & Caballo (1992).

When Taub (1976) compared 'long' sleepers (>9.5h) with those who regularly slept for an average duration of 7.5h-8.5h he found differences between the two groups along measures of performance and mood. EEG recordings were used to verify differences in sleep length - this amounted to an average increase of 109 minutes total sleep time during a single night for the group of long sleepers. Scores during prolonged (45 min) Wilkinson Auditory Vigilance testing averaged across three daily testing sessions showed long sleepers to miss fewer target tones in all, and to have faster reaction times than subjects in the comparison group. In addition to this, long sleepers were found to have lower self-rated scores on scales of depression and anger, whilst also showing increased cheerfulness, energy and activation. In interpreting these findings Taub (1976) suggested that there may be some individuals for whom a regular daily sleep allowance of around 8 hour is insufficient to maintain optimal levels of functioning, and that increased performance levels for the long sleepers in his study is evidence of the potentially beneficial effect of sleeping longer at night.

Johnson & Spinweber (1983) tackled the issue of sleep and occupational efficiency in their study of self-defined 'good' and 'poor' sleepers among American Navy personnel. The aim of this study was to monitor the relative success of individual recruits in terms of their actual experience of sleep on a regular basis. For this purpose almost 3,000 predominantly male subjects were studied over a 6-year period. Subjects were identified as either good or poor sleepers on the basis of their response to an item included in a sleep questionnaire. Individuals were defined as
'poor' sleepers if they rated their overall sleep to be either 'poor' or 'very poor', whilst 'good' sleepers were identified as rating their sleep to be either 'good' or 'very good' overall. Of the original sample only approximately 35% who rated themselves to be 'average' sleepers were excluded from further comparisons.

This approach was different to previous studies in that the duration of sleep was not actually specified as a defining feature of good or poor sleep, although it was considered to be of interest in further confirmation and analysis of the two groups. However, unlike the findings of Webb & Friel (1971) for example, whose final sample of 'long' and 'short' sleepers comprised less than 5% of the original population sampled, Johnson & Spinweber (1983) separated their groups using less extreme criteria, and consequently included a far greater number of individuals who were likely to deviate less from the average in their overall pattern of sleep than those of previous studies of individual differences in sleep duration.

The analysis performed by Johnson & Spinweber (1983) explored differences between objectively and self-defined good and poor sleepers with reference to the amount and quality of sleep experienced at night. This was then related to short-term performance using short-duration psychometric tests throughout the day, and overall performance in terms of positive career measures throughout the duration of the study. Apart from the obvious issues of individual safety and national security, the benefits of being able to predict the likely outcome of recruitment on the basis of an individual's sleeping habits, or at least to be able to contribute reliably to that prediction, for an organisation on this scale are likely to be far-reaching.

Subjects associated the following with the experience of being a poor sleeper: difficulty initiating sleep, intermittent waking during sleep, more easily disturbed by external events, i.e. light sleepers, nightmares, failing to feel well rested in the morning, difficulty getting up in the morning, and feeling comparatively more sleepy throughout the day. In contrast, good sleepers reported taking less than 20 minutes to fall asleep at night (poor sleepers estimated a sleep latency of greater than 60 min). Total sleep time during workdays was reported to be 7-8 hours for good sleepers compared with 5-7 hours for poor sleepers. However, both groups claimed to extend their sleep on days off to around 8-9 hours.

There was a significant difference between poor and good sleepers and self-reported mood using the Profile of Mood States (POMS) questionnaire to estimate mood throughout the day. Poor sleepers experienced more tension, depression, anger,
vigor, fatigue and confusion than good sleepers. Johnson & Spinweber (1983) were able to reproduce this finding of increased negative mood for a small sample of subjects whose reported sleep was reliably qualified using overnight EEG recording.

Another interesting finding to emerge from this study was the discrepancy between perceived and objectively measured poor sleep. Overnight EEGs recorded to objectively monitor the sleep of a sample of subjects showed, for some self-defined poor sleepers (over 60% of this sub-sample), a shorter latency to sleep onset than was generally claimed to occur. For all other sleep measures, poor sleepers with both shortened and prolonged sleep latencies were found to be the same. Only objectively defined poor sleepers differed from good sleepers in terms of actual sleep length. Subjective poor sleepers who failed to display extended sleep latencies during objective recordings were not found to differ from self-defined and objectively verified good sleepers along any objective measure of sleep. For these subjects, only subjective reports differentiated them from good sleepers.

In terms of employment record, health and general factors, significant differences were found between good and poor sleepers which led Johnson & Spinweber (1983) to describe poor sleepers overall as "less effective sailors". Throughout the six year course of the study, poor sleepers were found to have gained fewer promotions than good sleepers, were subsequently less well paid, were less likely to be recommended for re-enlistment and were more likely to leave the service. In addition to this, poor sleepers were more likely to experience health difficulties requiring a greater number of repeated hospitalizations than good sleepers.

On the face of it, this might appear to provide strong evidence for discrimination at the recruitment stage in favour of good rather than poor sleepers. However Johnson & Spinweber (1983) outlined three possible explanations for these apparent differences between poor and good sleepers. The first was that poor sleepers were actually getting less sleep than they needed, unlike good sleepers who subsequently performed better than their poor sleeping counterparts. Were this the case, as Johnson & Spinweber (1983) pointed out, then rather than sleep for an equivalent time on non-duty days, the poor sleeper would have been expected to take advantage of the opportunity for extra sleep by sleeping longer than the less deprived good sleepers. In addition, over 60% of reported poor sleepers were not found to sleep less than good sleepers when objective measures of sleep were used. However, these subjects actually believed themselves to be getting less sleep which,
because of the general expectations of requiring a minimum amount of sleep, may have encouraged them to expect the consequences associated with insufficient sleep. The second possibility was that the sleep of poor sleepers was of an inferior quality to that of good sleepers. Although Johnson & Spinweber (1983) stated that they found little evidence of this in terms of conventional sleep measures, and that apart from sleep latency, the sleep of both good and poor sleepers was very similar, the subjective reports of poor sleepers indicated a reduced level of sleep efficiency (reporting more external disturbances etc) for this group.

The third explanation, and the one favoured by Johnson & Spinweber (1983) suggested that poor sleep is associated with chronic psychological difficulties. For the sailors in this study, the development of sleep problems was indicative of the level of adjustment (or lack of it) to a novel set of circumstances. In their view, poor sleep developed in the absence of satisfactory coping skills. This might explain why the earlier records for the subjects involved in the study and objectively determined aptitude for naval recruitment failed to be predictive of future success. For Johnson & Spinweber (1983), persistent poor sleep, whether verified as such using objective measures, is symptomatic of an underlying psychological state. Therefore, it is not simply a question of how much sleep leads to optimal functioning, or whether one sleep length is superior in this effect than another, but more relevant to consider the factors responsible for the regular pattern of sleep in an individual.

In summary, it remains unclear as to whether there are any genuine differences between long or short sleepers or whether these individuals merely occupy extreme positions along a normally distributed pattern of sleep duration throughout the general population, without obvious detrimental effects to their well-being. The preference for either long or short sleep has not been consistently associated with psychological states, or personality types, although the volitional nature of sleep duration in response to these factors is important. However, it is also likely that psychological distress, such as depression or anxiety, will elicit different responses, in terms of changing sleep habits, between individuals.

1.4 Tolerating change in sleep patterns

Common sense dictates that sleep patterns can be adapted to incorporate the changing demands of work, social and domestic commitments, e.g. to suit the feeding schedule of a new baby, or revise for examinations, and that on the whole,
most individuals are able to tolerate a deviation from a normal pattern of sleep under such circumstances. This suggests considerable flexibility in a requirement for sleep in which fluctuations between a hypothetical physiological sleep requirement and the amounts of expected sleep (habitual sleep) and actual sleep are easily accommodated. It has been argued that, within limits, the overall effects of a sustained reduction in sleep are negligible (Webb & Agnew, 1975; Horne & Wilkinson, 1988).

There have been a number of attempts to determine the boundaries of a need for sleep, especially the lower limits. Whilst an acute loss of sleep for one or two nights has repeatedly been shown to produce substantial deficits in performance capacity coinciding with a sharp increase in subjective sleepiness (Carskadon & Dement, 1979; 1982), the study of chronic sleep reduction has provided evidence which is generally supportive of an individual's ability to tolerate long-term, gradual reductions of daily sleep (e.g. Rutenfranz, Aschoff & Mann, 1972; Webb & Agnew, 1974; Noles, Epstein & Jones, 1976; Friedman, Globus, Huntley et al, 1977; Horne & Wilkinson, 1988). The converse of this, the ability to take more than regular amounts of sleep has also been shown to be possible, although investigations are less numerous and have tended to concentrate on the extension of sleep over the short-rather than long-term (e.g. Carskadon & Dement, 1979, 1981; Roehrs, Timms, Zwyghuizen-Doorenbos et al, 1989). At present the only exceptions to this in which sleep was extended for a relatively longer period are the studies of Wehr, Moul, Barbato et al (1993) and Roehrs, Shore, Papineau et al (1994).

The amount of sleep taken during these study periods was considered to be detrimental to the well-being of the individual if there was found to be a relative deficit along measures of performance, subjective sleepiness or mood, and more recently, the tendency to fall asleep during the day in an environment conducive to sleep using the Multiple Sleep Latency Test (MSLT). The general consensus is that most individuals are able to tolerate a limited reduction to sleep over the long-term when the change to an habitual sleeping pattern is introduced gradually, preferably as a series of interim reductions (Webb & Agnew, 1974; Horne & Wilkinson, 1988). Webb & Agnew (1974) imposed a 5.5h sleep restriction on 15 adult male subjects for 60 nights. Regular overnight EEGs provided a record of change in actual sleep. In line with a previous study of sleep reduction which focused on change in sleep structure (Webb & Agnew, 1965), there was an increase in the amount of stage 4 sleep and a sharp reduction in REM sleep during reduced sleep (Webb & Agnew, 1974). Despite changes to sleep duration and architecture, Webb & Agnew (1974)
concluded that for a sleep duration of 5.5h over a 60 day period "the behavioural consequences were limited". Horne & Wilkinson (1988) added general support for this finding. Throughout this study (Horne & Wilkinson, 1988) the sleep of 12 regular, 7.5-8.0h sleepers was gradually reduced to an average of 6 h per night over a period of 42 nights. At this level of reduction overnight EEG recordings showed no significant change in the amount of stage 3 or stage 4 sleep. REM sleep and stage 2 sleep was significantly reduced during sleep reduction. In addition to this, sleep onset latency was shortened and intermittent wakefulness during sleep reduced. No overall increase in daytime sleepiness was found despite a 2 hour reduction of sleep for these subjects. Subjective reports indicated that a 6 h sleep duration was well tolerated (Horne & Wilkinson, 1988).

However, this does not account for the apparent increase in MSLT defined daytime sleepiness when sleep was reduced to 5 hours per night over a period of seven nights (Carskadon & Dement, 1981). The apparent discrepancy between a cumulative increase in sleepiness (which had not been shown to reach an asymptote after the seventh night of sleep reduction - Carskadon & Dement, 1981) and the ability to preserve baseline levels of performance capacity over relatively longer periods (Webb & Agnew, 1974; Horne & Wilkinson, 1988) raises the issue of how best to assess the consequences of sufficient (or insufficient) sleep. This will be addressed in Chapter 2 - What is sleepiness?

Table 1.1 gives a summary of chronic sleep reduction studies continuing for at least seven nights. Reports of negative subjective experience, performance deficit, and increased levels of objective sleepiness using the MSLT during the novel restricted sleep regime are included.

Subjective objections to even severe sleep regimes were relatively uncommon: only Blagrove, Alexander & Horne (1995) reported subjects' unwillingness to further reduce sleep, or continue with the final level of reduced sleep when a mean sleep duration of 5.3 hours had been reached. On the whole, increased levels of subjective sleepiness during sleep reduction were reported as either transient (Webb & Agnew, 1974) or negligible (Horne & Wilkinson, 1985). Noles et al (1976) reported subjective approval of increased wakefulness. Follow up measures indicated that, for some subjects, a substantial reduction of overall sleep duration from previous (pre-study) levels was maintained (Johnson & Macleod, 1973; Friedman et al, 1977, Horne & Wilkinson, 1988). Subjective or objective measures indicated a shortened sleep latency during reduced sleep (Webb & Agnew, 1974, Horne & Wilkinson,
In addition to this, early morning awakening proved problematic for some subjects on a restricted sleep schedule (Webb & Agnew, 1974).

<table>
<thead>
<tr>
<th>study</th>
<th>sleep duration (h)</th>
<th>study period (nights)</th>
<th>subjective complaints?</th>
<th>performance deficits?</th>
<th>MSLT-increased sleepiness?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Webb &amp; Agnew 1965</td>
<td>3</td>
<td>7</td>
<td>n/a</td>
<td>yes</td>
<td>n/a</td>
</tr>
<tr>
<td>Rutenfranz, Aschoff &amp; Mann 1972</td>
<td>5</td>
<td>&gt;90</td>
<td>n/a</td>
<td>no</td>
<td>n/a</td>
</tr>
<tr>
<td>Johnson &amp; Macleod 1973</td>
<td>4</td>
<td>21¹</td>
<td>yes</td>
<td>yes</td>
<td>n/a</td>
</tr>
<tr>
<td>Webb &amp; Agnew 1974</td>
<td>5.5</td>
<td>60</td>
<td>no</td>
<td>yes²</td>
<td>n/a</td>
</tr>
<tr>
<td>Noles, Epstein &amp; Jones 1976</td>
<td>6.5</td>
<td>n/a³</td>
<td>no</td>
<td>no</td>
<td>n/a</td>
</tr>
<tr>
<td>Friedman, Globus, Huntley, Mullaney, Naitoh &amp; Johnson 1977</td>
<td>4.5-5.0</td>
<td>28⁴</td>
<td>yes</td>
<td>no</td>
<td>n/a</td>
</tr>
<tr>
<td>Horne &amp; Wilkinson 1985</td>
<td>6</td>
<td>42</td>
<td>no</td>
<td>no</td>
<td>n/a</td>
</tr>
<tr>
<td>Carskadon &amp; Dement 1981</td>
<td>5</td>
<td>7</td>
<td>yes</td>
<td>n/a</td>
<td>yes</td>
</tr>
<tr>
<td>Blagrove, Alexander &amp; Horne 1995</td>
<td>(a) 5.2</td>
<td>28</td>
<td>yes</td>
<td>no</td>
<td>n/a</td>
</tr>
<tr>
<td></td>
<td>(b) 5.3</td>
<td>18</td>
<td>yes</td>
<td>no⁵</td>
<td>n/a</td>
</tr>
</tbody>
</table>

Table 1.1 Studies of chronic sleep reduction

1 this study lasted for a total of 5 months, during which subjects gradually reduced their sleep by an additional 30 min each week - the final level of 4 h each night was maintained for the last 3 weeks of sleep reduction.

2 only one performance measure (HITS on WAVT) showed a deficit which Webb & Agnew attributed to motivation rather than an intrinsic capacity to perform.

3 details of time period for reduction of sleep were not reported

4 this study lasted between 6-8 months during which subjects gradually reduced their sleep by an additional 30 mins every 2-3 weeks - the final level of sleep reduction achieved by each subject was maintained for a further month.

5 whilst performance decrements were not found using tests of logical reasoning or auditory vigilance, decrements in an embedded figures test were found when sleep was reduced to this level.

Impaired performance following extreme sleep reduction was found in the studies of Webb & Agnew (1965) - 3 h sleep, Johnson & Macleod (1973) - 4h sleep, Webb & Agnew (1974) - 5.5h sleep (see note 2 above). Other than this, performance capacity remained at baseline levels throughout the various reductions in sleep (see Table 1.1). This prompted Horne & Wilkinson (1985) to describe sleep above a certain amount as a flexible requirement. As such they suggested that the acknowledged 8 h sleep norm is bordered by a 2 hour window of opportunity for more or less sleep without serious consequences. The analogy of eating and a basic requirement for food is provided to describe the apparent ease with which changes in sleep duration can be tolerated. Thus, a basic, or obligatory requirement for sleep
Ch 1: The 8 hour sleep

is satisfied during the earlier stages of the night. Following this, Horne & Wilkinson (1985) describe the remaining portion of the night as a period for "flexible and facultative" sleep. Horne (1988) expands on this by contrasting the requirement for "core" and "optional" sleep. In this view, studies in which sleep is reduced below approx. 5.5 hours such as Webb & Agnew (1965) and Johnson & Macleod (1973) are likely to have exceeded the limits of "optional" sleep for their subjects, resulting in a deprivation of the more essential "core" sleep and subsequent deficits in performance.

This apparent flexibility in an individual's pattern of sleep is also evident as the capacity to take more sleep over the short- (Carskadon & Dement, 1979, 1981) and long-term (Wehr et al, 1993; Roehrs et al, 1994), although this is frequently interpreted as evidence of a pre-existing sleep debt. Details of the two long-term studies in which the consequences of extended sleep were investigated are summarised in table 1.2.

<table>
<thead>
<tr>
<th>study</th>
<th>time in bed (h)</th>
<th>study period (nights)</th>
<th>subjective improvements</th>
<th>performance improvements</th>
<th>MSLT-reduced sleepiness?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wehr, Moul, Barbato et al 1993</td>
<td>14h</td>
<td>28</td>
<td>yes</td>
<td>n/a</td>
<td>n/a</td>
</tr>
<tr>
<td>Rochrs et al 1994</td>
<td>10h</td>
<td>14</td>
<td>n/a</td>
<td>n/a</td>
<td>yes</td>
</tr>
</tbody>
</table>

**Table 1.2 Studies of chronic sleep extension**

Rather than an obligatory requirement for sleep of around 6 hours (Horne, 1988), the authors of the above studies of sleep extension interpret improvements following sleep extension as evidence of a sleep requirement in excess of the usual 8 hour sleep. One reason for this renewed interest in the optimal requirement for sleep and the apparently contradictory findings stems from recent advances in the conceptualisation of daytime sleepiness, and the development of appropriate tools for its measurement. For example, since the early eighties, a number of studies have focused on changes in the MSLT as a measure of daytime sleepiness following various lengths of sleep at night (Carskadon & Dement, 1979, 1982; Roehrs et al, 1989, 1994). These researchers have been less inclined to support earlier conclusions reached by, for example, Webb & Agnew (1975) and Horne & Wilkinson (1988) regarding a nominal change in daytime functioning when habitual sleep duration is reduced by up to approx. 2 hours. Clearly, it is significant that when the results of MSLT findings were prioritised, deficits in daytime sleepiness following a 5h schedule for 7 nights were found (Carskadon & Dement, 1981).
However, the interpretation of these findings in terms of everyday functioning has yet to be reconciled with performance based studies of sleep reduction which point towards an apparent flexibility in sleep patterns within limits.

It is therefore important to establish the relevance to everyday tasks of a change in a tendency for sleep. It is possible that, despite an increased tendency for sleep during the MSLT following a 2h sleep reduction, the actual effects on occupational tasks are nevertheless negligible. In this respect, it is necessary to be able to generalise from the laboratory to the conditions of a normal working environment. However, a gradual and controlled reduction of sleep over time in the laboratory is unlikely to mirror the general lack of regularity in sleep patterns associated with occupational areas which have attracted the greatest concern regarding the potential risks of insufficient sleep.

This is highlighted in the case of junior hospital doctors, for example, where recent calls for a reduction in working hours have been fuelled by a concern for a loss of efficiency during the working period as a direct result of lack of sleep (Durnford, 1988; Editorial - The Lancet, 1988). Despite these growing concerns for the safety of patients in circumstances in which doctors frequently work for a continuous period in excess of 16 hours (Durnford, 1988), actual performance deficits using tasks sensitive to sleep loss have not been consistently found. For example, Deaconson, O'Hair, Levy et al (1988) assessed hospital doctors following reduced sleep and concluded that there were no effects of sleep deprivation on the performance tasks used, although in this study financial incentives were offered (see below). Earlier, Poulton, Hunt, Carpenter et al (1978) had shown that junior doctors were capable of maintaining high performance until they reached a sleep debt of 8 hours (i.e. 8h less than their individual average sleep length over a 24 h period - for some this is basically a sleepless night).

The ability to maintain performance levels throughout prolonged and sporadic periods of sleep deprivation under genuine 'real-life' circumstances such as this reflects an essential difference between the ease of falling asleep during the MSLT and other sleepiness related measures. In particular, it has been shown that the maintenance of performance following sleep loss is largely a question of attitude and motivation (Horne & Pettitt, 1985). To assume a direct relationship between sleep duration and performance is therefore to ignore the relevance of a task to a given situation. This is a key issue in evaluating the effects of sleep loss as either performance deficits or increased sleep tendency. For the MSLT, the subjects' role
In countering potentially undesirable effects of sleep loss is largely ignored, yet using performance tasks this has been shown to be central in determining the actual consequences of reduced sleep (Horne & Pettitt, 1985). It is because of the importance of this factor that it has not been possible to predict, for example, whether or not a reduced physiological sleep tendency found using the MSLT will necessarily lead to inadvertent sleep onset.

In the laboratory, it has been shown that the effects of acute sleep loss are minimized through compensatory effort, and that this can be increased through incentive. Horne & Pettitt (1985) reported that subjects were able to preserve baseline levels of performance throughout a 30 min auditory vigilance task for the first 36 hours of total sleep deprivation. Thereafter, performance was significantly reduced from baseline despite the financial incentive, although it remained significantly better than for a comparison group who had endured the same length of sleep deprivation without additional incentives to perform. Even the actual tendency for sleep, assumed to be free from subjective motivation (Carsadon & Dement, 1982) has been shown to be reduced when the incentive to avoid sleep is increased (Alexander, Blagrove & Horne, 1988). Alexander et al (1988) compared two groups of subjects under equivalent conditions of acute sleep deprivation. Both groups were encouraged to resist sleep onset during repeated tests of sleep latency using an MSLT variant although an additional financial incentive was included in the instructions for one group. This was found to produce significant differences (reduced sleep tendency) for the group offered an incentive to try harder to resist sleep.

This capacity to combat extreme tiredness is likely to be an important feature in maintaining performance in the case of junior doctors etc., where the incentives to perform well are particularly high. In assessing comparative differences between sleep length, studies which are primarily based on MSLT findings are specifically directed towards a single aspect of daytime functioning. This has been described as an underlying physiological level of sleepiness, which is assumed to be an objective and reliable indication of status along the sleepiness/alertness continuum (Carskadon & Dement, 1982). Yet it has repeatedly been shown that the consequences of a chronic change to habitual sleep patterns are minimal using alternative tests (Webb & Agnew, 1975; Horne & Wilkinson, 1985).
To summarize, it has been shown that a reduction of nightly sleep by approx. 2 hours over the long-term can be accommodated relatively easily. The effects of this on daytime functioning are modest when measured using performance related or subjective measures, although impairment is more likely with increased reduction. Daytime sleepiness using the MSLT has been shown to be systematically related to sleep duration at night (Carskadon & Dement, 1982) although the relationship between the MSLT and other measures of sleepiness is unreliable. Impaired or improved performance following sleep reduction and extension, respectively, are not guaranteed despite MSLT changes.

It is more likely that the ability to adapt to circumstances necessitating a change in sleep duration and commonly observed differences in preferred sleep length reflect a basic flexibility in sleep need, whereby at any one point an individual can be described in terms of their position relative to popular sleep length and their own habitual sleep length. Current evidence suggests that most individuals are able to adapt to a reduction in sleep duration of around 2 hours with little noticeable effect. The factors relating to why a person sleeps for a certain amount at a particular point in time are arguably more influential in determining their ability to cope with this amount than any hypothetical notion of sleep satiation.

1.5 The 8 hour sleep

Spending approximately 8 hours of each 24 hour period asleep is commonly understood to represent a culturally determined norm widespread throughout western, industrialised societies. In terms of the amount of sleep the average individual requires the ideal of 8 hours sleep is arguably a relatively recent development. Webb and Agnew (1975) put forward the idea that dedicating as little as 8 hours for sleep each night might incur a chronic sleep deficit for some individuals. This is inferred from archival evidence of increased nightly self-reported sleep from the 1930's, the observed capacity for extra sleep by an average of 126 min when given the opportunity to spontaneously oversleep, longer sleep on the weekends compared to weekday nights, a reliance on alarm clocks, and complaints of residual tiredness on waking.
1.6 How much do people sleep?

The measurement of sleep routines in the general population is problematic; simply asking people how much they sleep is subject to a number of confounding variables likely to lead to error. The ability to estimate sleep times accurately has been found to be reduced for poor sleepers (Bixler, Kales, Leo & Slye, 1973; Carskadon, Dement and Mitler et al, 1976) and with certain psychological states, e.g. depression and high anxiety (Bliwise, Friedman & Yesavage, 1993). However, a number of medium- to large-scale surveys have offered some insight into popular support for the notion that 8 hours sleep represents sufficient sleep each night, but also into the range of factors likely to influence the amount of sleep an individual might choose to sleep at any given time. These include social, environmental, genetic, psychological, physiological and maturational considerations which result in considerable intra- and inter-individual variability in the duration and structure of sleep in any 24-hour period.

The importance of this variability has been questioned with respect to related health issues. Two well-known epidemiological studies reported similar findings of an association between extreme sleep duration and increased rates of mortality. One of these, a questionnaire based survey relating lifestyle with health and mortality was conducted throughout the Alameda County, California, USA in 1965. A sample of county residents over the age of 16 was contacted for this purpose yielding an eventual response rate of over 86% (6,928 respondents). Of specific interest were the number of hours slept each night, eating habits, exercise, smoking and alcohol intake. Belloc & Breslow (1972) reported an association between the pattern of these behaviours and physical health status, such that reports of positive aspects of each factor were related with an increased health status. In terms of nightly sleep durations, 5,290 individuals reported sleeping between 7-8 hours per night. This was related to increased health in comparison with individuals sleeping less or more than these amounts. However, as these authors point out, a more sophisticated understanding of the relationship between apparent health and lifestyle was beyond the scope of this type of survey. The reasons for adopting particular lifestyle practices, including sleep patterns, are likely to be complex and the possibility that the habit of sleeping for longer or shorter amounts than average may be brought on by a state of poor health cannot be discounted.

This issue is also relevant to further analysis of this data by Wingard & Berkman (1983). A nine year follow-up of the original respondents from the Alameda
County survey allowed a breakdown of subsequent mortality rates. Habitual sleep of 6 hours or less or 9 hours or more was associated with 1.7 times the likelihood of a death occurring for men and 1.6 times for women.

Between 1959 and 1960 over 1 million Americans over the age of 30 responded to a questionnaire distributed by the American Cancer Society. Kripke, Simons, Garfinkel, and Hammond (1979) found a mean self-reported habitual sleep time of between 8-9 hours per night. A six year follow-up showed that men sleeping less than 4 hours per night were 1.3 times more likely to have died, whereas women sleeping less than 4 hours per night were 1.48 times more likely to have died. At the other extreme, both men and women sleeping more than 10 hours per night were 1.8 times more likely to have died. Whilst these authors concluded that any causal relationship between sleep length and health could not be established due to the limitations of the data collected, this study reinforces a popular belief in the adequacy of the 8 hour sleep for optimum individual performance (Kripke et al, 1979).

A more recent study of the sleep habits of 1,877 50-65 year old men and women conducted by Bliwise, King & Harris (1994) emphasised the limitations of the Alameda County and The American Cancer Society Studies. In addition to estimating sleep duration, subjects in this study were asked to supply extensive information regarding the features of sleep. Bliwise et al (1994) found that factors specific to sleep pathology, such as snoring and excessive daytime sleepiness, were more likely to be associated with an increased likelihood of ill-health rather than sleep duration alone. Furthermore, it was also found that self-reported long sleepers were more likely to report excessive snoring. These authors suggest that variability in self-reported sleep duration should not be assumed to reflect personal choice (Bliwise et al, 1994). Particularly for long sleepers, the influence of an underlying sleep-related disorder on extreme sleep durations may be an important factor in reported associations between sleep duration and mortality.

However, whilst both the Alameda County and the American Cancer Society offer some insight into the sleeping habits of the American population on a large scale, sampling for both surveys was heavily weighted towards individuals from the middle-aged to elderly population. Two recent European investigations were targeted towards a younger age group: Lavie (1981) concentrated on 1502 pre-retirement Israeli men and women over the age of 16 years, whilst Billiard (1987) surveyed over 58,000 male French army recruits between the ages of 17-22. Lavie
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(1981) reported an habitual sleep length throughout the weekday nights of an average of 6.5 hours compared with 7.5 hours at the weekend. It was also noted that approx 60% of the respondents reported regular bedtimes with over 50% preferring not to use an alarm clock in the morning, being able to rely instead on spontaneous awakening.

Billiard et al (1987) found that over 60% of the 58,000 respondents to a sleep survey questionnaire reported sleeping between 7-8 hours each night. Long sleep episodes, greater than 9 hours, were relatively popular (18.3%) and almost as likely as short sleep lengths, less than 7 hours (20.7%). No differences were found between the number of respondents reportedly sleeping between 7-8 hours per night and 8-9 hours per night.

Investigations on this scale, although relatively few, suggest that 8 hours sleep at night is a close approximation of the amount of sleep taken by the average individual. The relative effect of all factors known to influence sleep duration are important considerations in determining the origins of this apparent trend in behaviour.

1.6.1 Social influences on sleep

It has been suggested that limited opportunities for activity or social interaction during the night, coupled with a need to maintain alertness throughout the day, provide sufficient incentives to comply with socially defined norms of sleep/wake behaviours, confining sleep to a consolidated period throughout the night (Campbell, 1984; Wehr et al, 1993). Furthermore, this pattern of sleep/wake activity is understood to reflect socially imposed occupational and recreational necessities rather than a physiological drive for sleep. According to Webb & Agnew (1975), the commonly observed spontaneous lengthening of sleep times on weekends and holidays is evidence that such situational restrictions on sleep times affect many people throughout the working week. Webb & Agnew (1975) also found that when subjects were free from a requirement to get up in the morning they were able to sleep an average of 126 minutes extra when compared with their habitual sleep length. In a study of self-reported sleep times of over 1500 Israeli workers, Lavie (1981) also reported an increase of approx. 1 hour at weekends compared with weekday nights.
Wirz-Justice & Pringle (1987) provided a single-case study of a young, apparently healthy male student who followed an erratic and unstructured pattern of sleep/wake activity over a nine month period. Using a daily self-report dairy, this individual exhibited extremes of both short and long sleep episodes, favouring an average bedtime of around 0500h and rise time of 1200h. An abrupt advance in bedtimes was found to coincide with the start of preparations for final examinations which was immediately followed by a return to later bed-times and extremely long periods of continuous wake (upto 60h maximum). These authors concluded that a lack of common social enforcers, which they referred to as social zeitgebers, such as regular schedules of work and recreation allowed this individual the opportunity to maintain extreme patterns of sleep/wake activity quite different to the normal 24hour cycle preferred by the majority of the population. Wirz-Justice & Pringle (1987) liken this to the unusual patterns of sleep often found under conditions of temporal isolation. Under circumstances in which there are few requirements in terms of daytime activities and every opportunity to sleep when preferred, conventional night/day activity patterns are abandoned. For this reason, a preference for phase delayed sleep-onset and erratic sleep/wake cycles is believed to be more common in social groups experiencing fewer social and occupational pressures throughout the day, e.g. many young adults. This suggests that, for some individuals, social and environmental factors provide powerful determinants in the timing and duration of sleep.

The influence of these factors on the structuring of sleep wake patterns has been studied under experimental conditions. With the elimination of temporal and social clues, Monk & Moline (1984) found an association between the timing of decisions to go to bed and circadian rhythms of temperature and subjective alertness. Requests to go to bed were more likely to occur as both subjective alertness and temperature fell, whilst waketimes coincided with a rise in temperature and alertness. When sleep was initiated at times other than during the falling phase of temperature and alertness cycles this was found to be shorter in duration and included more intermittent wakefulness, i.e. was less efficient. Monk & Moline (1984) concluded that in the absence of external distractions, the timing of human sleep follows a predictable and cyclic pattern, in synchrony with certain endogenous circadian features. However, it should be noted that the subjects in this study were encouraged to follow a monophasic pattern of sleep throughout, with short naps positively discouraged. Thus the experimental design imposed restrictions to a subjective preference for sleep.
Earlier attempts to eliminate the influence of all external factors on the timing, duration and structure of sleep have resulted in similar difficulties. For example, Ryback & Lewis (1971) included regular and extensive performance testing throughout the daytime period in a long-term continuous bedrest study, hence confining all sleep episodes to the night-time period.

Campbell (1984) attempted to overcome these difficulties by specifically controlling for all interactions or behaviours likely to be prohibitive to sleep. Changes in the timing and duration of sleep were investigated when 9 subjects were removed from all normal temporal or social cues throughout a 60 hour period of enforced bed-rest. This bedrest period was intended as a period of disentrainment and immediately followed the second of two polysomnographically monitored baseline nights timed to coincide with habitual sleep patterns. Thus subjects were assumed to be fully rested on commencement of the disentrainment phase of the experiment. It was found that subjects slept for almost 50% of the 60 hour period of disentrainment (mean 27.9 hours). During this time, the initiation of sleep episodes was dispersed throughout the 24 hour day. Episodes of both sleep and wake were typically shortened in comparison with baseline periods of habitual sleep/wake activity. Only 20% of all sleep episodes occurring throughout the 60 hour bed rest period were longer than 4 hours in duration. The mean sleep/wake cycle measured from the onset of one sleep period to the onset of the next was 6.1 hours although variability between and within subjects was considerable. However, the influence of circadian factors on the duration of sleep episodes was clearly visible; resulting in over half (54.7%) of all sleep gained during the disentrainment period occurring through the night (2300h - 0700h). Campbell (1984) referred to both the limited number of behavioural options and lack of environmental and cultural references in explaining the shift towards a polyphasic pattern of short yet frequent episodes of sleep under these conditions. It was argued that although circadian controls were evident as an increased likelihood of sustained sleep throughout the night for these subjects, increased flexibility in the placement of sleep in an essentially unstructured environment indicates a reliance on exogenous factors in individual sleep patterns (Campbell, 1984). These findings provide strong support for the view that contemporary sleep patterns have developed through the imposition of dominant situational factors.

Similar changes in sleep behaviour were reported by Wehr et al (1993) when sixteen healthy young subjects were studied throughout a schedule of 7 'short' nights (8 hours of enforced solitary bedrest in darkness) and 28 'long' nights (14 hours of
enforced solitary bedrest in darkness). Wehr et al (1993) hypothesised that under conditions offering limited incentives to remain awake a latent capacity to adapt to environmental change in photoperiod length (i.e. day length) would be revealed. In this view, the consolidation of human sleep into a single period of essentially undisturbed sleep throughout the nocturnal period represents a relatively recent imposition of social and cultural preferences. In particular, it was argued that with the development of artificial lighting human beings rely on socially defined photoperiods of extended days and shortened nights rather than cues from the natural environment. Consequently, it was expected that with chronic exposure to an alternative photoperiod, change in endogenous circadian rhythms (e.g. temperature, sleep, melatonin production) would occur (Wehr et al, 1993).

Wehr et al (1993) described a shift towards a bimodal pattern of nocturnal sleep when subjects transferred from the 'short' to 'long' nights conditions. Sleep became highly fragmented during the 14 hours 'long' nights; in keeping with Campbell (1984) less than 20% of all sleep and wake episodes were greater than 4 hours in length. However, despite an additional 6 hours of sleep opportunity, total sleep time throughout the night had only increased by approx 60 minutes by the fourth week of 'long' nights. Extra sleep was made up of more REM, stage 1 and stage 2 sleep with no differences to the amounts of stages 3 or 4 sleep each night. This suggests that there is a limit to the amount of extra sleep subjects are able to take even in circumstances totally lacking in alternatives to sleep other than quiet wakefulness in darkness. This is consistent with a study of long-term (4-7 days) total sensory and social deprivation (Potter & Heron, 1972). Following a marked increase in the proportion of time spent asleep during the first 2 days (predominantly increased stage 2 sleep), by the 3rd or 4th day sleep times returned to normal levels. However, Potter & Heron (1972) did not report details of placement of sleep or changes in sleep fragmentation etc.

For Wehr et al (1993), changes throughout the four week period of 'long' nights included a gradual increase in the time elapsed between lights out and the first sign of sleep, and an increase in the time from the final waking in the morning and lights on. However, reductions in sleep efficiency were not reflected in subjective measures of mood on waking. Scales of vigor and fatigue from the Profile of Mood States subjective scales indicated improvements in subjective wellbeing (less fatigue, more vigor) following a schedule of 'long' nights.
Lengthening of daily rhythms of temperature, and melatonin, cortisol, and prolactin production during the 'long' nights phase of the experiment were reported (Wehr et al, 1993). These changes were compared with those found with seasonal adaptation to photoperiod changes in animals, e.g. Siberian chipmunks.

Wehr et al (1993) interpreted the finding of increased total sleep time throughout 'long' nights to indicate that a socially defined 'short' night confined to a limited period of opportunity resulted in the restriction of sleep time, below the limits of individual necessity for many people. In this sense the pattern of sleep emerging throughout Wehr et al's (1993) experiment, featuring increased sleep onset latency and frequent interruptions, is presented as a more 'normal' state of sleep as individuals respond to the control of natural rather than artificial daylengths.

However, as Campbell (1984) had already shown lack of stimulation to lead to a substantial increase in the fragmentation of sleep, it is debatable whether the changes in sleep profiles for Wehr's subjects were a direct result of changes in the period of available light. Throughout long periods of enforced bedrest in darkness (14 hours during the 'long' nights phase) these subjects would be highly motivated to sleep whenever possible because of boredom and the lack of any suitable behavioural alternative to sleep.

Although Wehr et al (1993) provided a highly contrived opportunity for the modification of sleep patterns, the influence of a wide-range of social and environmental factors on sleep has emerged. For adults, change in the duration of self-reported sleep has been associated with season (Rosen & Rosenthal, 1991); workload (Hawkins and Shaw, 1992) and lifestyles (smoking and alcohol consumption - Palmer, Harrison & Hiorns, 1980). Whereas for children, large families (Macgregor & Balding, 1988) and lower social class (Rugg-Gunn et al, 1984) have been linked with relatively shortened sleep at night. It seems clear that the immediate physical and social environment provides a wide range of factors likely to have some bearing on sleep patterns, This implies a strong volitional element to sleep duration and placement throughout the day. Evidence also suggests that this is likely to be affected by change in psychological status.

1.6.2 Psychological influences on sleep

Differences in the tendency for extreme sleep habits have failed to be explained in terms of presumably fixed aspects of an individual's psychological make-up. Webb & Friel(1970) & Buela-Casal, Carlos, & Caballo (1992) found no differences in
personality profiles of 'long' and 'short' sleepers (e.g. sleeping less than 5 hours and more than 9 hours each night). However, state rather than trait related psychological events are likely to be associated with change in sleep behaviours, including sleep duration.

Clinical levels of affective disorder are commonly associated with sleep-related difficulties (Ehlers, Frank & Kupfer, 1988). Complaints of intermittent waking, increased sleep latency, and early morning awakenings resulting in an overall reduction of sleep times compared with a preferred sleep length are frequently reported in depressed patients (Kerkhofs, Linkowski, Lucas & Mendelwicz, 1991). Sleeping longer than normal, hypersomnia, has also been associated with mood states. Hawkins, Taub & Van de Castle (1985) found depressed young adults were able to sleep almost twice as long as non-depressed controls when both groups were instructed to sleep as long as they want. They suggested that the ability to take extra sleep provides a protective facility for the depressed subjects in relieving the overwhelming sensation of negativity experienced during wakefulness. In a similar approach, Kerkhofs et al. (1991) found that when depressed adults and controls were allowed to sleep whenever they wished throughout the day a similar percentage of both groups (depressed - 50%, controls - 60%) napped during the day. However, for controls, daytime napping occurred during the mid-afternoon (i.e. at the circadian dip in alertness) whereas depressed subjects were as likely to sleep at any point during the day. Shimizu, Hiyama, Yagasaki et al. (1979) had previously compared daytime sleep between depressed and control subjects when restrictions on daytime activities were lifted. Six depressed subjects slept for an average of 5 hours 42 minutes throughout the day, compared with an average of 2 hours 22 minutes for 9 controls.

However, evidence also suggests that psychological factors influence the subjective perception of sleep duration. This has led to individual complaints of excessive (Billiard, Dolenc, Aldaz et al., 1994) or reduced (Johnson & Spinweber, 1976) sleep which were not verifiable using objective measures. Billiard et al. (1994) found that patients complaining of both a mood disorder and excessive sleeping were significantly less likely to be found to have polysomnographic indications of hypersomnia, e.g. shortened MSLT scores or increased recorded nocturnal sleep times, in comparison with patients complaining of excessive sleepiness alone.

Estimating sleep durations has also been investigated in relation to affective disorder. Bliwise et al. (1993) found that when a large group (n=71) of mildly
depressed subjects were asked throughout a 14 day period to give estimates immediately on waking of how much they had slept the night before, only 10% reported that they had slept within 10 minutes of an initial prediction at the start of this period. This initial prediction was in response to the question 'How many hours of sleep do you usually get at night?', i.e. the same question used in the Alameda County and American Cancer Society studies. More than 20% misjudged their actual average sleep times for this period by over 1 hour. Psychological factors can therefore be influential in producing changes to actual and perceived sleep patterns.

1.6.3 Genetic influences on sleep

In addition to variable situational and affective conditions, genetic factors have been associated with individual differences in the amount of time spent sleeping each day. Partinen, Putkonen, Kaprio & Koskenvuo (1982) studied the coincidences in self-reported sleep times between over 11,000 adult Finnish twin pairs. Both zygosity and co-habitation were associated with an increased likelihood of similar sleep duration between twins. Whilst co-habitating monozygotic twins were most likely to sleep for a similar duration, monozygotic twins living apart maintained a similar pattern of sleep length, with dyzygotic twins living apart showing least similarity in sleep patterns. Partinen et al (1982) concluded that whilst environmental factors are important in explaining differences between habitual sleep patterns, part of the widespread variability in preferred sleep patterns is due to genetic factors.

Under more controlled conditions, Webb & Campbell (1983) studied similarities in the capacity for spontaneous oversleeping in 14 monozygotic and 14 dyzygotic twin pairs. Overnight EEGs were recorded for each subject, with twins sleeping separately, under the instructions to sleep 'as long as they could'. Correlations for monozygotic and dyzygotic pairs were highly significant for both total sleep time, and the length of extra sleep gained (when subtracted from a previous baseline recording). Additional similarities between monozygotic twins of measures of sleep efficiency were also reported (i.e. latency to sleep onset and wakefulness throughout the night). Webb & Campbell (1983) interpreted similarities in the capacity for extra sleep in terms of a latent endogenous capacity. As such, it was assumed to be less subject to motivational and situational factors than self-report habitual sleep times.

Linkowski, Kerkhofs, Hauspie & Mendlewicz (1991) focussed on the similarities in sleep structure between 26 pairs of twins living apart. Sleep stages 2,3 and 3+4, but not REM sleep, were found to be associated with genetic factors. In addition, and in
keeping with Webb & Campbell (1983), wake after sleep onset, as a measure of sleep efficiency was also shown to be regulated by genetic factors.

1.6.4 Sleep patterns and maturation

The duration of sleep at night is known to change considerably throughout an individual’s lifetime, (Webb, 1969). Whilst the range of sleep duration for any particular age group can vary widely, Webb (1969) collated data from a large number of sources to show that the relative duration of sleep is gradually reduced for most people over time.

For Carskadon, Vieira & Acebo (1993), the impact of maturational factors was considered to be most powerful throughout the transition from childhood to adulthood. These researchers noted not only a reduction of overall sleep time, but a shift in the timing of sleep in favour of later bedtimes and rise times throughout this period. In their study of over 400 adolescents they found change in sleep patterns to be more closely related to physiological change (i.e. pubertal development) than psychosocial factors. Young adults with a greater degree of pubertal development were more likely to have a preference for delayed bedtimes and reduced sleep duration than those relatively undeveloped, regardless of actual age. It is possible that this is because visible physiological change enhanced individual autonomy leading to reduced parental influence on sleep habits. However, additional psychosocial factors, such as interaction with older peers or the presence of older potentially influential siblings, were found to be less important than physiological development alone. The authors of this study concluded that change in sleep behaviour around early adolescence is determined by biological factors rather than external factors, as is commonly supposed (Carskadon et al, 1993).

Nevertheless, the coincidence between change in sleep habits and age-related change in lifestyle cannot be discounted. For example, early adulthood often involves ‘overnight’ shifts between lifestyles of contrasting demands, such as the transition from college to employment. It is perhaps not surprising therefore to find that this type of change is accompanied by a volitional adjustment to sleep patterns. Wicks, Acebo & Carskadon (1994) found college students typically preferred an earlier bedtime and early rise time following graduation in comparison with their preferred sleep times as undergraduates. Furthermore, for those in full-time employment, as opposed to the less stringently regulated lifestyle of postgraduate study, change in sleep habits was more pronounced.
It is therefore difficult to establish the differential effects of an age-related change in physiological requirement for sleep from those stemming from the imposition of conventional age-related changes in life-style. Apart from physiological changes, the ageing process itself entails a socially-determined restriction to the range of everyday activity available. For example, the transition to adulthood is marked by a change from college to employment, whilst retirement from work is a normal requirement following the transition from middle- to old-age. As has previous been argued, prevailing environmental and social factors, such as those associated with various life 'stages', are likely to have a direct effect on sleep at night. The degree to which age alone requires an adjustment to patterns of sleep is therefore difficult to determine.

Attempts to explain differences in self-reported and measured sleep habits have led researchers to a number of conclusions regarding the apparent widespread variability in the population. Although the average person claims to sleep between 7-8 hours each night, extremely long or short sleepers are also relatively common. External factors have been held to be responsible for both restricted, extended and erratic sleep habits in both the short- and long-term. Carskadon and Dement (1989) suggested that the development of an habitual adult sleep pattern depends on the influence of known variables, such as social and psychological sleep factors, acting on an underlying genetic sleep need. Thus inter- and intra-individual variability in sleep patterns result from the interaction of competing factors at a given time and place. For some individuals this will entail relative stability of sleep habits, whereas for others, rapidly changing environmental or psychological events are likely to produce corresponding disruptions to sleep.
1.7 Summary

The purpose of this introductory review was to consider the contribution of previous research towards an understanding of a requirement for sleep. The following key findings have emerged:

- The majority of people claim to sleep between 7-8 hours each night, although estimates of sleep duration are prone to inaccuracy.

- Studying individual differences in preferred sleep patterns has failed to provide an optimal sleep time - the functional capacity of regular long or short sleepers remains unaffected despite unusual sleep habits.

- Contemporary sleep patterns are greatly influenced by a combination of social, psychological and genetic factors. In addition to pathology, these factors are responsible for intra- and inter-individual variability in sleep behaviour.

- Change to an established pattern of sleep can be well tolerated - in the highly motivated individual, performance remains unaffected by a chronic reduction of approx 2 hours sleep.

- Motivation is an important element in ensuring successful adaptation to a novel sleep routine.

- In contrast to this, a growing preference for alternatives to performance testing for the assessment of sleep need, particularly the MSLT, has recently generated concern to identify an optimal level of alertness following sleep. This work implies a limited scope for deviation from this level without serious consequence.

The following section provides a closer look at the way in which a need for sleep has been assessed in relation to its presumed consequences - in particular, the relationship between sleep at night and sleepiness throughout the day.
What is Sleepiness?
2.1 What is sleepiness?

In recent years, sleepiness during the day has been identified as the single most significant consequence of not getting enough sleep at night (Carskadon & Dement, 1987; Roth et al., 1982). Considerable effort has been directed towards an understanding of this state, with particular emphasis on the control of excessive sleepiness resulting from pathology or personal habit. Everyday language provides a broad range of words to describe sleepiness. Whilst adjectives such as 'sleepy' and 'tired' are frequently used to suggest an immediate desire for sleep, in certain contexts, the need for sleep as a physiological necessity is not always clear. For example, it is often assumed in the scientific literature that sleepiness occurs as the manifestation of a physiological drive for sleep fluctuating in accord with the amount of prior wakefulness. As such, this physiological drive can be accessed and measured objectively, and even predicted as the interaction between prior wakefulness and circadian phase (Carskadon and Dement, 1982). At any point in time, it is theoretically possible to measure an individual's level of sleepiness as an ongoing state.

However, this idea runs counter to a subjective experience of sleepiness which, as the range of alternative descriptors in common usage attest to, can occur in many forms and for many reasons other than an obvious physiological need for sleep. To convey this verbally relies on a familiarity between users which is dependent on a combination of both local consensus and etymology. Within a family for example, word preferences, which might otherwise be misconstrued by an outside group, are successfully used to make fine definitions between the various states of sleepiness. In addition to a predictable development of sleepiness towards the end of a long day, sleepiness-related terms are commonly used to describe situations which might equally be considered to be characterised by a pervasive sense of sleepiness. These might include the effect of a drawn-out familiar task, a long, uneventful car journey, the fatigue of personal despair and frustration, or circumstances in which there is a general lack of motivation to remain alert. Words which imply sleepiness, such as lethargic, listless, drowsy, fatigued, exhausted, weary etc. can be made specific to these conditions suggesting that it is possible, even at a basic level, to discern differences between different forms of sleepiness.

All of the above conditions qualify, in lay terms, as a state of sleepiness. In contrast, for the sleep researcher, sleepiness has recently undergone a transformation of conceptual and operational methods, such that limitations to acceptable terminology
used in its study are imposed through consensus and peer review of the scientific literature. The words "sleepiness/sleepy", as distinct from "tiredness/tired" etc., are generally advocated when referring to a drive for sleep. As an example, Moldofsky (1992) distinguished between sleepiness, as the physiological drive for sleep, and tiredness as a non-specific subjective description. In his view, the term "sleepy" is attributable to a state which precedes the onset of restorative sleep. By way of contrast, examples are given of a number of conditions for which tiredness is a common complaint, e.g. chronic fatigue syndrome, affective disorder, but for which sleep is neither imminent nor predictably restorative (Moldofsky, 1992). Underpinning this distinction is the belief that 'true' sleepiness can be verified in terms of the inevitability of actual sleep.

Implicitly, this is to suggest that all episodes of sleep are preceded by a period of sleepiness, and this is consistent with a fundamental tenet of contemporary sleep research. Carskadon & Dement (1982) have argued that only physiological sleepiness will lead to sleep, as opposed to the subjective consequences of environmental conditions or motivational factors which fail to stimulate an individual towards full alertness. In this view, it is therefore logical to assume that, given the means to access this physiological level of sleepiness, it would be possible to assess the adequacy of sleep behaviour for a given individual.

It might be argued that this policing of the language of sleepiness promotes a simplistic view of sleepiness as a single state, varying only by degree. Beyond a simple claim "I feel sleepy", the richness of everyday language to describe sleepiness is largely superfluous to empirical and clinical investigations. However, by limiting the operational means of describing and measuring sleepiness we have been encouraged to rely on a unidimensional view of sleepiness, whereby sleep is located at the extreme of a sleepiness/alertness continuum.

Broughton (1992) has questioned this conceptualisation of sleepiness and the ramifications of assessing a basic requirement for sleep on these terms. In his view, it is probable that separate states of sleepiness occur for a variety of reasons, and that these can be differentiated in terms of their subjective and behavioural consequences. Broughton (1992) argues that inconsistencies in levels of sleepiness using different measures (e.g. the MSLT, subjective scales, performance tests) do not reflect differences in relative sensitivity to sleepiness between these tests, but the likelihood of 'fundamentally different states of sleepiness' originating for different reasons. Over a range of contexts, the sensitivity of a particular test is therefore
enhanced according to the appropriateness of its usage to the exact nature of sleepiness under study.

Thus, at least two prominent theoretical positions regarding the measurement of sleepiness can be identified: (i) Sleepiness is a unidimensional state for which successful measurement relies on the sensitivity of the test. The MSLT has been promoted as unique in directly accessing this single physiological state of sleepiness in human subjects. (ii) Sleepiness is caused by a range of factors resulting in different kinds of sleepiness. The appropriate test to use is indicated by identifying the source of sleepiness.

These two positions have particular relevance for the assessment of a need for sleep: the first assumes that this can be achieved by determining the point at which more sleep at night will no longer lead to a reduction of daytime sleepiness. The second position implies that, as not all sleepy people are short of sleep, this would not necessarily or logically follow. It is the pervasiveness of the first position which has, in recent years, led to substantial and controversial scepticism regarding the efficacy of a conventional 8 hours of sleep at night in satisfying a need for sleep in the average individual.

2.2 SLEEPY→SLEEP→ALERT:
Sleepiness as a unidimensional state.

A significant number of sleep researchers support a view of sleepiness as a unidimensional state in their methodological approaches to its measurement (Carskadon & Dement, 1982; Roth et al, 1982). This implies a reciprocal relationship between sleepiness and a physiological need for sleep. Underlying a preference for the MSLT, for example, in determining levels of sleepiness throughout the day, or change following the manipulation of sleep duration at night, is the assumption that sleepiness is symptomatic of an endogenous call for sleep. Early in its development, Richardson et al (1978) regarded the MSLT as encapsulating an intuitive 'face validity' in that the speed with which a person falls to sleep is a direct reflection of the need for sleep at that particular time.

From this perspective, sleepiness plays a significant role in maintaining optimal alertness for the individual by signalling the impending need for sleep, with the promise of restored alertness to follow. The behavioural consequences of ignoring this signal to sleep, are likely to be severe and cumulative (Carskadon & Dement,
In keeping with this view, Roth et al (1982) argued that only a limited number of factors influence a physiological state of sleepiness. These include reduced or fragmented sleep, circadian rhythms, sleep pathology (e.g. narcolepsy), and drugs acting on the central nervous system. For the individual free from sleep disorders or drug use, Roth et al (1982) argued that measures for the detection of sleepiness have consistently highlighted the importance of sleep duration at night and circadian phase in determining levels of sleepiness throughout the day (e.g. Carskadon & Dement, 1979, 1982; Roehrs et al, 1989, 1994). It follows that, for this type of individual, the only relief from sleepiness is actual sleep. Furthermore, Roth et al (1982) reported that correlations between available measures of sleepiness (the MSLT, subjective rating scales and certain performance tasks) are relatively high. However, in terms of reliability, the MSLT is described as the only measure of sleepiness reliably free from the distortions of subject motivation (Roth et al, 1982).

This view is also consistent with Borbély's (1982) influential model of sleep regulation. Borbély (1982) proposed a two-process model of sleep in which a physiological need, or pressure for sleep develops as the interaction between the amount of prior wakefulness and circadian phase. These two processes are identified by Borbély (1982) as Process S and Process C. The development of Process S throughout wakefulness can be summarised as an exponential increase in pressure for sleep that is reversed with the onset of sleep. At this point, Process S is assumed to decline exponentially as the pressure for sleep is discharged. Variability in the build-up of Process S following different intervals of wakefulness prior to sleep has been shown to produce systematic changes in the amount and power of SWS during the first three cycles of NREM sleep (Borbély, Baumann, Brandeis et al, 1981).

Whereas Process S is largely dependent on exogenous factors, i.e. behavioural decisions in timing and duration of sleep, Process C represents the influence of endogenous circadian variability in a pressure for sleep. This produces cyclic variations in the likelihood of sleep. Borbély (1982) described the regulation of human sleep in terms of the interaction between these two processes. The development of MSLT scores throughout an extended testing day (0800h-2400h) bear a strong resemblance to the changes in a pressure for sleep predicted by this model (Carskadon, 1992). Because of this it might be argued that the MSLT is responsive to the anticipated consequences of Process S and Process C. This is shown as a circadian derived alertness during morning and early evening trials.
against a background of gradually increasing sleepiness throughout the day. Taken from this perspective, sleepiness provides a direct regulatory control at a behavioural level (i.e. to counter alternatives to sleep) for the management of a sleep requirement.

The widely held belief that sleepiness is located as a continuous, measurable state between full alertness and sleep is also mirrored in the design of many subjective self-rating scales. For the individual, the opportunity to describe their experience of sleepiness using these scales is often limited to indicating their position relative to adjectives assumed to describe the extremes of sleepiness (e.g. 100mm Visual Analog Scale anchored by 'very sleepy' and 'very wide awake' - Carskadon & Dement, 1981). The Stanford Sleepiness Scale (SSS - Hoddes, Dement, Zarcone, 1972) is an extensively used form of the subjective rating scale. This scale includes previously validated sleepiness-related terms ranging from '1. - Active and alert, wide awake' through to '7. - Almost in reverie, sleep soon, lost struggle to remain awake', and is a popular choice in an experimental setting (e.g. Carskadon et al, 1986; Roehrs et al, 1989).

Alternatively, the Karolinska Sleepiness Scale (KSS - Åkerstedt & Gillberg, 1990) provides the individual with five levels of sleepiness [Extremely alert, Alert, Neither alert nor sleepy, Sleepy-but no difficulty remaining awake, Extremely sleepy-fighting sleep] positioned at alternate points along a 9-point scale. For each of these scales, subjects are instructed to indicate which word/phrase or position along the scale best describes how they are feeling at that time. Clearly, in the presentation of these scales the respondent is obliged to interpret the two extremes as poles of the same variable, and between point intervals as equivalent.

Thus the assumption of a reciprocal relationship between sleepiness and a need for sleep is reinforced. However, there is increasing evidence to suggest that, at times, this relationship between sleepiness and sleep is not always so clear cut. For example as Folkard & Åkerstedt (1992) have shown, a relatively high level of subjective sleepiness following sleep (i.e. sleep inertia) can be present despite an obvious reduced need for, or likelihood of, sleep. In this event, the relationship between the lower end of a rating scale (i.e. optimal alertness) and the higher end (extreme sleepiness emphasised as the likelihood of sleep) is compromised. That is, inertia, or increased sleepiness on waking, occurs independently of sleep need.
Folkard and Åkerstedt (1992) suggested that sleep is unlikely in these circumstances and have proposed an alternative three-process model based on additions to Borbély's (1982) two hypothetical processes. This included the introduction of an additional component - Process W - to account for a period of reduced alertness (lasting approx. 3 hours) following normal sleep which, to a limited extent, effectively counteracts the anticipated rise in alertness following the discharge of Process S (Folkard & Åkerstedt, 1992).

To recap, a unidimensional approach to sleepiness presupposes the existence of a single physiological state, varying only by degree. This is reflected by a preference for the MSLT and the design of popular subjective rating scales. Sleep need is assumed to be systematically related to the duration and quality of recent sleep and circadian phase. This is consistent with Borbély's (1982) popular view of sleep regulation and suggests a role for sleepiness in the discipline of individual sleep habits. However, it has also been shown that aspects of the way self-rated sleepiness evolves across the day are not predicted by this view. This suggests that, as with the example of sleep inertia, it is possible that there are qualitatively different forms of sleepiness to that assumed to influence performance during the MSLT.

2.3 Reasons for sleepiness: an alternative to the unidimensional approach

Broughton (1992) considered the possibility of qualitatively different states of sleepiness and proposed at least five 'causes' of sleepiness, originating from "fundamentally different mechanisms": (i) sleep loss, (ii) circadian effect, (iii) selective pressure for REM or NREM sleep, (iv) impaired arousal, and (v) impaired sleep onset mechanisms. In his view, it would be inappropriate to routinely apply unidimensional tests of sleepiness, such as the MSLT and subjective rating scales, as different forms of sleepiness are likely to have different behavioural and subjective consequences.

Dinges (1989) has also contributed to undermining the popular belief that sleepiness is always a direct manifestation of a physiological need for sleep (e.g. Carskadon & Dement, 1986). For him, conditions other than sleep loss and circadian factors which play a direct causal role in the development of sleepiness include sleep inertia following sleep, the effects of age, health, drugs, and environmental and contextual features. In support of this view, there is considerable evidence to suggest (i) paradoxical findings between tests of sleepiness, and (ii) factors likely to influence
objective tests to indicate high levels of sleepiness without obvious sleep need. It shall be argued that these findings combine to undermine the reliability of a unidimensional approach to sleepiness.

**(i) paradoxical findings between tests of sleepiness**

Broughton (1992) has argued that inconsistencies between measures are common and reflect a basic fallibility of the unidimensional approach to sleepiness. In particular, it is suggested that objective levels of sleepiness can often be misleading when applied routinely. Broughton (1992) illustrated this point with reference to the frequent complaint of severe daytime sleepiness in many self-reported insomniacs despite a failure to sleep when the opportunity arises during the MSLT. Seidel, Ball, Cohen et al (1984) and Stepanski, Zorick, Roehrs et al (1988) reported similar findings of insomniacs with verified poor sleep at night, and complaints of severe subjective sleepiness, "with no measurable daytime sleep tendency". Johnson, Spinweber, Gomez et al (1990) and Johnson, Freeman, Spinwber et al (1991) also pointed out the lack of predictable association between objective and subjective measures of sleepiness for normal subjects. Easterbrook, Maclean & Knowles (1994) concluded that despite a gradual increase in MSLT determined sleep propensity and increased subjective sleepiness (SSS) following one night of sleep loss, it was nevertheless difficult to assess the effects of this in terms of performance capacity even when sleep-sensitive tasks were used.

Dinges (1989) has also noted that not all measures of sleepiness give the same result, or are sensitive to changes in sleepiness following the experimental manipulation of sleep to the same degree. Two reasons for this are considered: the first assumes sleepiness to be a single physiological process for which not all measures are equally sensitive. The second reason, and the one favoured by Dinges (1989), is that differences between measures reflect the need to consider the specific circumstances in which sleepiness occurs, and to provide the appropriate test for that context. The challenge for sleep research is to provide new dimensions of sleepiness, other than that based on a need for sleep, in order to interpret these frequently reported inconsistencies between measures.

**(ii) high levels of sleepiness without obvious sleep need**

One problem which has received considerable attention recently concerns the interpretation of relatively high levels of objectively determined sleepiness without an obvious need for sleep. Whilst MSLT scores of between 5-10 min for regular
sleeping, healthy, young adults have been interpreted as indicative of increased sleepiness due to a chronic sleep debt (e.g. Levine, Roehrs, Zorick et al, 1988; Manni, Ratti, Barzarghi et al, 1991), Johnson (1992) has questioned the significance attached to shortened sleep latencies for these subjects. However, the conclusions of Levine et al (1988) and Manni et al (1991) are consistent with the views of the American Sleep Disorders Association (ASDA, 1992) reporting on the significance of the MSLT score. Recommended guidelines for the interpretation of the MSLT score support the classification of three broad categories of sleepiness dependent on actual scores (<5 min = severe sleepiness; 5-10 min = moderate sleepiness; >10 min = mild sleepiness - ASDA, 1992). This suggests that the same score will have the same significance (i.e. will represent the same level of physiological sleepiness) for different individuals.

Nevertheless, Johnson (1992) considered the lack of association between objective and subjective measures of sleepiness to be problematic for this group. It is suggested that:

"..... the same sleep latency may not have the same significance in two different persons and perhaps not even for the same person at two different points in time. We found that subjects with an average sleep latency of less than 5 min do not differ significantly in performance from those with an average latency of over 10 min".

Johnson (1992)

This implies that differences between MSLT scores for some individuals might be dependent on factors other than physiological sleepiness, for example the efficiency of sleep onset mechanisms. However, as the MSLT is widely used for the assessment of individual sleepiness, it is necessary to be able to predict the consequences of a shortened sleep latency on the MSLT. In particular, whether or not this is likely to impair performance across a range of common tasks, or increase the probability of unwittingly falling asleep in potentially dangerous situations. To that end, it might first be necessary to revise the current concept of sleepiness in order to explain variation in MSLT scores more completely. That is, in addition to the three broad categories of sleepiness proposed by ASDA (1992).

It has already been established that the ability to resist sleep can be separated from sleep propensity on the MSLT (Hartse, Roth & Zorick, 1982; Sugerman & Walsh, 1989; Alexander et al, 1991). Sugerman & Walsh (1989) reported that subjects were able to resist sleep in a similar setting to the MSLT (lying down in a quiet, darkened room) despite extreme sleepiness (i.e. when the MSLT score fell to
around 1-2 min). Alexander et al (1991) compared the effects of introducing an additional financial incentive and found that the ability to resist sleep using an MSLT variant despite acute sleep loss was further enhanced. It has been argued that the instructions for the MSLT - "try to go to sleep" measure physiological sleepiness whilst "try to resist sleep" (Maintenance of Wakefulness test - MWT) taps into the "capacity for wakefulness" (Sangal, Thomas & Mitler, 1992). This presents difficulties for a unidimensional view of sleepiness as both are assumed to describe independent features of the sleep/wakefulness system whilst relying on the same measure - latency to sleep onset.

We can think of being able to resist sleep despite an acute sleep loss as the converse of being able to fall asleep easily without an obvious sleep need. It has also been shown that it is possible, despite sleepiness to be motivated to maintain performance (Horne & Pettitt, 1985). It seems plausible, therefore, to expect some degree of volitional control over the ease of falling asleep. Taken to an extreme this might emerge in otherwise non-sleepy individuals as the ability to fall asleep easily in non-arousing situations. When this is formalised as the MSLT test this type of individual is likely to be classified as suffering from severe or pathological sleepiness. The problem is therefore how to extrapolate from the levels of sleepiness found using the MSLT to conditions outside of the laboratory.

One solution is to move away from the premise that all sleep is purposeful with regard to satisfying a physiological requirement. As Horne (1988) has argued, the apparent flexibility with which many individuals are able to reduce their sleep by moderate amounts over the long-term without serious consequences might suggest that, at least part of a daily diet of sleep is dispensable. Horne (1988) refers to this as "optional" sleep, as opposed to the indispensable "core" sleep. Core sleep is presumed to account for all of human slow wave sleep (hSWS) and some REM sleep. As hSWS is confined to the first part of the night the requirement for core sleep is fulfilled within this period. Horne (1988) suggests that there is a period (of around 2 hours) towards the end of the night which is normally dedicated to optional sleep, and consequently of little restorative value.

Horne (1991) has also differentiated between two types of sleepiness which are identifiable as a response to a need for either core or optional sleep. In his view, the consequences of a development of either core or optional sleepiness are quite different in terms of their effects on both sleep propensity and performance tasks, Horne (1991). When sleep loss cuts into the obligatory core sleep, the resultant
sleepiness is often profound and overwhelming. However, as only core sleep is essential, sleepiness due to a loss of optional sleep is more responsive to compensatory or volitional attempts to overcome it.

Horne (1991) described sleepiness due to a need for optional sleep as a learned response, i.e. it is essentially a habit. As with most habits, the effects of withdrawal (from optional sleep) are essentially transient and can be overcome by increasing motivation to remain alert. When sleep is gradually reduced over the long-term, sleepiness due to the loss of habitual sleep eventually passes (as long as the amount of core sleep remains intact). In this view, therefore, the components of at least one type of sleepiness are under the control of volitional elements. It is also reasonable to expect that, for some individuals, this basic flexibility in the control of sleep mechanisms will be more highly developed than for others, thereby accounting for apparent differences in the ability to both resist and initiate sleep. In view of the increasing number of reports of rapid sleep onset during MSLT trials for apparently normal, otherwise non-sleepy individuals (e.g. Levine et al, 1988; Manni et al, 1992), this represents a plausible alternative to the belief that sleep onset is a direct indication of a physiological need for sleep which cannot be facilitated by motivation (Carskadon & Dement, 1982).

2.4 Summary

In conclusion, although it has been claimed that the more popular approaches to sleepiness (the MSLT, subjective scales, and performance tests) access a single physiological state of sleepiness (Roth et al, 1982), the reliability of this view has been questioned (Broughton, 1992). A number of factors have been shown to interfere with the capacity to fall asleep on the MSLT yet this has been readily accepted as the gold standard in the objective measurement of a physiological need for sleep (Roth et al, 1992). Inconsistencies between tests suggests that we should at least be cautious in prioritizing a particular method in the light of contradictory findings. In particular the singling out of individuals presumed to be suffering moderate to extreme sleepiness without corroborative data from performance tests or subjective accounts highlights the basic inadequacies of a view based on the premise that all sleepiness originates from a physiological need for sleep.

The following studies were undertaken with these issues mind. Throughout the first experiment ten healthy, young adults who regularly slept between 7-8 h at night were monitored throughout 14 consecutive nights of extended sleep. Throughout
this time, they attempted to sleep for a maximum of 10 h each night in their normal environment. Measures of physiological and subjective sleepiness, mood and psychomotor performance were used to assess vulnerability following habitual sleep and change following extended sleep. As previous studies in this area had been limited to short-term laboratory based experiments, the feasibility of extending sleep over the long-term within existing domestic, social and work constraints was emphasised.

In the second section, three separate studies address the question of the significance of physiological sleepiness throughout the day in healthy, subjectively alert young adults. This is achieved by examining (i) the sensitivity of the MSLT test at this level of sleepiness and its relative inefficiency in detecting change following experimental manipulation using current scoring methods, (ii) the role of motivation in falling asleep throughout the day, and (iii) the occurrence of MSLT defined pathological sleepiness in otherwise normal, alert individuals.
3.

Extended Sleep
3.1 Support for the 8 hour sleep

Survey data has shown that there is extensive support for the idea that 8 hours sleep is sufficient for most people, and represents a sensible goal for those people who, for a variety of reasons, experience difficulties with sleep. This includes difficulties finding time to sleep because of work and/or social commitments, as well as the large number of people who feel they spend a less than satisfactory length of time asleep each night, and complain of difficulties initiating sleep and maintaining sleep throughout the night until an acceptable time in the morning. The purpose of this experiment was to establish the status of the average individual following 8 hours sleep at night, and to ask whether being able to regularly sleep for this amount, or aspiring towards this amount, represents a sensible compromise between a need to maximise both alertness through sleep, and the time spent in waking activity. Following on, a comparison was made between measures of physiological, functional and subjective status when the same subjects were encouraged to sleep for longer than their normal amounts over a two week period.

3.2 Experiment 1: Long-term extension to sleep

3.3 INTRODUCTION

Reports of excessive daytime sleepiness in normal, healthy, young people following Multiple Sleep Latency tests (MSLTs) have prompted concerns that such subjects are chronically sleep deprived (Levine et al, 1988; Manni et al, 1991). This view is endorsed by findings of apparent reductions in daytime sleepiness when similar subjects are given the opportunity to sleep for longer periods at night (Carskadon, Mancuso, Keenan, Littell & Dement, 1986; Roehrs et al, 1989). Earlier, Webb & Agnew (1975) argued from a comparison of archival (circa 1910) and contemporary data on sleep trends that nowadays there is a socially imposed restriction (approx. 90 min.) of sleep below optimal levels. These authors also claimed that the common finding of sleeping longer at week-ends also pointed to an additional sleep need. The view that there may be an underlying discrepancy between social determinants of sleep patterns and a basic physiological sleep need has far-reaching implications.

Recently, Carskadon & Davis (1989) and Acebo, Davis, Herman and Carskadon (1991) found in college students an association between increased subjective
daytime alertness and increased nocturnal sleep. In a similar vein, Hawkins and Shaw (1992) showed that sleep was perceived by student subjects to be improved qualitatively with increased duration on weekend nights. However, a reduction in perceived sleep quality was not found with voluntary reductions in night-time sleep as the semester progressed. Hence there may be confounding psychological factors.

In a systematic approach, Wehr et al (1993) compared the effects of 28 consecutive "long" nights (14h bed-rest in darkness) with 7 "short" nights (8h bed-rest in darkness). Findings of increased total sleep time (TST), improved self-reported daytime vigor, and reduced daytime fatigue were viewed by the authors as evidence of a pre-existing sleep deficit. Gradual changes in both sleep period time (SPT) and TST were seen throughout the four weeks of long nights. For the initial 1-2 days of this period, sleep was achieved relatively easily, despite the 18:00h commencement of the bedrest in darkness. The mean sleep onset latency was about 30 min.; similar to that of the short night period. As daytime activity was not monitored during the study, it is possible that subjects altered their daytime behaviour in response to the constraints on night-time activity. Furthermore, despite SPT and TST increasing abruptly and significantly with the transition to long nights, there was a gradual fall in both measures, particularly with mean TST, which declined to <8h, and was still falling on termination of the study. Compared with the baseline, an additional 360 min. opportunity for sleep led to an extra 60 min. of sleep. In order to achieve this amount of additional sleep therefore, there was a considerable cost in terms of hours of productive wakefulness lost. Had the long nights continued, a further reduction in TST seems possible. Finally, whilst Wehr et al (1993) attributed a number of improvements in mood scores to the sleep extension, Totterell, Reynolds, Parkinson et al (1994) found that an earlier than usual sleep onset, rather than sleep duration, was more likely to be associated with improved mood (cheerfulness, alertness) throughout the following day. Thus the apparent relationship between sleep extension and subsequent mood found by Wehr et al (1993) may have been confounded by additional factors such as the timing of the additional sleep with respect to habitual sleep times.

The increasing number of MSLT studies on normal, asymptomatic subjects apparently point to excessive daytime sleepiness in many cases. Levine et al (1988) examined MSLT scores in two groups (18-29y and 30-80y) following 8 h sleep, and found significantly shorter scores coupled with higher sleep efficiency throughout night-time sleep in the young subject group, which the authors concluded to be indicative of mild sleep restriction. However, although Manni et al (1991) reported
similar findings, of mean MSLT scores of less than about 10 min. from a group of 18 young, healthy, non-complaining students, there was no other evidence of marked daytime sleepiness using sleep-sensitive performance tasks or subjective measures.

A number of studies have focused on short-term changes in objective measures of daytime alertness following sleep extension. Carskadon and Dement (1979) measured daytime MSLT scores following four nights of extended sleep (10 h in bed). After the last extension night, an improvement in daytime sleepiness was reflected by the subjects' failure to sleep during 80% of MSLT trials, compared with 50% of trials following an initial baseline night. Whilst the authors viewed this as evidence for a chronic sleep debt, it must be noted that with half of the baseline tests having MSLT scores > 20min this conclusion must be qualified. Nevertheless, in a later description of this study, Carskadon and Dement (1982) refer to these findings in support of the view that "the 8 h bed-time represents a "chronic sleep deprivation" condition in young adults". In a further short-term study, Carskadon et al. (1986) found a significant improvement in MSLT scores following one night of extended sleep (11 h in bed), after a night of restricted (6.5 h in bed) or normal (8.5 h in bed) sleep. Compared with baseline values, MSLT scores improved by 4-5 min. after the sleep extension. However, no improvement was found in performance, and subjective sleepiness worsened during the extension.

Roehrs et al (1989) compared MSLT changes following 6 nights of extended sleep in two groups of subjects deemed "alert" (baseline MSLT >16 min.) and "sleepy" (baseline MSLT <6 min.). Neither group of subjects had complained of daytime sleepiness. Nevertheless, extended sleep led to improved MSLT scores in both groups, which was greater and more immediate for the "sleepy" than for "alert" subjects. After 6 nights of extended sleep, MSLT scores improved by about 2 min. and 5.5 min. in "alert" and "sleepy" subjects, respectively. There were small improvements in performance (although the authors conceded that this may have been due to a practice effect).

Roehrs, Shore, Papineau, Rosenthal & Roth (1994) reported a gradual improvement in MSLT scores throughout a 14 day period of extended sleep for subjects described as "sleepy". These subjects were asymptomatic healthy young individuals, regularly sleeping between 7-8h at night, with baseline MSLT scores of <6 min. It had previously been suggested that, in comparison with "alert" subjects, these subjects experienced a greater disparity between socially determined sleep
schedules and a biological sleep need. "Sleepy" subjects following a schedule of 14 nights of extended sleep (10 h in bed) were found to experience a gradual improvement in MSLT score from the baseline period (8 h in bed). Despite substantial increases in sleep latency (SL) and wake after sleep onset (WASO) these changes were interpreted by Roehrs et al (1994) in support of a view of chronic sleep deprivation following a 7-8h sleep pattern for these subjects. However, it should also be noted that, for a control group of "sleepy" subjects following a schedule of 8 h in bed each night throughout the same period, considerable differences in MSLT scores between baseline testing days (up to approx. 3 min.), casts some doubt on the stability of the MSLT score for this particular subject group. In addition to this, improvements in MSLT scores following extended sleep were not found for one third of the extended sleep group (3/9 subjects). Again this suggests that factors other than the amount of sleep gained at night will determine MSLT measured daytime sleepiness for this group.

Evidence of a detrimental effect of extended sleep, using measures other than the MSLT, suggest that there may have been undue emphasis on the MSLT as the objective measure of daytime sleepiness. For example, Taub (1971) reported decrements in the Wilkinson vigilance task and on a complex motor performance task following two nights of extended sleep (TST = 9.1h). In a further study comparing a single night of extended sleep counterbalanced with a regular 7-8h baseline night, Taub (1981) reported reduced vigilance performance following the sleep extension, coinciding with increases in subjective sleepiness. Such a discrepancy between the various objective measures used to assess the effects of extended sleep was endorsed by Manni et al (1991) who concluded from MSLT scores obtained from normal, healthy young adults, that several subjects experienced a "fairly marked objective drowsiness" even though sleep-sensitive performance tasks had failed to show any significant impairment. However, it should be remembered (see above) that Roehrs et al (1989) have provided some evidence of improved MSLT scores linked to enhanced performance.

Extended sleep may have a detrimental effect on mood, which Globus (1969, 1970) identified as the "Worn Out" syndrome, typified by thick-headedness and lethargy. Hawkins et al (1985) reported worsened subjective fatigue among normal subjects when they were allowed to sleep longer than usual. Carskadon et al (1986) found an increase in subjective sleepiness following extended sleep despite an improvement in MSLT scores. Such findings appear to contradict a view of widespread chronic
sleep deprivation. But the relatively short duration of these and other sleep extension studies may have been insufficient to reveal any true effects.

Finally, the effects of extending sleep at night have only been monitored when this extra sleep is gained in a laboratory setting. It is necessary to establish the feasibility of extending sleep in a domestic environment in order to consider the wider implications of a chronic sleep debt amongst the general population. Bearing in mind that the individuals involved in previous studies were already regularly sleeping 7-8h per night, and were not experiencing subjective levels of daytime sleepiness, then for the changes following extended sleep to be convincing to them, the benefits to daytime functioning should be substantial. Most importantly, the effort to actually take extra sleep in the home, and the disruption to established work and social schedules, should not be disproportionate to any perceived benefits. Given that there already exists substantial popular support for the belief that 8 hours sleep each night is sufficient for most individuals, any identifiable shortfall in satisfying a physiological sleep requirement following this amount of sleep will inevitably need to be balanced against the influences of the immediate social environment.

In this study these issues were addressed by investigating the effects of a prolonged period of extended nocturnal sleep in a home setting in terms of a number of specific questions: does longer night-time sleep lead to less daytime sleepiness? does this coincide with improved performance? can this be sustained for many nights? is there increased daytime alertness, and if so, any potential benefit to daytime functioning? For these purposes, the subjects involved attempted to sleep for up to 10 h at night over 14 consecutive nights before returning to habitual sleep lengths. Measures were taken of MSLT, vigilance performance, subjective daytime sleepiness, mood, and night-time sleep. Subjects slept in their own home throughout in order to assess the feasibility of extending sleep within a social as opposed to strictly laboratory environment.

3.4 METHOD

Subjects -Eleven subjects (7F/4m; mean age 23.6 y; range 18-36 y) were recruited on the basis of being regular 7-8h sleepers per night with little variation in sleep habit between nights. All potential subjects were screened to exclude those with sleep difficulties, regular daytime napping, and illness or medication that affect sleep, see Appendix 1. Subjects were neither morning nor evening types (Horne & Ostberg, 1976) and were in the normal range on Trait Anxiety in the Spielberger
State-Trait Anxiety Inventory (Speilberger et al, 1977). All subjects engaged in regular daytime work, moderate physical activity and periods of relaxation.

**Design** - Subjects underwent 26 consecutive nights of home-based sleep monitored by wrist actimetry. Subjects slept alone throughout. EEGs were undertaken on selected nights. The schedule was (see Table 3.1): 7 nights of baseline (BASE) sleep (7-8 h/night), 14 nights of extended (EXT) sleep (up to 10 h/night), and 4 nights of recovery (REC) sleep (7-8 h/night). To ensure a regular sleep schedule, bedtimes were held constant within all conditions: lights out= 23:30 h (BASE and REC), 22:00 h (EXT) sleep. During EXT subjects remained in bed until 08:00 h and were exhort to sleep throughout the period in bed.

**Actimetry** - Ten minutes prior to lights out (all conditions) subjects had to begin their normal preparation for sleep. At this time an actimeter (Gaehwiler Electronics - Hombrechtikon, Switzerland), set to sample at 30 sec intervals, was attached to the dominant wrist. Exact time of lights out was recorded in a sleep diary. Actimeters were calibrated each week against a master clock, to within +/- 5 sec of real time. Sleep onset (SO) was determined by software designed for this purpose, and previously validated against the following EEG criterion (Horne, Pankhurst, Reyner, Hume & Diamond, 1994): "the beginning of first period of continuous sleep lasting more than 6 min. and consisting of stage 2 sleep or deeper". Sleep period time was the period between SO and final awakening.

**Overnight EEG recordings** - In addition to actimetry, Medilog 9200 ambulatory EEG recorders (Oxford Medical Systems, UK) were used to monitor overnight sleeping in the subjects' home. Recorders were pre-set with the precise time. Subjects were instructed to use an event marker on the recorder to indicate the time of lights out and final waking. The following montage was adopted: two channels of EEG (C4/A2 and C3/A1), bilateral electro-oculogram (EOG) and submental electromyogram (EMG). Recordings were made on night 4 of BASE (adaptation), night 7 of BASE sleep, nights 4,7,11 and 14 of EXT sleep, and night 4 of REC sleep. EEGs were scored into sleep stages in one minute epochs using the Oxford Medilog sleep stage software in conjunction with blind visual scoring by an experienced scorer. A small number of recording nights from the EXT sleep period were lost due to technical problems and sleep staging was directed towards night 7 of BASE, nights 7 and 14 of EXT and night 4 of REC sleep, for which full recording sets were available for all subjects.
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Table 3.1: Testing Schedule Throughout 26 Nights of Monitored Sleep
The following definitions were used:

**Sleep Onset Latency (SOL)** - the time from "lights out" to the onset of 6 min. of continuous sleep, as defined above.

**Sleep Period Time (SPT)** - time from SOL until final awakening.

**Total Sleep Time (TST)** - SPT less any interim wakefulness.

**Sleep Efficiency (SE)** - TST as a percentage of SPT.

The first 300 min. of artefact-free EEG following sleep onset were also subjected to spectral analysis using the RHYTHM software package (Stellate Systems, Quebec, Canada), after digitising the EEG at 128 Hz and sampling in 4 sec epochs, using 0.25 Hz frequency bins within the range 0.75-31.0 Hz. Only data with regard to delta activity (0.75-4.0 Hz) will be reported on here. Absolute power values were averaged within this frequency band for each 60 sec of the sleep period.

**Sleep Diary** - Each morning, 5 min. after awakening subjects entered times of sleep onset (estimated) and final awakening. They also assessed:

i) sleep quality ("how well did you sleep last night?") on a scale of 1 to 10 (1=slept extremely badly; 10 slept extremely well). See Appendix 2.

ii) sleepiness on the Karolinska Sleepiness Scale (Åkerstedt and Gillberg, 1990), which is a 9-point subjective scale: 1= Extremely Alert; 3= Alert; 5= Neither Alert nor Sleepy; 7= Sleepy but not fighting sleep; 9= Extremely Sleepy, Fighting Sleep, Effort to Stay Awake. See Appendix 3.

iii) the Vigor and Fatigue scaled from the Profile of Mood States (POMS - McNair, Lorr & Droppleman, 1971). Both these scales deemed by McNair et al. to be independent factors, include adjective relevant to the detection of the Worn Out syndrome (see above), e.g. "bushed, sluggish, energetic, weary". See Appendix 4.

**Daytime Sleepiness**

Daytime sleepiness was monitored using both objective and subjective measures:

**MSLTs** - These were performed following nights 4 and 7 of BASE sleep, nights 4,7,11 and 14 of EXT sleep, and night 4 of REC sleep. The experimental schedule throughout these days is shown in Figure 3.1. On each testing day subjects came into the sleep laboratory at 09:30h for the application of electrodes. Four MSLT trials were administered at 10:00h, 12:00h, 14:00h and 16:00h. A single channel of EEG (C3-A1) and two channels of EOG were recorded according to the guidelines to the MSLT (Carskadon et al., 1986). Subjects were told to lie down on a bed in a
quiet, darkened room for a period of up to 20 min., with the instructions to, "lie still, and with your eyes closed try to go to sleep.". Between trials subjects participated in light studies. Heavy meals, caffeinated drinks and vigorous exercise were prohibited throughout the whole of the testing day period. For each MSLT trial a sleep latency score was determined as the time in minutes from "lights out" to the first continuous 90 sec of stage 1 sleep, or 30 sec of another sleep stage, according to MSLT guidelines (Carskadon et al, 1986). The test was terminated either at this point or after 20 min. if there was no sleep.

<table>
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**Fig 3.1 Testing schedule throughout the day.**
Karolinska Sleepiness Scale (see above) - This was used as the subjective measure of daytime sleepiness and was given following preparation for each MSLT testing trial (Fig 3.1).

Performance Testing - Following night 5 of BASE sleep, nights 5 and 12 of EXT sleep and night 5 of REC sleep, subjects underwent 55 minutes of the Wilkinson Auditory Vigilance Task (Wilkinson, 1968). Prior to BASE they underwent two practice sessions. In the main study, testing commenced at 14:00 h and was performed in a quiet, darkened room, free from external distraction. The task required responses to computer simulated target tones embedded within a series of non-target tones. Pilot studies were used to determine auditory thresholds for the detection of the target tone, and the minimum target-tone difference for discrimination. Tones were presented at 2 sec intervals, and lasted for either 350 ms (target) or 500 ms (non-target). Target signals were randomly presented in the ratio 1:20 non-target tones. The following were calculated: probability of correct identification of a target signal (HIT), probability of false report (FALSE REPORT), and the metric "D prime" (Swets, 1959), as an integration of hits with false reports.

In addition to this, a single channel (C3/A2) of EEG recording was taken throughout the test using Medilog 9200 ambulatory EEG recorders (see Overnight EEG recordings for details). An event marker on the EEG recorder was pressed at the commencement of the Wilkinson test. The EEG signal throughout these recordings was found to contain problematic amounts of muscle artefact. This was accounted for largely by the movement of the eyes as subjects were explicitly instructed to remain with eyes open throughout the test, resulting in frequent blinking and scanning movements. Visual analysis identified recordings for which the distortion of the EEG signal due to muscle artefact was relatively reduced. This produced a subgroup of six subjects.

For these six subjects analysis was directed towards recordings taken throughout BASE, the second EXT and REC test sessions. The first 5 min and last 2 min of the EEG recordings were discarded as subjects were more likely to be restless during these periods. The remaining 48 min of EEG were subjected to spectral analysis using the RHYTHM software package (see above), after digitising the EEG at 128Hz and sampling in 4 sec epochs, using 0.25 Hz frequency bins within the range 0.75-31.0Hz. As change in theta activity has been shown to be most closely related with change in objective and subjective sleepiness (Åkerstedt & Gillberg, 1990)
only activity within this range will be reported on here. Also, unlike slower frequencies, theta activity was less likely to have be distorted by activity other than EEG, e.g. rolling eye movement etc. Absolute power values were averaged within this frequency band for each 60 sec of the sleep period.

Further analysis was obtained using the Questar analysis system currently under development (Oxford Medical Systems, UK). This entailed subjecting the EEG to a neural network analysis previously trained using segments of pre-scored (Rechtshaffen & Kales, 1968) EEG recordings. At the present stage of development, this analysis produces an output in terms of 3 underlying processes: Wake, REM/light sleep, and Slow Wave. A probability value of each state occurring is given for each 1 sec of the EEG analysed. Only data with regard to the probability of Wake throughout the vigilance test will be reported on here. The percentage of total test time during which the probability of Wake was greater than 0.9. is given for each test as an indication of alertness throughout the test.

**Mood** - Before each MSLT trial (see Fig 3.1) subjects again completed the POMS Vigor and Fatigue scales (see above), and the State Anxiety questionnaire of the Speilberger State-Trait Anxiety Inventory (Speilberger, 1977).

Only ten of the eleven subjects completed each night of the study in accordance with the specified sleep schedule. One subject (female, 32 y) withdrew after four nights of extended sleep complaining of unacceptable levels of sleep disturbance, frequent awakenings throughout the night and considerable difficulty with re-initiating sleep. Overnight EEG recording confirmed that during her final night of participation, TST had been reduced to <5 h (from >7 h) with evidence of excessive intermittent waking. There was no satisfactory explanation for this in terms of personal events, high levels of anxiety etc. Her data were excluded from the following analyses.

The results are presented in two sections allowing (i) an assessment of the adequacy of a preferred sleep schedule for these subjects of approx. 8 hours sleep each night in terms of their levels alertness throughout the next day, and (ii) a comparison of individual change in terms of any perceived or actual benefit following the opportunity to sleep longer at night.
3.5 RESULTS - Part I

The effects of 8 hours sleep at night on sleepiness, performance and mood throughout the following day.

The initial stages of analysis were concerned with the functional status of subjects following their preferred sleep schedule. This includes particular emphasis on any signs of impaired alertness throughout the day, from which it might be inferred that there had been a failure to satisfy a physiological requirement for sleep in a subject's recent history.

3.5.1 Night-time Sleep

Actigrams - Mean SPT's calculated for each subject throughout the 7 nights of BASE are shown in Figure 3.2. It can be seen that throughout this period intra-subject variability in sleep time is low for most subjects.

![Actigraphy chart showing sleep period variability over 7 nights for 10 subjects.](image-url)

**Fig 3.2** Actigraphically determined nocturnal sleep throughout 7 Baseline nights
Overnight EEGs - Figure 3.3 shows individual breakdown of recordings from BASE7 into sleep stages, from the moment of sleep onset until final awakening, and including all periods of intervening wake. A mean SPT for these subjects of 449 minutes confirmed subjective estimations of sleep duration of approximately 7½- 8 hours sleep. Although all EEGs were edited following automatic sleep staging, the analysis results for sleep stage 1 proved to be problematic. For two subjects in particular, subjects 3 & 5 (see Fig 3.3) the amount of stage 1 in minutes seemed greater than might be expected for fully rested, non-anxious subjects, sleeping in the own home following an opportunity to adjust to recording equipment during a recent adaptation night.

Carskadon & Dement (1989) offer general guidelines to sleep architecture in young, normal subjects. In their view, sleep across the night for the type of subjects used in this study would roughly consist of the following proportions:
5% WASO
2-5% Stage 1
45-55% Stage 2
3-8% Stage 3
10-15% Stage 4
20-25% REM

For each subject, time spent in individual stages of sleep are presented as a percentage of SPT, Table 3.2. The proportion of time spent in sleep stage 3,4 & REM sleep for these subjects is in agreement with the above guidelines (Carskadon & Dement., 1989).

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<td>17.0</td>
<td>38.4</td>
<td>55.4</td>
<td>9.1</td>
<td>2.1</td>
<td>25.2</td>
<td>8.2</td>
</tr>
</tbody>
</table>

Table 3.2 Breakdown of individual sleep stages as a percentage of SPT.

The combined proportions of stages 1 & 2 is also as might be expected for all subjects except subject 5. The reason for an apparent increase in stage 1 (at the cost of stage 2 sleep) for some subjects is unclear. It is possible that subjects had not adapted to recording equipment causing it to interfere with the progression of sleep and prolong periods of 'light' sleep. However, there was no corresponding increase in recorded wake after sleep onset suggesting that this is not the case. It seems more likely that the automatic sleep staging failed to distinguish efficiently between stages 1 & 2 sleep for some subjects. The criteria for the automatic scoring of stage 2 relies on the detection of transient features in the EEG (i.e. K complexes and sleep spindles). Whereas rules for the scoring of stage 1 emphasise changes in frequency
(i.e. general slowing of EEG with a reduction in alpha activity) and coincident changes in EOG, the detection of features of stage 2 depend on the satisfaction of specific amplitude thresholds. For this reason, the scoring of stage 2 is likely to be more prone to error as differences in bone density, poor electrode contact etc. will produce misleading rather than accurate measures of the amplitude generated. This would explain why combining the two stages went some way towards rectifying an apparent imbalance in sleep stage distribution.

Means and standard deviations of sleep parameters for all subjects are included in Table 3.3. These findings are generally consistent with the guidelines to normal human sleep presented by Carskadon & Dement (1989).

<table>
<thead>
<tr>
<th>Sleep Period Time (SPT)</th>
<th>mean (min)</th>
<th>standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Sleep Time (TST)</td>
<td>411.1</td>
<td>36.2</td>
</tr>
<tr>
<td>Stage 1</td>
<td>66.5</td>
<td>35.8</td>
</tr>
<tr>
<td>Stage 2</td>
<td>163.2</td>
<td>21.7</td>
</tr>
<tr>
<td>Stage 3</td>
<td>42.7</td>
<td>15.0</td>
</tr>
<tr>
<td>Stage 4</td>
<td>49.1</td>
<td>27.4</td>
</tr>
<tr>
<td>SWS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>REM</td>
<td>89.7</td>
<td>23.5</td>
</tr>
<tr>
<td>Wake after sleep onset</td>
<td>37.7</td>
<td>14.6</td>
</tr>
<tr>
<td>(WASO)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep Latency</td>
<td>22.4</td>
<td>8.5</td>
</tr>
<tr>
<td>Sleep efficiency</td>
<td>91.8 (%)</td>
<td>2.9</td>
</tr>
</tbody>
</table>

Table 3.3 Means (and SDs) of sleep structure throughout BASE7 (n=10)

Subject 5 was exceptional in that over 70% of all sleep for this night was scored as either stage 1 or stage 2 sleep, with less than normal total REM sleep (12.8% / 47 minutes). Subject 5 was also unusual in having the shortest SPT for this night (368 minutes). This was despite an estimated habitual sleep time of 7½-8 hours. The EEG for this night was further subjected to a spectral analysis. Absolute power (averaged over 1 minute intervals) within the delta range during the first 360 minutes of sleep is illustrated in Figure 3.4. This gives an indication of change in slow wave activity (SWA) across the night without being subject to the rule-based criteria of sleep stage scoring. The progression of SWA activity for this night
indicates an abnormal delay in peak activity. For normal, healthy, non-sleep deprived subjects Borbély (1982) describes a pattern of 3-4 peaks in SWA throughout the night, with each peak showing a decrease in power relative to the previous one. This is explained in terms of a gradual reduction in Process S, seen as a pressure for SWA, as sleep continues. For this subject, power in the first two cycles appeared to be reduced relative to the final cycle. One explanation for this might be that this subject experienced undue anxiety at the start of the night which was prohibitive to the development of normal sleep. As the night progressed, pressure for SWA would be unrelieved, resulting in the eventual development of maximum delta power around 4.00 am. Further difficulties emerged for this subject as the experiment progressed (to be reported later).

Fig 3.4 Absolute power within the delta range throughout the first 6h of sleep for subject 5 (BASE7)
3.5.2. Daytime Sleepiness:

MSLT scores -

Daily MSLT scores for individual subjects for BASE7 are given in Table 3.4. The number of actual sleep onsets recorded throughout the day are included. Only one subject failed to sleep during any of the four trials. Sleep onset occurred during 50% of all trials.

<table>
<thead>
<tr>
<th>subject</th>
<th>daily mean MSLT (min)</th>
<th>actual sleep onsets</th>
</tr>
</thead>
<tbody>
<tr>
<td>s1</td>
<td>5.6</td>
<td>4</td>
</tr>
<tr>
<td>s2</td>
<td>17.5</td>
<td>2</td>
</tr>
<tr>
<td>s3</td>
<td>19.5</td>
<td>1</td>
</tr>
<tr>
<td>s4</td>
<td>17.8</td>
<td>2</td>
</tr>
<tr>
<td>s5</td>
<td>18.5</td>
<td>2</td>
</tr>
<tr>
<td>s6</td>
<td>19.0</td>
<td>1</td>
</tr>
<tr>
<td>s7</td>
<td>20.0</td>
<td>0</td>
</tr>
<tr>
<td>s8</td>
<td>18.5</td>
<td>1</td>
</tr>
<tr>
<td>s9</td>
<td>7.3</td>
<td>4</td>
</tr>
<tr>
<td>s10</td>
<td>18.5</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 3.4 Daily mean MSLT scores throughout BASE 7

This is consistent with previous findings for healthy young subjects following an habitual schedule of 8 hours sleep at night (Carskadon & Dement, 1979; Carskadon & Dement, 1981; Clodore, Benoit, Forest & Bouard, 1990; frequency of sleep onset during all baseline MSLT trials = 50%, 40% and 53%, respectively). Scores ranged from 5.6 min to 20.0 min with a daily mean score for all subjects of 16.22 min. This was taken as the Baseline MSLT score for future analysis. There was a strong negative correlation between frequency of sleep onset and daily MSLT score (Pearson product moment; r = -0.91).

Daily mean MSLT scores also correlated positively with SPT (r=0.75), TST (r=0.62) and amount of REM sleep (r=0.65) during the previous night. There were no further significant correlations between MSLT scores and remaining nocturnal sleep parameters.
Mean latency scores for individual trials across the day are presented in Figure 3.6. A reduction in time to sleep onset throughout the afternoon trials compared with the morning trials coincided with an increase in the likelihood of sleep occurring.

**Figure 3.6 Change in mean MSLT score and Frequency of sleep onset throughout BASE7 trials (n=10)**

**Subjective Sleepiness: The Karolinska Sleepiness Scale**

Individual means for scores on the Karolinska Sleepiness Scale throughout BASE7 are given in Table 3.5. A high score on this scale indicates extreme sleepiness; a low score extreme alertness.

A mean daily score for all subjects of 3.1 roughly equates to a feeling of 'ALERT' on this scale. Throughout the day scores ranged from 1 (EXTREMELY ALERT) through to 6 (between 5 - NEITHER ALERT NOR SLEEPY and 7 - SLEEPY
BUT NOT FIGHTING SLEEP.). Extreme levels of alertness as opposed to sleepiness were more frequently reported.

Sleep latencies did not correlate with subjective ratings taken immediately before MSLT trials. Unlike MSLT scores mean values for subjective sleepiness for individual trials did not show a clear pattern of change across the day (mean scores on Karolinska Sleepiness Scale: 1000h - 3.3; 1200h - 2.7; 1400h - 3.3; 1600h - 3.1). This was due to individual differences in the direction of change across the day.

<table>
<thead>
<tr>
<th>subject</th>
<th>mean score</th>
<th>range throughout day</th>
</tr>
</thead>
<tbody>
<tr>
<td>s1</td>
<td>3.5</td>
<td>1-5</td>
</tr>
<tr>
<td>s2</td>
<td>3.8</td>
<td>3-5</td>
</tr>
<tr>
<td>s3</td>
<td>3.8</td>
<td>2-5</td>
</tr>
<tr>
<td>s4</td>
<td>3.5</td>
<td>3-4</td>
</tr>
<tr>
<td>s5</td>
<td>2.8</td>
<td>2-4</td>
</tr>
<tr>
<td>s6</td>
<td>2.3</td>
<td>1-4</td>
</tr>
<tr>
<td>s7</td>
<td>3.0</td>
<td>2-4</td>
</tr>
<tr>
<td>s8</td>
<td>3.3</td>
<td>2-6</td>
</tr>
<tr>
<td>s9</td>
<td>1.5</td>
<td>1-2</td>
</tr>
<tr>
<td>s10</td>
<td>3.8</td>
<td>2-6</td>
</tr>
</tbody>
</table>

Table 3.5 Daily mean scores on the Karolinska Sleepiness Scale for all subjects throughout BASE7.

Six subjects showed a clear increase in subjective sleepiness during the afternoon, whilst the remaining four subjects experienced a reduction in subjective sleepiness as the day progressed. Differences between these two groups are illustrated in Figures 3.7a and 3.7b.
Subjects experiencing an increase in subjective sleepiness throughout the day (base 7 - n=6)

![Graph showing increased subjective sleepiness throughout the day with data points for subjects S1 to S9.](image)

**Figure 3.7a Increased subjective sleepiness throughout BASE7.**

There was no correlation between estimated quality of sleep at night, duration of sleep at night and subjective sleepiness at any point throughout the day. Differences between the pattern of change in alertness throughout the day are likely to reflect a combination of fixed traits (i.e. circadian type) and attitudinal differences to rating scales. The degree of relative change from the first measurement of the day was not suggestive of extreme change. Nor was there any evidence of extreme dips in alertness, or periods of overwhelming sleepiness for any of the subjects.

Subjects experiencing a reduction in subjective sleepiness throughout the day (Base 7 - n=4)

![Graph showing reduced subjective sleepiness throughout the day with data points for subjects S5, S6, S8, and S10.](image)

**Figure 3.7b Reduced subjective sleepiness throughout BASE7.**
3.5.3. Mood : The Profile of Mood States

A single score for separate states of Fatigue & Vigor was calculated for each subject, at each testing time, according to the guidelines of The Profile of Mood States (Mc Nair et al, 1971). This involved marking each adjective within a scale as follows:

NOT AT ALL = 0
A LITTLE = 1
MODERATELY = 2
QUITE A BIT = 3
EXTREMELY = 4

A total score was calculated for individual states of Vigor and Fatigue by summing the scores of adjectives attributable to that state. See Appendix A4 for further details of POMS Vigor and Fatigue mood states.

**On waking**

Daily mean POMS Vigor and Fatigue scores taken from the morning sleep dairies for the seven mornings of the baseline period are shown in Table 3.6.

<table>
<thead>
<tr>
<th>morning</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatigue</td>
<td>5.1</td>
<td>3.9</td>
<td>4.9</td>
<td>7.3</td>
<td>6.9</td>
<td>5.6</td>
<td>5.0</td>
</tr>
<tr>
<td></td>
<td>(5.0)</td>
<td>(1.6)</td>
<td>(2.2)</td>
<td>(6.8)</td>
<td>(6.7)</td>
<td>(6.9)</td>
<td>(1.8)</td>
</tr>
<tr>
<td>Vigor</td>
<td>10.1</td>
<td>11.1</td>
<td>9.7</td>
<td>8.4</td>
<td>9.1</td>
<td>10.0</td>
<td>6.1</td>
</tr>
<tr>
<td></td>
<td>(11.1)</td>
<td>(6.6)</td>
<td>(8.9)</td>
<td>(9.5)</td>
<td>(9.9)</td>
<td>(9.9)</td>
<td>(4.7)</td>
</tr>
</tbody>
</table>

Table 3.6 Mean Vigor and Fatigue (SDs) on waking during BASELINE period (n=10).

Maximum scores possible for Vigor & Fatigue are 32 and 28, respectively. Scores along both scales are relatively low and stable throughout this period. Large standard deviations for all scores reflect considerable individual differences in subjective estimations of mood states.
Before each MSLT Trial

Mean subjective ratings of Vigor & Fatigue taken before each MSLT trial throughout BASE7 are presented in Table 3.7. Large standard deviations suggest considerable inter-individual differences in estimating mood.

<table>
<thead>
<tr>
<th></th>
<th>1000h</th>
<th>1200h</th>
<th>1400h</th>
<th>1600h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatigue</td>
<td>3.33 (2.73)</td>
<td>2.39 (2.22)</td>
<td>3.29 (2.71)</td>
<td>3.56 (3.05)</td>
</tr>
<tr>
<td>Vigor</td>
<td>14.06 (6.04)</td>
<td>15.17 (5.04)</td>
<td>14.89 (6.46)</td>
<td>14.33 (6.35)</td>
</tr>
</tbody>
</table>

Table 3.7 Mean Vigor and Fatigue (SDs) at testing points throughout BASE7 (n=10)

Visual inspection of the data suggested that individual reference points along the scale varied widely. In particular it was noted that scores from high scoring subjects influenced the means disproportionately. An alternative approach was adopted in an effort to avoid problems due to inter-individual differences in scoring levels. This was aimed at reducing the relative contribution of extreme scores to overall means for Vigor and Fatigue. A mean score was calculated for each component (i.e. each adjective) of the state. The effect of this on overall mood states is illustrated in Figures 3.8a and 3.8b. For both Vigor and Fatigue, differences between trials when calculated as the sum of component means is very similar to means of conventional subject scores for each trial (see Table 3.6). Both scoring approaches suggest that, whilst Fatigue is increased at 1000h relative to all other times of day Vigor is at its lowest at this time. At 1200h subjects experienced lowest levels of fatigue and highest levels of Vigor for that day.
Fig 3.8a Mean Fatigue throughout the day (BASE 7 - n=10)

Fig 3.8b Mean Vigor throughout the day (BASE 7 - n=10)
3.5.4 Time course of sleepiness variables across the day.

Visual analysis indicated differences in the time course of the sleepiness variables across the testing day (BASE7). Lowest levels of physiological sleepiness (MSLT scores) were measured for the first trial of the day (1000h) with a gradual increase in sleepiness throughout each subsequent trial. On the other hand, subjective sleepiness measured using the KSS indicated no systematic change throughout the day. As previously detailed, subjects varied widely in the development of subjective sleepiness relative to the start of the day. Differences throughout the day were apparent between mood scores. Fatigue was at its highest at the 1000h and showed a gradual decline throughout the day. Vigor showed an opposite change, with lowest levels at 1000h and a gradual increase throughout the day.

Changes across the day are illustrated for each variable as relative change from the first trial (1000h), see Figure 3.9.

![Figure 3.9 Time course of sleepiness (MSLT and KSS) and POMS Vigor and Fatigue throughout BASE7 (n=10)](image)

It can be seen that the greatest reduction in Fatigue occurred between 1000h and 1200h. Afternoon trials showed a similar, although less marked reduction in Fatigue from the 1000h. Vigor, also showed greatest change between the 1000h and 1200h...
trials, although a marked increase in vigor from the first trial was experienced throughout the day. In contrast, actual physiological sleepiness, measured as latency to sleep onset using the MSLT showed a gradual increase as the day progressed. Whilst there was very little difference between the two morning trials, throughout the afternoon physiological sleepiness increased noticeably.

Between test differences were explored using a Friedman ANOVA for each of the variables (MSLT, KSS, Fatigue, VIGOR) followed by Wilcoxon signed rank tests where appropriate. The following significant findings emerged:

<table>
<thead>
<tr>
<th>Measure</th>
<th>Friedman ANOVA</th>
<th>Wilcoxon Signed Rank Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>MSLT</td>
<td>df=3, chi-square=14.51, p&lt;0.001</td>
<td>T1&gt;T3 (p=0.01) T1&gt;T4 (p=0.01) T2&gt;T4 (p=0.04) T1 &amp; T2 ns T2 &amp; T3 ns T3 &amp; T4 ns</td>
</tr>
<tr>
<td>KSS</td>
<td>ns</td>
<td></td>
</tr>
<tr>
<td>Fatigue</td>
<td>df=3, chi-square=8.31, p&lt;0.05</td>
<td>T1&gt;T2 (p=0.02) T1&gt;T3 (p=0.04) no diff between remaining trials</td>
</tr>
<tr>
<td>Vigor</td>
<td>df=3, chi-square=12.41, p&lt;0.005</td>
<td>T1&lt;T2 (p=0.01) T1&lt;T3 (p=0.03) T1&lt;T4 (p=0.05) T2&gt;T3 (p=0.02) T2&gt;T4 (p=0.01) T3&amp;T4 = ns diffs</td>
</tr>
</tbody>
</table>

As predicted, morning trials on the MSLT showed an increased sleep latency compared with afternoon trials. Change in relative frequency of sleep onset throughout the day (refer to Fig 3.6) suggests that for the morning trials, longer sleep latencies reflect a low probability of actual sleep occurring. For these subjects,
physiological sleepiness only begins to have a measurable effect during the afternoon, as evidence by both increased sleep onset frequency and shortened sleep latencies. This progressive increase in physiological sleepiness was not detected using a subjective measure of sleepiness (KSS). Mood scores were indicative of reduced Vigor and increased Fatigue for the first trial of the day. This was not reflected in performance during the MSLT or KSS ratings.

3.5.5. Performance: The Wilkinson Auditory Vigilance Task

Vigilance scores for the BASELINE testing period are summarised in Table 3.8. Scores were consistently high for all subjects. Scores for Misses or False Positives were low throughout.

<table>
<thead>
<tr>
<th></th>
<th>(n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>probability of a HIT</td>
<td>0.84</td>
</tr>
<tr>
<td></td>
<td>(0.14)</td>
</tr>
<tr>
<td>Dprime</td>
<td>3.86</td>
</tr>
<tr>
<td></td>
<td>(1.17)</td>
</tr>
<tr>
<td>RT (ms)</td>
<td>602.4</td>
</tr>
<tr>
<td></td>
<td>(145.1)</td>
</tr>
</tbody>
</table>

Table 3.8 Mean (SD) scores for 55 minutes Wilkinson Auditory Vigilance Task during BASE5.

Reaction times for each correct HIT were separated into four blocks of 12 minutes to explore time on task characteristics, see Figure 3.10. Responses from the start (5 min) and end (2 min) of the test were ignored to avoid distortions due to a settling period or end of test effort. Means for each block were compared using the Wilcoxon Signed Rank Test. A significant reduction in reaction time was found between block 1 and block 2. Differences between all other blocks were non-significant. This suggests that at this level of alertness the Wilkinson Auditory Vigilance Task remains sensitive to time on task changes in the ability to perform. Despite adequate training during previous sessions, by the 17th minute of this test subjects react more slowly whilst maintaining a high level of accuracy throughout.
Table 3.10 Mean Reaction Time throughout the Wilkinson Auditory Vigilance Task (Base7 - n=10)

3.6 DISCUSSION - PART I

Measuring the consequences of sleep at night; inconsistencies between tests.

Throughout the baseline period actigraphs and subjective reports confirmed subjects' estimations of preferred sleep times. With the exception of one subject, analysis of overnight EEGs recorded during the final night of the baseline period provided evidence that these subjects were indeed 'average' in their sleep habits. SPTs and TSTs showed sleep duration to be around 7.5 - 8.0h with moderate amounts of disturbance during sleep. All other sleep state parameters were normal.

Mean MSLT scores throughout the day indicated actual levels of physiological sleepiness to be relatively low following 7.5-8.0 hours sleep. A mean daily score of around 16 min is interpreted in line with the recommendations of the American Sleep Disorders Association (1992) as less than "mild" levels of sleepiness for these subjects. This is with the exception of two subjects for whom MSLT scores were relatively low: daily mean score of 7.3 and 5.6 min. Subjective sleepiness was also found to be relatively low with only modest change throughout the day.
Further analysis of sleepiness variables in relation to POMS mood scores for Vigor and Fatigue showed that each measure followed a different time course across the day. There was a period of relatively intense Fatigue and reduced Vigor during the first morning trial (1000h) which had effectively dissipated by noon. Levels of Fatigue and Vigor at 1000h, which together might be described as reflecting a subjective 'Vitality', were difficult to reconcile with a view of physiological sleepiness which suggested that, for this time of the day, sleepiness was at its lowest point. Furthermore, change in relative 'Vitality' throughout the day was incongruous with change in physiological sleepiness.

One explanation for this is that the early morning levels of mood found for these subjects are characteristic of a residual sleep inertia. Sleep inertia has been described by Folkard & Åkerstedt (1992) as a familiar complaint following normal sleep. They add that, despite indications of sleepiness, actual sleep is unlikely to follow. Hence the discrepancies in this study between subjective ratings and likelihood of sleep during the 1000h trial.

However, it is also seems likely that changes in subjective 'Vitality' throughout the day play an important role in the manifestation of physiological sleepiness. For example, although latency to sleep onset is shortened throughout the day, it could be argued that a relative increase in 'Vitality' would be sufficient, in terms of increased motivation, to influence an individual's willingness to resist physiological sleepiness. Thus, willingness to combat sleepiness is likely to be an important factor in avoiding sleep in a real-world setting.
3.7 Results - Part II

Sleeping in excess of 8 hours

3.7.1 Night-time Sleep

Actigrams - Mean SPTs for each night of the study are shown in Figure 3.11. It can be seen that for the first seven BASE nights SPT remains relatively stable at about 7.5 h, then rises to 9.4 h on the first EXT night, steadily declines to about 8.6 h over the EXT period and then drops back to 7.5 h on REC.

Overnight EEGs - Changes to sleep parameters over these nights are shown in Table 3.9 (also giving significance levels).

<table>
<thead>
<tr>
<th></th>
<th>BASE7</th>
<th>EXT7</th>
<th>EXT14</th>
<th>REC4</th>
<th>MANOVA; F= (df=3.27)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep Period</td>
<td>446.95 (42.37)</td>
<td>532.35 (24.77)</td>
<td>529.60 (38.44)</td>
<td>424.55 (41.34)</td>
<td>34.00; p&lt;0.01</td>
</tr>
<tr>
<td>Time (SPT)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Sleep</td>
<td>412.00 (41.37)</td>
<td>477.90 (22.68)</td>
<td>471.45 (44.96)</td>
<td>390.95 (37.59)</td>
<td>17.84; p&lt;0.01</td>
</tr>
<tr>
<td>Time (TST)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 1</td>
<td>44.65 (19.46)</td>
<td>75.05 (36.39)</td>
<td>69.20 (31.53)</td>
<td>51.55 (29.56)</td>
<td>4.67; p&lt;0.01</td>
</tr>
<tr>
<td>Stage 2</td>
<td>170.45 (26.33)</td>
<td>197.25 (29.00)</td>
<td>201.30 (40.63)</td>
<td>141.15 (47.48)</td>
<td>10.18; p&lt;0.01</td>
</tr>
<tr>
<td>Stage 1 &amp; 2(total)</td>
<td>215.10 (34.07)</td>
<td>272.30 (27.99)</td>
<td>270.50 (32.29)</td>
<td>192.70 (51.51)</td>
<td>15.53; p&lt;0.01</td>
</tr>
<tr>
<td>Stage 3</td>
<td>42.75 (12.39)</td>
<td>44.80 (11.20)</td>
<td>43.95 (8.96)</td>
<td>42.65 (11.28)</td>
<td></td>
</tr>
<tr>
<td>Stage 4</td>
<td>49.65 (24.96)</td>
<td>44.95 (24.37)</td>
<td>39.45 (26.57)</td>
<td>43.70 (30.96)</td>
<td></td>
</tr>
<tr>
<td>REM</td>
<td>104.40 (19.51)</td>
<td>115.85 (21.78)</td>
<td>117.55 (31.04)</td>
<td>111.90 (28.91)</td>
<td></td>
</tr>
<tr>
<td>Wake After Sleep Onset</td>
<td>34.70 (11.69)</td>
<td>54.45 (26.39)</td>
<td>58.15 (25.22)</td>
<td>33.60 (13.10)</td>
<td>3.53; p&lt;0.05</td>
</tr>
<tr>
<td>(WASO)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep Latency</td>
<td>22.70 (8.84)</td>
<td>34.00 (12.53)</td>
<td>34.60 (18.54)</td>
<td>17.80 (8.47)</td>
<td>4.48; p&lt;0.05</td>
</tr>
<tr>
<td>Sleep Efficiency (%)</td>
<td>92.17 (2.59)</td>
<td>89.89 (4.64)</td>
<td>88.98 (4.98)</td>
<td>92.16 (2.87)</td>
<td></td>
</tr>
</tbody>
</table>

Table 3.9 Means (and SDs) of sleep structure during the three phases of the study (n=10).
Fig. 3.11. Actigraphically determined nocturnal sleep throughout baseline and extended recovery periods (n=10).
SPTs correspond quite well with their counterparts determined by actimetry (Figure 3.11), being for BASE 7, EXTs 7 and 14, and REC 4 respectively: 450 min. actimetry vs 447 min. by EEG, 520 min. actimetry vs 532 min. EEG, 515 min. actimetry vs 530 min. EEG, 440 min. actimetry vs 425 min. EEG.

MANOVAs on the above data give several significant findings, mostly as expected: TST increases by approximately one hour for both the EXT nights compared with the BASE. This increase comprises significantly more stages 1 and 2 sleep. SOL lengthens significantly during EXT nights, as does wake after sleep onset (WASO), and thus sleep efficiency also declines by a small but similarly significant extent. Post hoc Tukey tests showed the following significant differences at p<0.05: For Stage 1: EXT 7 greater than BASE 7; For Stage 2: EXTs 7 and 14 greater than REC 4; For Stages 1+2: EXTs 7 and 14 greater than BASE 7 and REC 4; For SPT, TST and WASO: EXTs 7 and 14 greater than BASE 7 and REC 4; for SOL: EXTs 7 and 14 longer than REC4.

Spectral Analysis Figure 3.12 shows mean absolute delta activity levels across 7 subjects, for BASE 7, EXT 14 and REC 4. The first six hours of sleep presented here represent the highest common denominator for uninterrupted sleep across these subjects and conditions. Sleep EEGs during the EXT nights for the other three subjects contained problematic amounts of wakefulness during this six hour period, which became difficult to integrate with the other data. Figure 3.12 shows a clear diminution in delta activity during EXT, particularly during the first cycle. Mean delta power levels for these three conditions are: BASE 240mV^2 (sd= 106 mV^2), EXT= 165 mV^2 (sd= 79 mV^2), REC= 249 mV^2 (sd= 104 mV^2). A Wilcoxon test (see below for the method used) showed a significant (P<0.05) reduction of delta power during EXT. Preliminary examination of spectra beyond the first 6 hours shows no compensatory increased rebounds of delta activity during the last part of EXT nights, and there would indeed seem to be an overall reduction in delta activity during EXT, which is probably at least partly due to the reduced length of prior wakefulness (Borbely, 1982).

3.7.2 Sleep Fragmentation

Rasta plots show periods of sleep and wake throughout the night during nights BASE 7, EXT 14 and REC 4 for these seven subjects (Fig 3.13a-c). These give an indication of individual difficulties in extending sleep. For example whilst subject DR
Fig. 3.12. Delta activity (0.75-4.0 Hz) throughout first 5 hours of sleep during baseline, extended & Recovery nights (n=7).

<table>
<thead>
<tr>
<th>Time From Sleep Onset (min)</th>
<th>Power MV²</th>
</tr>
</thead>
<tbody>
<tr>
<td>4:00:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:01:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:02:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:03:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:04:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:05:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:06:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:07:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:08:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:09:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:10:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:11:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:12:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:13:00</td>
<td>0.000</td>
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<tr>
<td>4:14:00</td>
<td>0.000</td>
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<tr>
<td>4:15:00</td>
<td>0.000</td>
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<tr>
<td>4:16:00</td>
<td>0.000</td>
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<tr>
<td>4:17:00</td>
<td>0.000</td>
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<tr>
<td>4:18:00</td>
<td>0.000</td>
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<tr>
<td>4:19:00</td>
<td>0.000</td>
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<tr>
<td>4:20:00</td>
<td>0.000</td>
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<tr>
<td>4:21:00</td>
<td>0.000</td>
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<tr>
<td>4:22:00</td>
<td>0.000</td>
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<tr>
<td>4:23:00</td>
<td>0.000</td>
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<tr>
<td>4:24:00</td>
<td>0.000</td>
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<td>4:25:00</td>
<td>0.000</td>
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<tr>
<td>4:26:00</td>
<td>0.000</td>
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<tr>
<td>4:27:00</td>
<td>0.000</td>
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<tr>
<td>4:28:00</td>
<td>0.000</td>
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<tr>
<td>4:29:00</td>
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<tr>
<td>4:30:00</td>
<td>0.000</td>
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<tr>
<td>4:31:00</td>
<td>0.000</td>
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<tr>
<td>4:32:00</td>
<td>0.000</td>
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<tr>
<td>4:33:00</td>
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<tr>
<td>4:34:00</td>
<td>0.000</td>
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<td>4:35:00</td>
<td>0.000</td>
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<td>4:36:00</td>
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<tr>
<td>4:37:00</td>
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<td>4:38:00</td>
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<tr>
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<td>4:40:00</td>
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<tr>
<td>4:41:00</td>
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<tr>
<td>4:42:00</td>
<td>0.000</td>
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<tr>
<td>4:43:00</td>
<td>0.000</td>
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<tr>
<td>4:44:00</td>
<td>0.000</td>
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<td>4:45:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:46:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:47:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:48:00</td>
<td>0.000</td>
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<tr>
<td>4:49:00</td>
<td>0.000</td>
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<td>4:50:00</td>
<td>0.000</td>
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<td>4:51:00</td>
<td>0.000</td>
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<tr>
<td>4:52:00</td>
<td>0.000</td>
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<td>4:53:00</td>
<td>0.000</td>
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<td>4:54:00</td>
<td>0.000</td>
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<td>4:55:00</td>
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<tr>
<td>4:56:00</td>
<td>0.000</td>
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<tr>
<td>4:57:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:58:00</td>
<td>0.000</td>
</tr>
<tr>
<td>4:59:00</td>
<td>0.000</td>
</tr>
</tbody>
</table>

Legend:
- Rec
- Ext
- Base
Fig. 3.13a. Sleep & Wakefulness Across the Night during Night 7 of Baseline Sleep

<table>
<thead>
<tr>
<th>Subject</th>
<th>Rise Time</th>
<th>Time</th>
<th>Lights On</th>
<th>Lights Off</th>
</tr>
</thead>
<tbody>
<tr>
<td>AS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VN</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TB</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>AM</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>JR</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FN</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DR</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Ch 3: Extended Sleep

[Graph depicting sleep patterns with labels for each subject (AS, N1, T1B, and others).]

---

Figure 3.13B: Sleep as Wakefulness Across the Night during Night 14 of Extended Sleep
FIG 3.13c: Sleep & Wakefulness across the night during Night 4 of Recovery Sleep
showed long episodes (approx. 10-15 min. of wake during the night for EXT14) this was not dissimilar to the pattern of wake experienced by this subject during BASE7 & REC4. However, long periods of wake during night EXT14 for subjects MN and AM is quite different to the continuous, highly efficient sleep experienced by these subjects during BASE7 and REC4.

### 3.7.3 Daytime Sleepiness

**MSLT Scores** Mean values are given in Table 3.10, and changes across conditions are further illustrated as "survival" curves for each testing day (Fig 3.14), expressed as the percentage of subjects scored as asleep each minute throughout all trials.

![Fig 3.14 Change in sleep propensity throughout MSLT trials (n=10)](image)

MSLT scores are displayed to reflect the % of subjects scored as unequivocally asleep at time intervals throughout the trial on Baseline, Extended and Recovery sleep schedules.

After 7, 11 and 14 nights of EXT there appears to be less likelihood of failing asleep during an MSLT than following BASE or REC nights. However, under these latter conditions the relatively high proportion of 20 min. scores violates the assumption of interval or ratio data for the application of parametric statistics, and so the Wilcoxon Signed Rank Test was used. For each subject the baseline MSLT score was subtracted from the respective scores for EXT4, EXT7 EXT11, and EXT14.
prediction that MSLT scores were greater during EXT than BASE was tested by ranking the differences by magnitude from highest positive to lowest negative. Significantly more higher ranked positive scores would support that hypothesis. The results were non-significant.

<table>
<thead>
<tr>
<th></th>
<th>BASE7</th>
<th>EXT4</th>
<th>EXT7</th>
<th>EXT11</th>
<th>EXT14</th>
<th>REC4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily</td>
<td>16.2</td>
<td>16.7</td>
<td>16.9</td>
<td>16.7</td>
<td>16.9</td>
<td>16.0</td>
</tr>
<tr>
<td>MSLT</td>
<td>(4.9)</td>
<td>(4.4)</td>
<td>(5.0)</td>
<td>(5.2)</td>
<td>(5.3)</td>
<td>(5.2)</td>
</tr>
</tbody>
</table>

Table 3.10. Means (and SDs) of daily MSLT scores during the three phases of the study (n=10).

Figure 3.15 shows mean MSLT scores across the conditions for the four times of day of the test. There is a small but more obvious improvement during EXT during the 16:00 h session, which, by using the above test, gives a significant result \([W=4; N=10; p<0.01]\). The other times of day were non-significant.
**Subjective Sleepiness** - The group means for the Karolinska sleepiness scale obtained on the testing days (Fig 3.1) are shown in Table 3.11. A MANOVA showed no significant changes. Similarly, there were no significant changes during EXT for the sleepiness ratings obtained from the morning sleep diaries.

<table>
<thead>
<tr>
<th>Karolinska Sleepiness Scale (daily mean)</th>
<th>BASE7</th>
<th>EXT4</th>
<th>EXT7</th>
<th>EXT11</th>
<th>EXT14</th>
<th>REC4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3.4 (1.2)</td>
<td>3.5 (1.3)</td>
<td>3.0 (0.9)</td>
<td>3.1 (1.0)</td>
<td>2.8 (1.0)</td>
<td>3.5 (1.3)</td>
</tr>
</tbody>
</table>

Table 3.11 Means (and SDs) of Karolinska Sleepiness Scale during the three phases of the study (n=10).

Figures 3.16a and 3.16b show mean subjective sleepiness scores across the conditions for mornings and afternoons. This showed a trend towards increased alertness (lower scores) at 1000h morning during the extended sleep phase (EXT7-14) in comparison with BASELINE and RECOVERY scores, however this was non-significant.

![Figure 3.16a Mean subjective sleepiness scores during the morning throughout each testing day (n=10)](image-url)

Figure 3.16a Mean subjective sleepiness scores during the morning for BASE, EXT and REC days (n=10)
During the afternoon change in subjective sleepiness was haphazard.

### 3.7.4 Mood : The Profile of Mood States

Daily mean POMS Vigor and Fatigue scores from the morning sleep diaries reveal no significant or otherwise obvious changes during EXT or REC. These scores were assessed specifically for each testing day: i) on awakening, and ii) for the rest of the day. The mean values are shown in Table 3.12.

The small improvements in Vigor and Fatigue on awakening during EXT testing days (see Figure 3.17) were not significant (MANOVA), and neither were those for the whole day. With respect to the Worn Out syndrome, whilst most subjects were able to tolerate the experimental requirements with no obvious negative effect on mood, one subject out of the ten showed clear signs of a depressed and irritable mood throughout EXT, which manifested itself in a reduction in Vigor and increase in Fatigue each morning throughout this period. This was partially alleviated with a return to REC, see Figure 3.18.

---

**Figure 3.16b** Mean subjective sleepiness scores during the afternoon for BASE, EXT and REC days (n=10).

During the afternoon change in subjective sleepiness was haphazard.
### Table 3.12 Means (and SDs) of POMS Vigor and Fatigue scales:

1. **on morning awakening for all days,** and
2. **throughout the testing days.**

#### Mood on Waking (0800h)

<table>
<thead>
<tr>
<th></th>
<th>BASE</th>
<th>EXT4</th>
<th>EXT7</th>
<th>EXT11</th>
<th>EXT14</th>
<th>REC4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>VIGOUR</strong></td>
<td>7.8 (7.6)</td>
<td>9.7 (6.2)</td>
<td>9.4 (4.8)</td>
<td>10.6 (6.2)</td>
<td>9.3 (7.1)</td>
<td>8.3 (7.2)</td>
</tr>
<tr>
<td><strong>FATIGUE</strong></td>
<td>5.7 (3.7)</td>
<td>4.2 (4.8)</td>
<td>3.8 (5.2)</td>
<td>4.2 (5.7)</td>
<td>5.7 (4.4)</td>
<td>6.4 (3.5)</td>
</tr>
</tbody>
</table>

#### Mood throughout Day (1000-1600h)

<table>
<thead>
<tr>
<th></th>
<th>BASE</th>
<th>EXT4</th>
<th>EXT7</th>
<th>EXT11</th>
<th>EXT14</th>
<th>REC4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>VIGOUR</strong></td>
<td>14.6 (5.8)</td>
<td>12.8 (5.3)</td>
<td>12.8 (5.1)</td>
<td>11.9 (4.7)</td>
<td>13.5 (6.4)</td>
<td>13.0 (6.1)</td>
</tr>
<tr>
<td><strong>FATIGUE</strong></td>
<td>3.4 (2.7)</td>
<td>3.6 (3.8)</td>
<td>4.1 (5.4)</td>
<td>3.6 (4.1)</td>
<td>3.4 (4.9)</td>
<td>3.7 (3.8)</td>
</tr>
</tbody>
</table>

![Mood on Waking throughout all Testing Periods (n=10)](image)

**Figure 3.17** Moderate improvements in vigor and fatigue on waking (n=10).
3.7.5 Performance: The Wilkinson Auditory Vigilance Task

Vigilance scores for each testing period are summarised in Table 3.13. EXT results are the average of the two testing periods throughout EXT. The probability of a HIT and D prime show small but non-significant improvements during EXT, which return to BASE levels during REC.

<table>
<thead>
<tr>
<th></th>
<th>BASELINE</th>
<th>EXTENDED</th>
<th>RECOVERY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Probability of a HIT</td>
<td>0.84</td>
<td>0.87</td>
<td>0.80</td>
</tr>
<tr>
<td></td>
<td>(0.14)</td>
<td>(0.13)</td>
<td>(0.22)</td>
</tr>
<tr>
<td>Dprime</td>
<td>3.86</td>
<td>4.15</td>
<td>3.93</td>
</tr>
<tr>
<td></td>
<td>(1.17)</td>
<td>(0.90)</td>
<td>(1.02)</td>
</tr>
<tr>
<td>Reaction Time (ms)</td>
<td>602.4</td>
<td>527.4</td>
<td>592.2</td>
</tr>
<tr>
<td></td>
<td>(145.1)</td>
<td>(150.1)</td>
<td>(164.7)</td>
</tr>
</tbody>
</table>

Table 3.13 Mean (SD) scores throughout sleep conditions for 55 Wilkinson Auditory Vigilance Task (n=10)

There was a significant difference in reaction times between the three sleep conditions (BASE, EXT, REC), (Friedman ANOVA; chi-square = 12.2; df=2; p<0.005). Individual change in reaction time across conditions can be seen in Figure 3.19. The direction of change was relatively consistent. Wilcoxon matched pairs signed rank tests showed i)BASE and REC did not differ significantly ii)EXT significantly different from both BASE (P<0.01) and REC (p<0.005).
Ch 3: Extended Sleep

Figure 3.19. Individual reaction time throughout different alerting conditions for three sleep conditions.
The relationship between performance and EEG activity throughout BASE, the second EXT and REC tests was explored for a subgroup of six subjects using the following measures:

(i) probability of a HIT
(ii) mean reaction time across the test
(iii) mean theta activity throughout the test as the average of the 48 absolute power values (calculated at 1 minute intervals) within this range.
(iv) total theta activity as the sum of the 48 absolute power values within this range.
(v) probability of Wake - expressed as the percentage of test time with a probability of Wake between 0.9-1.0 (QUESTAR analysis).

Table 3.14 shows means and SDs for each of the measures across the 6 subjects. Spearman rank correlations revealed the following significant relationships: a positive correlation between probability of wake and HITS ($r=0.73; p<0.005$), a negative correlation between total theta and HITS ($r=-0.73; p<0.005$), and a negative correlation between total theta and probability of wake ($r=-0.49; p<0.05$).

<table>
<thead>
<tr>
<th>Measure</th>
<th>BASE</th>
<th>EXT</th>
<th>REC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prob of HITS</td>
<td>0.85</td>
<td>0.82</td>
<td>0.78</td>
</tr>
<tr>
<td></td>
<td>(0.15)</td>
<td>(0.17)</td>
<td>(0.27)</td>
</tr>
<tr>
<td>RT (ms)</td>
<td>550.50</td>
<td>487.33</td>
<td>611.20</td>
</tr>
<tr>
<td></td>
<td>(132)</td>
<td>(116)</td>
<td>(119)</td>
</tr>
<tr>
<td>Mean Theta</td>
<td>42.45</td>
<td>48.75</td>
<td>42.42</td>
</tr>
<tr>
<td></td>
<td>(10.93)</td>
<td>(18.23)</td>
<td>(39.56)</td>
</tr>
<tr>
<td>Total Theta</td>
<td>1978.00</td>
<td>2101.17</td>
<td>2022.20</td>
</tr>
<tr>
<td></td>
<td>(524.85)</td>
<td>(922.75)</td>
<td>(1904.54)</td>
</tr>
<tr>
<td>Prob of Wake (%)</td>
<td>83.67</td>
<td>77.72</td>
<td>84.52</td>
</tr>
<tr>
<td></td>
<td>(8.16)</td>
<td>(18.51)</td>
<td>(16.96)</td>
</tr>
</tbody>
</table>

Table 3.14 Means and SDs of EEG and performance measures throughout three sleep conditions ($n=6$)

Comparisons between sleep conditions were not considered appropriate due to the small number of subjects involved. As noted previously, apart from reaction times, statistical analyses of the entire subject group failed to reveal significant differences in performance measures following extended sleep. For both EEG measures (theta activity and probability of Wake), the means for each sleep condition were similar,
although variability in theta activity was considerably increased throughout both EXT and REC testing sessions.

### 3.8 DISCUSSION - Part II

*Change in daytime sleepiness, performance and mood following more sleep at night*

Figure 3.20 summarises the main findings of the study. Subjects can sustain about an extra hour of sleep at night, for at least 14 nights, but for only marginal improvements in the MSLT and in psychological performance, and doubtful improvements to subjective sleepiness. The extra sleep is at some cost: a longer sleep onset latency, more interim wakefulness, and reduced sleep efficiency. In conclusion, these subjects were not chronically sleep deprived to any marked degree, despite being able to increase their night-time sleep to a significant extent.

![Graph showing sleep at night and MSLT Score](image)

**Fig 3.20 Daytime sleepiness in relation to nocturnal sleep length (n=10)**

In these circumstances being able to take extra sleep would not seem to be indicative of a physiological need for this sleep. An analogy is that the total amounts of food and drink we consume are not wholly at the behest of physiological needs, as many people eat and drink in excess of these needs (particularly with fluid intake). Such excesses
are influenced by psychological and sociological factors, as well as by "indulgence" if the opportunities to eat and drink to excess are present. Thus, this extra sleep would seem to be "optional" (Horne, 1988)

It might be argued that a group mean MSLT score of 16 min for these subjects indicates that they were not sleepy to begin with, and thus there may have been very little room for improvement. That is, they had little physiological need for more sleep. Nevertheless, the key point is that they were still able to extend their sleep. So, if they were not sleepy to begin with why were they able to extend sleep? On the other hand, if they actually needed more sleep then, either this does not have any bearing on daytime sleepiness, or they were more sleepy than both they realised and objective measures indicated. Parsimony points to these findings indicating that subjects can extend sleep without necessarily being sleepy during the day, or showing marked reductions in sleepiness with extended sleep.

Throughout 14 nights of EXT, daily MSLT scores were improved by one minute on average. Notwithstanding no statistical significance overall, this effect is consistent with previous studies of an association between MSLT scores and nocturnal sleep duration (Carskadon & Dement, 1982), particularly with regard to extended sleep studies (Carskadon & Dement, 1979; Carskadon et al, 1986; Roehrs et al, 1989). The main effect of extended sleep on MSLT trials occurred during the afternoon.

In addition to this, weekly testing using the Wilkinson Auditory Vigilance Task revealed significant changes between BASE and EXT for reaction times, but not for hits of D'. Levels of theta activity in the EEG were found to be loosely related to performance (probability of HIT increased with reduced theta and increased probability of WAKE) although between condition differences, other than increased variability in the likelihood of theta following EXT and REC sleep, were not found. This increased variability in theta activity may have resulted from a combination of boredom and increased confidence with the test requirements which, despite familiarisation exercises, would have allowed the subject to relax more and concentrate less throughout later tests. It should be noted that, actual levels of performance remained high throughout the study period.

Reaction times were improved (approx. 12% faster) during EXT compared with BASE, and returned to near BASE levels with REC sleep. This is comparable with the improvements in reaction times reported by Roehrs et al (approx. 10% faster following 6 nights of extended sleep). Significant improvements in afternoon sleep
latencies and faster reaction times (also scheduled for mid-afternoon) suggest that the afternoon dip in alertness might be partially avoided following a regime of extended sleep at night.

For some researchers this might represent a convincing argument for advocating more sleep at night for the average individual. However, for those individuals particularly vulnerable to a period of reduced alertness during the afternoon, a more convenient and equally effective solution would be to take a short nap at a suitable point throughout the day. For example, Gillberg, Kecklund, Axelsson & Åkerstedt (1994) found a short nap to be effective in maintaining performance throughout both mornings and afternoon trials of a vigilance task following a night of restricted (4h) sleep. As for the optimal duration of such a nap, Naitoh (1992) concluded that a nap as short as 4 min (and no more than 20 min) can be sufficient to maintain performance. As a more practical option, a short nap has the added benefit of being more easily and discretely incorporated into an individual's existing schedule, e.g. during a working break, unlike extending sleep at night (the subjects in this study spent an extra 2h in bed to achieve an extra 1h of sleep). It is highly likely therefore, that the most promising potential benefit from extending sleep at night could be achieved equally successfully by other means, and with less disruption to habitual daily patterns.

There was some indication of enhanced mood on waking throughout the extended sleep period, which may have been due to an earlier than normal bedtime (Totterdell et al, 1994) rather than a direct consequence of sleeping longer. Other than this, levels of fatigue, vigor and subjective sleepiness were not enhanced to any degree that could be seen to benefit these subjects. This is consistent with previous reports of a lack of association between MSLT scores, performance tests and subjective sleepiness (Carskadon et al, 1986; Manni et al, 1991). By differentiating between physiological and manifest sleepiness, Carskadon and Dement (1982) suggest that both performance and introspective measures of sleepiness are influenced by environmental and subjective factors, unlike the MSLT which is the only test reliably free from motivational influences. That is, subjects may be sleepy but do not realise it, or that they are putting extra compensatory effort into the performance tasks. However, Kribbs, Pack and Dinges (1994) found sleep latency to be reduced when the latency trial was immediately preceded by a 10 min auditory vigilance task. This suggests that the level of physiological sleepiness assumed to be directly accessed by the MSLT is facilitated by previous contextual variables. In addition to this, Alexander, Blagrove & Horne (1991) have shown that the MSLT is also vulnerable to
motivational variables within the subject. Thus, in measuring change following the manipulation of nocturnal sleep at night, a reliance on a single measure - the MSLT - may have been overstated.

Of the eleven original participants in this study, two subjects found extended sleep difficult to adapt to; one of whom withdrew. The remaining subjects were able to tolerate 14 nights of extended sleep with no adverse effects from sleeping longer at night. For these subjects, there was no evidence to suggest a detrimental effect of extended sleep on subjective sleepiness, or self-rated mood using POMS vigor and fatigue scales. This may have been due to the timing of the opportunity for extra sleep in relation to potential chronobiological influences on mood and subjective sleepiness. Recent reports suggest that mood throughout the day is enhanced following an earlier than normal bedtime (Totterdell et al, 1994), and that the timing of final awakening, more specifically the closeness to the circadian acrophase of the temperature rhythm, is associated with increased ratings of sleep quality, feeling refreshed and ease of awakening (Åkerstedt et al, 1994).

Noticeably, previous reports of impaired subjective sleepiness following extended sleep were specifically concerned with the consequences of ad lib sleep conditions, i.e. where extra sleep was gained at the end, rather than the beginning, of a normal sleep period. This includes, Taub (1981) who found increased subjective sleepiness, coupled with impaired performance, followed a single night of ad lib extended sleep (subjects determined the actual timing of sleep extension, resulting in both phase delayed onset and offset of sleep). The 'Worn Out' syndrome described by Globus (1969, 1970) is attributed to taking extra sleep by sleeping later than normal. Carskadon et al (1986) reported increased subjective sleepiness following a night of sleep extension with no prior sleep debt. In this case the timing of sleep onset was normal with final wake time being delayed until mid-morning. In studies in which sleep was extended using an earlier than normal bedtime, with normal wake times preserved, there have been no claims of a detrimental effect on subjective sleepiness or mood the following day (e.g. Carskadon & Dement, 1979; Roehrs et al, 1989; Wehr at al, 1993).

The findings of this study, showing that a sleep extension of about one hour can be maintained over a prolonged period complement those from short-term studies (see Introduction), and more recent long-term studies (Wehr et al, 1993; Roehrs et al, 1994). In contrast with previous studies, these subjects slept in their normal home environment throughout, therefore establishing the feasibility of taking more sleep in
the real world. Sleep quality during EXT nights is consistent with that reported by Wehr et al (1993) in that additional sleep can be accounted for primarily by longer periods of stages 1 and 2 sleep (in this case, approx. 56 min) and REM sleep (approx. 12 min). The reduction in stages 3+4 (about 9% by EXT 14) is more apparent when spectral analysis is used.

However, whilst excessive amounts of wake after sleep onset and increased sleep latency periods might be seen as evidence of undesirable sleep quality following extended sleep, it has also been argued that slow transition to sleep and intermittent wakefulness throughout sleep periods are features of 'normal' human sleep in the sleep satiated individual. Wehr et al (1993) point out the similarities between this pattern of sleep (found throughout 28 enforced "long" nights in darkness) and the sleep patterns followed by many animals. That is, these subjects had more efficient sleep during baseline because they were partially sleep deprived. In this view, the emergence of consolidated, rapid onset sleep is linked with the development of modern, industrial societies, and a dependence on artificial lighting in determining day/night periods.

But, Åkerstedt, Hume, Minors & Waterhouse (1994) found subjective sleep quality to be highly correlated with sleep efficiency. We can also infer from the widespread use of medication for the treatment of sleep onset and sleep maintenance difficulties that these latter features represent undesirable characteristics of nocturnal sleep for many adults. For these reasons, I have interpreted the enhancement of sleep efficiency as a valued aspect of sleep behaviour. As such, the emergence of increased wakefulness throughout the night is considered to be an undesirable consequence of extended sleep.

In conclusion, for changes in MSLT scores of around one minute, and modest improvements to reaction times following sleep extension, the ecological worth of such small changes seems doubtful. Unless it is possible to show a significant improvement to other aspects of daytime functioning as a direct result of sleep longer at night, then there is little "real-life significance" for the actual improvements in MSLT scores and reaction times. Conversely, despite 14 consecutive opportunities to sleep longer at night, there was no evidence to suggest that alertness, performance or subjective experience were compromised by sleeping between 7-8h at night.
4. Measuring Sleepiness

4.1 Measuring sleepiness in the emotionally indirect, sleep-satisfied individual.
4.1 Measuring sleepiness in the essentially alert, sleep satiated individual.

One of the main findings to emerge from experiment 1 was that of increased sleep time at night with only slight change to MSLT scores throughout the day. Although, this undermines the assumption that all sleep is purposeful in terms of satisfying a requirement for sleep, it is perhaps not surprising to find that subjects with a mean MSLT score of around 16 min at baseline showed no indication of excessive sleepiness using either subjective or performance related measures. Furthermore, individual differences in baseline sleepiness (MSLT scores) ranged from 5.6 to 20.0 min, yet the relatively 'sleepy' individuals did not feel any the worse for this, nor did their performance suffer relative to the more 'alert' subjects.

The following section deals with the question of how to evaluate the significance of MSLT scores for the essentially alert, sleep satiated individual. The first part highlights the difficulties involved in interpreting MSLT scores, or change following experimental manipulation, for subjects who frequently fail to register any level of sleepiness using this method. Alternative criteria aimed towards enhancing the sensitivity of the MSLT for this subject group were applied to the MSLT findings of the first experiment. Following from this, a second study examined the role of motivation in initiating sleep during the MSLT for the non-clinical, relatively alert subject. This involved encouraging subjects to apply more effort to falling asleep during the trials. Finally, a number of case studies are presented of subjects who were observed to fall asleep quickly during the MSLT, despite sufficient and regular sleep at night.

4.2 Using EEG to determine the onset of sleep.

Whilst current methods for the detection of sleep onset in both research and clinical settings often assume partial knowledge of the mechanisms involved in the initiation of sleep (e.g. the MSLT), a more thorough understanding would be an extremely valuable aid; not only in the treatment of patients for whom these mechanisms appear to have broken down, but also in order to prevent the inadvertent onset of sleep in potentially hazardous situations. Historically, attempts to study the onset of sleep have relied to a great extent on an intuitive understanding of sleep as a psychological state. Sleep is often described in terms of a number of generalised key features, with the assumption of a shared familiarity with sleep as a universal
periodic experience. Thus sleep is characterised in terms of the absence of conscious awareness, with the onset of sleep marked by a shift in perceptual attention away from external and towards internal events. At times this may include the intrusion of illogical thought or 'visual fantasies'. Whilst purposeful action is generally understood to be prohibited during sleep (Davis, Davis & Loomis, 1937).

As a direct result of focusing on the experiential qualities of sleep in this way considerable insight into the nature of sleep has been gained. For example, Davis et al (1937) were able to demonstrate an association between self-reported incidence of loss of awareness or sense of waking from sleep with changes in levels of alpha activity in certain areas of the cortex. Over the next half century interest in the process of falling asleep (and sleep in general) gathered pace, as more and more researchers endeavoured to reduce the process of sleep onset to a series of physiological events. To some extent this may have reflected a more generalised rejection of introspective techniques in the psychological sciences in favour of empirical measures, however the assumption that a state experienced primarily in the psychological sphere can be reduced effectively to a physiological level has presented researchers with a number of difficulties.

In so far as it is satisfactory to offer an operational definition of the sleep onset period, at least three approaches to the interpretation of EEG measurements can be identified. These include the determination of wake and sleep states through visual inspection of the EEG as standardised by Rechtschaffen & Kales (1968), the identification of a transitional period of reduced awareness leading to sleep as a combination of behavioural and EEG changes (Ogilvie, McDonagh, Stone & Wilkinson, 1988; Ogilvie, Wilkinson, Allison, 1989; Ogilvie, Simons, Kuderian, MacDonald & Rustenburg, 1991; Torsvall & Åkerstedt, 1988), and an interest in cyclical changes in arousal related EEG activity as an indication of impending sleep (Evans, 1992; 1993).

The standardised method of scoring episodes of EEG in terms of wake and sleep stages developed by Rechtschaffen & Kales (1968) is perhaps the most widespread and commonly used approach to the scoring of the recorded EEG. Following the recommendation of techniques for the detection and recording of EEG relevant to sleep, records are separated into discrete epochs of an arbitrary duration - usually between 20-30 sec. A single score (Wake, Movement, Sleep stage 1-4, or REM sleep) is assigned to each epoch according to criteria relating to both frequency and amplitude of the generated signal.
In describing the EEG changes during the transition from wake to sleep it becomes apparent from the recommendation of 30 sec epoch lengths, and the emphasis on a combination of both generalised and transient features within the context of neighbouring epochs, that the initiation of sleep is recognised as a gradual, rather than instantaneous process. Individual variation in EEG composition contributed further to the difficulties of identifying sleep onset and so preventing a description in terms of a single, predictable combination of events. As a result, in this view 'sleep' in the form of stage I sleep is signalled by either the appearance of 'relatively low voltage, mixed frequency EEG' predominantly in the range of 2-7Hz (for non-alpha producers), or the reduction of alpha activity to less than 50% of a 30 sec epoch (for alpha producers). In support of these general changes in the appearance of the EEG, confirmation of stage I is gained by the appearance of transient EEG features (vertex sharp waves) and slow rolling eye movements (SEMs).

As a means of detecting sleep onset, this method has been criticised over the apparent lack of synchronisation between EEG evidence of the emergence of sleep characteristics and both behavioural and subjective characteristics of sleep (Ogilvie et al, 1988; 1991). Ambiguity as to the relevance of stage I sleep to psychological aspects of sleep has led to support for a more conservative estimate of sleep onset as equivalent to criteria identified as stage 2 sleep by Rechtschaffen & Kales (1968).

As one of the most common techniques for the measurement of daytime sleepiness, the MSLT tends to incorporate a very simplistic view of the nature of sleep onset. In keeping with Rechtschaffen & Kales (1968) guidelines, sleep onset is verified with the added provision of requiring 3 consecutive 30 sec epochs of stage I sleep, or 1 epoch of another sleep stage (Carskadon et al, 1986; Roehrs & Roth, 1992). As this allows for some ambiguity in detecting sleep onset (periods of up to 14 sec of arousal related activity in any or all of the 3 epochs are acceptable within this criteria) Roehrs & Roth (1992) issued guidelines to scoring the MSLT suggesting that, unless the recording is unequivocal, it is preferable to wait until further 'clearer signs' become evident. For this purpose, sleep spindles and K complexes (features of stage 2 sleep) are suggested.

In summary, whilst the standardisation of Rechtschaffen & Kales (1968) offers a common framework for the exchange of knowledge and ideas in the study of sleep, it has perhaps proved to be most useful in the identification and separation of important key patterns of the EEG signal during sleep, enabling quantification of the resultant sleep 'stages'. Without consideration for the progressive nature of state
changes, as with the pattern of intermittent arousal frequently observed as sleep approaches (Evans, 1992, 1993), there is some limitation to the descriptive power of this approach. As an aid to the detection of sleep onset, many of the distinctions between waking and sleep EEG rely on the appearance or otherwise of unpredictable, non-universal features of which little is known concerning the sub-cortical mechanisms involved in their generation, or their functional significance to the process of sleep.

Rather than using a visual analysis of the EEG as a starting point, Ogilvie et al (1988, 1991), devised a series of experiments aimed at revealing parallel changes in behavioural and electrophysiological measures during the process of falling to sleep. For this purpose sleep onset was determined as the point at which there was a failure to respond to external stimuli. This stimuli was in the form of an irregular, faint auditory tone terminated by the depression of a switch device fitted to the inside of the hand.

Following a night of sleep deprivation (i.e. under increased pressure for sleep) subjects were instructed to press the switch at the sound of each tone as they attempted to sleep (Ogilvie et al, 1988). When EEG records were scored according to Rechtschaffen & Kales (1968) at the point of each stimuli onset it was found that, although these subjects were more likely to make a response during wake, they were still able to respond during both stage I and stage 2 sleep. Even so, response times for both these stages were significantly increased compared with response times during wake. Further analysis of response time in relation to corresponding EEG revealed that, when applying a sleep onset threshold of a failure to respond within a 5 sec limit to 50% of the stimuli during that stage, then for all subjects, 'sleep' occurred during stage 1 sleep and before the onset of stage 2 sleep (Ogilvie et al, 1989). This would seem to indicate a relationship between EEG defined sleep states and levels of conscious awareness, for which stage 2 sleep represents a final curtailment in response capacity. A process for which, it is claimed, the use of discrete epoch to epoch scores over 30 sec epochs of EEG, as recommended by Rechtschaffen & Kales (1968) is relatively insensitive to (Ogilvie et al, 1988).

In this case, the description of sleep onset as a gradual descent from wake, through a period of drowsiness and reduced awareness, before sleep, is believed to offer a more precise representation of the changes entailed. This is an assumption undermined by the possibility of idiosyncratic thresholds of behavioural response cessation, an issue raised by Ogilvie et al, 1988) but not further developed. Without
accounting for this potential source of variation, the complexity of this process could be lost in an oversimplified account of the relationship between physiological indicators of sleep and psychological and behavioural events.

Bonnet & Moore (1982) highlighted this factor by examining the relationship between changes in the EEG and the perception of sleep. Throughout a series of sleep opportunities, subjects were awakened at intervals following objective indications of sleep onset in the EEG, i.e. the appearance of sleep spindles. Subjects were then asked to say whether they had actually fallen asleep. A reliable positive response (i.e. 50% of the time) was found to occur if a minimum of 2-4 min had elapsed from the first sleep spindle. In contrast, auditory threshold, measured as the amount of noise required to elicit waking, was found to increase dramatically within 1 min of the first sleep spindle. This is consistent with Ogilvie et al's (1988) findings of a failure to respond to auditory stimuli coinciding with EEG determined stage 1 and stage 2 sleep.

Bonnet & Moore (1982) concluded that although the appearance of a sleep spindle marks an objective change in arousal state, the subjective identification of sleep is dependent on the length of time asleep, and is therefore likely to occur at a later stage in the sleep onset process. My own experience of conducting MSLT trials has often involved 'waking' subjects following the identification of 3 consecutive epochs of stage 1 sleep, only to find that they had no recollection of being asleep, nor displayed any obvious physical signs of that being the case.

Ogilvie et al (1991) conducted a further study using spectral analysis across standard frequency bands in an attempt to further enhance the analysis of the EEG. A predictable reduction of power in the alpha frequency range during reduced, but not totally diminished response capacity (suggestive of drowsiness rather than wake or sleep), coupled with an increase in theta power during this period reaffirms the description of stage 1 sleep provided by Rechtschaffen & Kales (1968). In addition to this it was suggested that a more precise distinction between wake and sleep could be made on the basis of an observed sharp increase in spectral power across all frequency bands (delta, theta, alpha, sigma, beta) in association with the onset of sleep determined as a behavioural response failure (Ogilvie et al, 1991). This is consistent with a previously reported association between increased power across alpha, theta and delta frequency bands and a failure to detect a visual target during a vigilance task (presumed to indicate 'dozing off' - Torsvall & Åkerstedt, 1988).
Whilst to some degree endorsing the physiological criteria for the detection of sleep proposed by Rechtschaffen & Kales (1968), it is assumed that by integrating both behavioural and physiological aspects of sleep, this approach offers a more intuitive and ecologically thorough account of the transition from wake to sleep. However, by anchoring each unit of EEG analysed to a potentially arousing measure (an auditory tone), it could be argued that the behavioural demands of this approach produce an artificially distorted view of sleep onset. In this way much of the natural fluctuation between wake and sleep, i.e. the waxing and waning of sleep onset, may have been lost.

Evans (1992, 1993) measured the periodicity of EEG determined transitions back to arousal during the sleep onset process. EEG records previously scored as sleep were separated into sleep stages 1, 2, 3 & 4. Stage 1 was further subdivided into early stage I (no vertex sharp waves) and late stage I (presence of vertex sharp waves). For each stage, cycles of spontaneous arousal were measured as the elapsed time between consecutive bursts of alpha separated by a period of sleep activity. (An arousal cycle was measured in seconds, beginning with the onset of a discrete alpha period, including the ensuing sleep period, and terminating with the onset of the following alpha period.) Evans (1993) reported dominant periodicities in arousal of approx. 15-18sec during early stage 1 sleep compared with 31-34sec during later stage 1, and 51-60sec during stage 2 sleep.

These findings are interpreted with reference to the role of transient EEG features, e.g. vertex sharp waves, sleep spindles and K complexes, as part of an arousal inhibitory mechanism which acts to preserve the sleep onset process throughout both exogenous and endogenous disturbance. This approach assumes an appropriate description of sleep onset is made available through the division of the EEG into 'natural' epochs of alpha/theta bursts, and identified in terms of the changing rate of spontaneous arousal in conjunction with the appearance of EEG transients assumed to be active in the preservation of sleep.

These three approaches offer very different views of the transition from wake to sleep: as discrete states separated by generalised EEG changes (Rechtschaffen & Kales, 1968), as a reduction in behavioural response capacity coincident with changes in the EEG (Ogilvie et al, 1988; 1991), and as a gradual change in the rate of spontaneous arousal (Evans, 1992, 1993). Whilst there is some dispute over the point of separation between these two states, there is a shared assumption in that quantifiable differences can be found to separate the waking from the sleeping
However, for the measurement of sleepiness in the essentially alert, sleep satiated individual, the most popular method - the MSLT - incorporates the rationale for the effective detection of sleep onset provided by Rechtschaffen & Kales (1968). As Ogilvie et al (1988; 1991) and Evans (1992, 1993) have shown, behavioural and temporal factors in the progression of sleep are often at variance with the description of the sleep onset process provided by Rechtschaffen & Kales (1968). Ogilvie (1988, 1991) has questioned the relevance of considering stage 1 to be actual sleep as behavioural and subjective evidence is contradictory. Evans (1992, 1993) highlights the limitations of using an arbitrary temporal criteria for the definition of sleep given the rate of fluctuation in arousal during the early stages of sleep.

Throughout the following sections the effectiveness of monitoring change in the EEG in providing a measure of daytime sleepiness in the essentially alert individual is assessed. The issues to be explored include: i) problems specific to the scoring of sleep onset in subjects who are particularly prone to fluctuations in sleep/wake states, ii) motivational influences throughout latency trials, and iii) the distribution of sleep latencies across 'normal' subjects and the implications of a high 'sleepiness' MSLT score for daytime functioning.

4.3 When does sleep onset occur during the MSLT?

4.4 INTRODUCTION

In reporting on the clinical relevance of the MSLT, The American Sleep Disorders Association identified three broad bands of sleepiness (ASDA, 1992). These are described as; mild, moderate and severe sleepiness with mean daily sleep latencies of 10-15min, 5-10min, and <5min, respectively. Whilst moderate and severe sleepiness levels indicate an increased probability of pathological sleep disorder, it is suggested that levels of mild sleepiness are prevalent in a normal, healthy population.

With this in mind, and with the MSLT now firmly established in a clinical setting, it is perhaps not surprising to find a preference for the MSLT in the evaluation of change following experimental manipulation using normal subjects. It is argued that by offering direct access to an underlying physiological state, the MSLT is more accurate for this purpose than many alternative performance tests where sensitivity to sleep manipulation can be undermined by compensatory effort (Roth, Roehrs &
Zorick, 1982). Sleep latency during the MSLT is assumed to be predictable and a valid index of previous nocturnal sleep and circadian phase. The MSLT operates by 'unmasking' the tendency for sleep otherwise obscured by moment to moment response to distracting stimuli from both the immediate environment and internal resources (Carskadon & Dement, 1982). Nevertheless, whilst a relationship between night-time sleep duration and daytime sleepiness has been demonstrated following sleep reduction (Carskadon & Dement, 1981), the sensitivity of the MSLT in subjects experiencing mild levels of sleepiness, i.e. towards the limit of the sleepiness range measurable by the MSLT, remains questionable. This will be the major theme under investigation here. However, there are two methodological problems that must first be addressed.

A failure to sleep during the MSLT and the 20 min scoring convention Established guidelines for the detection of sleep onset during the MSLT (Carskadon et al, 1986) determine sleep as 'unequivocal' in the event of three consecutive epochs of stage 1 sleep or a single epoch of another sleep stage using standardised sleep scoring criteria. Sleep latency is measured as the time from lights out to the first such 30sec epoch of sleep. As the protocol for the MSLT necessitates an 'on-line' decision in order to minimise the accumulation of sleep between trials, Carskadon et al (1986) argue in favour of the 90sec threshold for sleep onset as a preventative measure against ambiguity and subsequent premature termination. However, in the event of a failure to satisfy this criterion a nominal sleep latency of 20min is assumed. At low levels of sleepiness commonly found in good, healthy sleepers following a night of regular sleep, actual sleep onset can be relatively infrequent during the MSLT (Carskadon & Dement, 1979; Carskadon & Dement, 1981; Clodore, Benoit, Foret & Bouard, 1990; frequency of sleep onset during all baseline trials = 50%, 40% & 53%, respectively). The resultant highly censored data sets present a number of interpretative difficulties. From a statistical point of view, a high proportion of 20min scores violates assumptions of both normality and interval data (the 20min score is essentially a descriptive category). Minimising the use of the 20min score would be advantageous regarding the appropriate application of parametric statistical techniques.

In addition to this, ambiguity over levels of sleepiness on termination of the test inevitably undermine the sensitivity of the MSLT in measuring treatment or experimental effect. This becomes most apparent in the comparison of change from baseline following experimental manipulation between two or more subject groups. With the allocation of the 20min score, the range of sleepiness beyond this point is
disregarded, even though sleep onset may be imminent for some subjects, yet an hour or so away for others. In the absence of a firm baseline score, interpretation of change is limited to differences in absolute levels of sleepiness. Attempts to compare the differential effects of experimental conditions in terms of degree of change are problematic. For example, if subject groups, divided along some characteristic such as age, performance, treatment, disorder etc., are tested at baseline to include a high incidence of trials without sleep, we could not say with complete confidence that one group changed more than the other solely on the basis of comparative differences in absolute MSLT scores.

This is illustrated in the case of Roehrs et al (1989) where improvement in daytime sleepiness following sleep extension is reported for both 'sleepy' subjects (baseline MSLT scores <6min) and 'alert' subjects (baseline MSLT scores >16min). Both groups showed a significant increase in mean MSLT scores following 6 nights of sleep extension. The difference between MSLT scores following a night of baseline sleep and the 6th night of sleep extension is reported to being greater for the 'sleepy' subjects than for the 'alert' subjects (and this is interpreted to suggest that the sleepy subjects benefitted more from the longer sleep). However, the 'alert' subject group included subjects with a baseline MSLT score of 20min. For these subjects, the measurement of change beyond this level is likely to be comparatively reduced due to the restriction of a terminal score and not necessarily because this group actually changed less than the other. (Roehrs et al, 1989).

We might extend this argument to include instances where there are no apparent changes in sleepiness between MSLT testing days. Seidel and Dement (1981) interpret a ranked correlation of $r = 0.65$ between repeated MSLTs to be in favour of the MSLT as a highly reliable technique for the measurement of daytime sleepiness, despite the inclusion of a large proportion of testing days during which no sleep was recorded. It might be argued that sleepiness beyond the time limitation of the MSLT is minimal and therefore of no interest, however as the 20min limitation to the experimental version of the MSLT is essentially an arbitrary point, we should be wary of assuming that differences in sleepiness beyond this limit are of any less significance than 'real' differences detected further down the scale.

**How much is 'sufficient' sleep for the detection of sleep onset?** In discussing the difficulties involved with setting sleep onset criteria, Carskadon and Dement (1979) emphasised the role of the MSLT in detecting a tendency for sleep rather than sustained sleep. A number of points concerning the use of a
minimum threshold of stage 1 sleep for the measurement of sleep onset during the MSLT were discussed. In particular, it was argued that this threshold should reduce the potential for prolonged sleep during trials, that it should be of sufficient duration to measure a tendency for sleep (for this purpose stage 1 sleep was claimed to be the first clear sign of the cessation of wakefulness), and that the test should remain viable for subject groups lacking the ability to sustain sleep (the example of sleep apneics is given) (Carskadon & Dement, 1979).

However, recent developments towards an understanding of sleep onset processes suggest that, as an operational definition of sleep onset, the current protocol for the experimental version of the MSLT (Carskadon et al, 1986) fails to take into account an increase in fluctuation between sleep/wake states during the MSLT for the regular, essentially sleep satiated subject (Evans, 1992, 1993), see p91. With a reduced pressure for sleep, the transition from wake to sleep is likely to be more episodic, whereas progression to stage 2 and sustained sleep is understood to occur more readily with increased sleep need (Rechtschaffen & Kales, 1968).

For healthy, young regular sleepers, therefore, the measurement of daytime sleepiness using the MSLT is likely to be characterised by a large proportion of trials without sleep, and, in the event of sleep, frequent shifting between sleep and wake states. However, in determining sleep onset during the MSLT, Carskadon et al (1986) rely on evidence of sustained sleep in support of sleep tendency. Furthermore it is assumed that an arbitrary threshold of 90s of stage 1 sleep is compatible with all levels of sleepiness.

In view of the range of protocols currently used during the experimental version of the MSLT, the purpose of this investigation was to examine (1) the effects of different temporal criteria for the determination of sleep onset, and (2) fluctuation between wake and sleep during the MSLT in essentially non-sleepy individuals. In particular, whether this fluctuation between waking and sleep states could be accommodated within the standardized guidelines for scoring sleep stages (which allows for up to 14secs of wake within a 30sec epoch; Roehrs & Roth, 1992). The 90s threshold may represent a sensible compromise between minimizing premature termination through ambiguity and subjective error during on-line scoring and maintaining the sensitivity of the MSLT for the discrimination of change at low levels of sleepiness. Alternatively, the discontinuity in the sleep onset process may produce an extended delay between the first indications of sleep and eventual satisfaction of the criterion for 'unequivocal' sleep.
4.5 METHOD

Data from 240 MSLT trials performed throughout Experiment 1 were further analysed. These consisted of 6 MSLT testing days (BASE7, EXT4,7,11& 14, and REC4) for each of the ten subjects who participated throughout the whole of the experimental subject. Each testing day was made up of four MSLT trials (1000h, 1200h, 1400h and 1600h) hence each subject contributed data from 24 MSLT trials.

For each MSLT trial a sleep latency score was determined as the time in minutes from 'lights out' to the first 30sec epoch of 3 consecutive epochs of stage I sleep, or 30sec of another sleep stage, according to MSLT guidelines (Carskadon et al, 1986). At this point the test was terminated and the subject woken up. In the absence of 3 consecutive epochs of stage 1 sleep (or 1 epoch of another sleep stage) the test was terminated after 20min and a score of 20min assumed. Throughout all trials evidence of the transition to stage 2 sleep was infrequent and ambiguous.

In addition, a further two sleep latencies were determined as follows : firstly, the time in 0.5min units from lights out until the first 30sec epoch of sleep. This latency score was determined using conventional scoring rules for the identification of stage 1 or stage 2 sleep (Rechtschaffen & Kales, 1968). This allowed for the inclusion of a maximum of 14sec of wake within each 30sec epoch. Secondly, a sleep latency score was determined as the time in 0.5min units from lights out until the appearance of the first 5sec episode of continuous sleep. Because of the shortened epoch length the Rechtschaffen & Kales (1968) scoring rules could not be used for the detection of this latency score. Nevertheless the criteria applied for the detection of a 5sec episode of sleep were dependent on recognisable changes in the EEG previously identified and adopted by Rechtschaffen & Kales (1968). As such a 5 sec epoch of sleep was indicated in the event of each of the following conditions being satisfied throughout a continuous period of 5sec or more of recording:

(i) a relative shift in EEG frequency to produce low voltage waveforms within the 3-7Hz range lasting for at least 5sec
(ii) the absence of alpha activity in the EEG
(iii) the absence of movement artifact in the EEG
(iv) the absence of blinking, movement, artifact etc. in the EOG channels other than that produced by slow rolling eye movements
Where the transition from a waking EEG to sleep as defined by the 5sec criteria was ambiguous, further clarification was dependent on the presence of clearly defined vertex sharp waves, K complexes or spindles. This allowed the comparison of three separate thresholds of sustained sleep in the determination of sleep onset using 5s, 30s, and 90s of relatively continuous sleep.

As an estimation of the extent to which the EEG fluctuated between sleep and wake activity during the MSLT, the EEG records were re-scored into 5s epochs following satisfaction of the 30s sleep onset criterion. Re-scoring was started at the beginning of this 30s epoch of sleep and continued until the end of the recording. As detailed previously, recording was terminated in the event of 3 consecutive epochs of stage 1 sleep, 1 epoch of another sleep stage, or 20min after the start of the trial. It should be noted that, in line with standardised scoring techniques, the guidelines for the MSLT allow the inclusion of upto 14s of wake in any single 30s epoch of stage 1 sleep. The purpose of a 5s epoch was to explore the transition from wake to sleep in a subject group having relatively low levels of sleepiness, as difficulties in sustaining sleep had previously been highlighted as a concern in setting sleep onset criterion for the MSLT (Carskadon & Dement, 1979).

Each epoch was scored as a period of wake or unambiguous sleep. The previously described criteria for detecting short episodes of continuous sleep were applied (see above). Again, it was necessary for these criteria to be satisfied throughout the whole of the 5 sec epoch. If within a given epoch the criteria of 5sec of continuous sleep was not satisfied then that epoch was scored as wake.

For both sleep (S) activity and wake (W) activity the duration of each discrete episode following sleep onset was determined by adding together all adjacent epochs of identical activity. This approach enabled us to determine i) how long a typical period of sleep would be sustained following satisfaction of this sleep onset criterion, and ii) approximately how often sleep continuity would be interrupted by a period of wakefulness throughout these MSLT trials.
4.6 RESULTS - Part III

4.6.1 Differences between the 3 scoring criteria for the detection of sleep latency

Sleep diaries and actimeters confirmed subjects compliance throughout the three sleep conditions. Overnight EEG recordings, (see section 3.7.1), also confirmed that for each night prior to an MSLT testing day subjects were able to sleep as required. Significant differences were found between conditions for sleep period time (SPT) and total sleep time (TST). Mean SPT across study nights ranged from 425min for REC4 to 532min for EXT7.

Using the conventional 90s scoring threshold, the level of daytime sleepiness found with these subjects at baseline (approx 16 min - see section 3.5.2) was consistent with a view of minor levels of sleepiness in the average 7.5-8h regular sleeper. Change in daytime sleepiness following extended sleep was moderate, giving a reduction in mean MSLT score of between approx. 1-2min. The implications of this reduction with regard to daytime functioning have been discussed (section 3.8).

The three scoring criteria for the determination of sleep latency were evaluated by addressing the following key issues: frequency of sleep onset during the MSLT, time of day differences in MSLT scores, and sensitivity to experimental manipulation.

4.6.2 Frequency of sleep onset during the MSLT

A relatively low incidence of trials involving an actual sleep onset has been identified (see above) as a potential problem in using the MSLT for healthy, regular 7.5-8h sleepers. As the influence of experimental manipulation for these subjects is commonly reported in terms of the change from baseline and following recovery, the frequency of sleep onset between sleep onset thresholds for all baseline and recovery trials were compared (Table 4.1).
Table 4.1. Frequency of Sleep Onset (%) using the standard MSLT criterion compared with 5sec and 30sec sleep onset criteria during BASE and REC MSLT trials (n=10)

For each time of day throughout this period there was a relative increase of between 13-117% in the proportion of trials for which an actual sleep onset was scored. This difference is illustrated for sleep onset criteria of 5sec, 30sec and 90sec (Figure 4.1).

Figure 4.1 Frequency of sleep onset throughout BASE and REC MSLT trials comparing standard MSLT scoring criterion with 5sec and 30 sec scoring criteria (no of trials =80).
4.6.3 Sensitivity to experimental manipulation

Daily mean MSLT scores for each scoring threshold are shown in Table 4.2.

<table>
<thead>
<tr>
<th>Threshold</th>
<th>BASE7</th>
<th>EXT4</th>
<th>EXT7</th>
<th>EXT11</th>
<th>EXT14</th>
<th>REC4</th>
</tr>
</thead>
<tbody>
<tr>
<td>90 sec</td>
<td>16.2</td>
<td>16.9</td>
<td>16.9</td>
<td>16.7</td>
<td>16.9</td>
<td>16.0</td>
</tr>
<tr>
<td></td>
<td>(1.2)</td>
<td>(0.9)</td>
<td>(1.3)</td>
<td>(1.3)</td>
<td>(1.3)</td>
<td>(1.2)</td>
</tr>
<tr>
<td>60 sec</td>
<td>14.8</td>
<td>15.5</td>
<td>16.4</td>
<td>16.1</td>
<td>15.9</td>
<td>13.6</td>
</tr>
<tr>
<td></td>
<td>(1.2)</td>
<td>(1.5)</td>
<td>(1.4)</td>
<td>(1.3)</td>
<td>(1.4)</td>
<td>(1.6)</td>
</tr>
<tr>
<td>5 sec</td>
<td>13.0</td>
<td>13.4</td>
<td>15.3</td>
<td>14.9</td>
<td>15.0</td>
<td>12.3</td>
</tr>
<tr>
<td></td>
<td>(1.5)</td>
<td>(1.8)</td>
<td>(1.6)</td>
<td>(1.5)</td>
<td>(1.4)</td>
<td>(1.5)</td>
</tr>
</tbody>
</table>

Table 4.2 Comparison of sleep onset criteria using thresholds of 5sec, 30sec, and 90 sec of stage1 sleep for daily mean sleep latency (s.e.) throughout the three sleep conditions (BASE, EXT and REC; n=10).

The application of different criteria in scoring the latency trials had essentially produced three different dependent variables in the form of 5s, 30s and 90s latency scores. However, absolute differences between these variables were not explored as this was predetermined by the temporal relationship of the scoring criteria, i.e. for each trial the following would necessarily apply:

- 90s latency score ≥ 30s and 5s latency score
- 30s latency score < 90s and ≥ 5s latency score
- 5s latency score ≤ 90s and 30s latency score.

Instead, differences between scoring thresholds were explored by assessing the relative sensitivity of individual thresholds to the experimental manipulation.

A one way ANOVA was calculated to compare MSLT scores throughout the individual EXT testing days. No significant differences were found. Likewise, scores for BASE and REC testing days were not found to differ significantly. The data were then reduced by calculating a single EXT score for each subject as the median score of all EXT days for each of the trials. A single BASE score was also calculated as the median score throughout individual trials during BASE and REC days. This was repeated for each of the scoring thresholds.
A multivariate ANOVA was performed to explore the following factors: sleep duration (2 levels), time of day (4 levels) and scoring threshold (3 levels). The following significant differences were found:

(i) a significant main effect of sleep duration ($f=6.73$, $df=1.9$, $p<0.03$)
(ii) a significant main effect of time of day ($f=6.47$, $df=3.27$, $p<0.002$)
(iii) a significant main effect of scoring threshold ($f=15.32$, $df=2.18$, $p<0.001$).

Post hoc Tukey tests were used to compare differences between means of BASE and EXT trials produced using the three thresholds. At the 1000h and 1200h trials, only the 5sec threshold was found to produce significantly higher scores ($p<0.05$) during EXT than BASE. At the 1600h trial MSLT scores for EXT were found to be significantly higher ($p<0.05$) than BASE scores for all four thresholds. No difference between BASE and EXT scores at 1400h trials were found using any of the three scoring thresholds. The relative sensitivity of the 5sec threshold in detecting change in MSLT scores throughout these trials following sleep extension is illustrated in Figures 4.2(a-c).

Variability in scores increased with less stringent sleep onset thresholds, particularly the 5s threshold. This was due to the ceiling effect of the MSLT which produced a greater proportion of 20 min scores using a 90s threshold, thus reducing overall variability for this threshold.

![Figure 4.2a: Sleep latency scores using a 5s THRESHOLD at four testing trials following BASE and EXT sleep (n=10)](image_url)
Fig 4.2b Sleep latency scores using a 30s THRESHOLD at four testing trials following BASE and EXT sleep (n=10)

Fig 4.2c Sleep latency scores using a 90s THRESHOLD at four testing trials following BASE and EXT sleep (n=10)
4.6.4 Fluctuation in Sleep/Wake states during the MSLT

Throughout all conditions sleep onset occurred (using the 30s scoring threshold) during 51.3% (121) of MSLTs. Following these sleep onsets, 1061 periods of sleep were interspersed with 969 wake periods. The mean number of discrete sleep episodes in those MSLT trials where sleep onset occurred was 8.8. The relative frequency of episodes of sustained sleep and wake activity lasting between 5s and >70s is illustrated in Fig 4.3.

Following 8h and 10h of sleep at night, the most typical duration of sustained sleep activity during the MSLT was 5s (greater than 45% of all sleep periods). Whilst less than 33% of all sleep periods were sustained for longer than 15s. The interval between the occurrence of the first 5sec of sleep and the 90 sec sleep onset threshold was calculated for each subject as the average interval across all trials involving at least 5sec of sleep. This was found to range from over 9 minutes for subject 1 during the 1400h trial, to no interval at all for subject 10 during all except the 1400h trial (illustrated in Fig 4.4).

![Figure 4.3. Frequency of Sleep and Wake episode durations following sleep onset (30 sec threshold) and until eventual termination of the trial for all MSLT trials (no of trials = 240; total Sleep episodes = 1061; total Wake episodes = 969).](attachment:image.png)
Fig 4.4  Mean interval per subject between the first 5sec of sleep and 3 consecutive 30sec epochs of sleep. All latency trials containing at least 5sec of sleep were included.

Following a 30sec threshold, fluctuation in sleep wake states was also found to be considerable. Changes in sleep/wake activity during periods of increased sleep propensity, i.e. during the afternoon trials, are illustrated for BASE & REC conditions (Fig 4.5a), and EXT conditions (Fig 4.5b) as arousal states between satisfaction of the 30sec and 90sec sleep onset thresholds. Each horizontal bar represents activity throughout a single trial following sleep onset and until eventual termination according to previously described criteria. Individual subjects are identified with alphabetic characters 'a' - 'j'. For illustrative purposes, trials are arranged in order of degree of fluctuation between sleep/wake states during the sleep onset period. The elapsed time between satisfying a 30s sleep onset threshold and 'unequivocal' sleep as described by Carskadon et al (1986) ranges from between 0s to approx. 300s.
Fluctuation between Wake & Sleep following Sleep Onset (30 sec threshold) for BASE & REC trials at 1400h & 1600h

[BLACK = Sleep : WHITE = Wake]

Fig 4.5a

Fluctuation between Wake and Sleep following Sleep Onset (30 sec threshold) for EXT trials at 1400h & 1600h

[BLACK = Sleep : WHITE = Wake]

Fig 4.5b
4.7 DISCUSSION - Part III

The main findings of this study can be summarised as follows:

- using a reduced sleep onset threshold of either 5 sec or 30 sec of observed sleep effectively increased the number of trials for which an actual sleep onset was scored. This was more pronounced throughout the morning rather than afternoon trials.

- Other than the 1600h trial, there was no change in MSLT scores following EXT sleep using established scoring criteria. However, by concentrating on shortened periods of sleep (5 sec), it was possible to enhance the sensitivity of the MSLT to a change in night-time sleep duration throughout morning trials.

- Overall effects on daily mean MSLT scores were minimal.

- Without a pressing need for sleep, the most typical bout of sleep experienced by these subjects throughout trials lasted for approx. 5-9 seconds.

- For these subjects, there was a delay of as much as 9 min from the first sign of sleep (5 sec) and 5 min for a single 30 sec epoch of sleep, before established sleep onset criteria (3 consecutive epochs) were satisfied.

Is the MSLT an appropriate test when subjects are not very sleepy?

Following extended sleep, a reduced sleep tendency is largely accounted for by the MSLT findings during the 1600h trial. This is consistent with change in Roehr et al's (1989) 'alert' subjects (i.e. baseline MSLTs >16 min) following extended sleep. The relatively modest change in daytime sleepiness found was evident using the conventional scoring criteria, and further enhanced for each of the two alternative scoring criteria. Additional improvements in daytime sleep latency measures during EXT were found throughout both morning trials, but only when using a 5 sec sleep onset criteria. Therefore, for these subjects, it was possible to enhance the sensitivity of the MSLT to a limited extent by adopting a less restrictive threshold of sustained sleep for the detection of sleep onset. This amounted to a relative increase of up to 117% in the proportion of trials for which an actual sleep onset was scored.
The main advantage of being able to distinguish between 20 min scores, or 'nonsleepers', produced using conventional criteria is to effectively minimise problems due to the non-normal distribution of scores.

The difficulties involved with handling a large number of 20 min MSLT scores resulting from the experimental study of normal subjects were discussed by Clodore, Foret & Benoit (1986). They applied a threshold of two consecutive 30s epochs of stage 1 sleep for the detection of sleep onset throughout 186 MSLT trials. The frequency of the onset of sleep during a trial was reported as approx. 50% of all trials and ranged from 25.8% at 2000h through to 77% at 1400h. A comparison was made between daily mean MSLT scores i) inclusive of all trials (using a score of 20min for non-sleep trials) and ii) only including trials for which an actual sleep onset was scored. This resulted in daily mean MSLT scores of 15.4min and 12.0min, respectively. Unfortunately, in recognising the potential difficulties arising from the convention of a 20min score, the simple exclusion of all non-sleep trials prevents comparison with available normative data for this type of subject group. In this case for example, which produces a mean daily MSLT score of 12.0min, it gives a misleading impression of the level of sleepiness under study.

Clodore et al (1986) and Clodore et al (1990) also pointed out that the time of day effect typically reported for MSLT trials (reduced sleep latency at 1400h compared with 1000h or 2000h) was only apparent when 20min scores were included in the analysis. This, it is suggested, indicates that the early afternoon dip in alertness results from an increased likelihood of sleep occurring for some individuals rather than an overall reduction in actual sleep latency between trials. In a similar vein, improvements in MSLT scores in healthy, young, regular sleepers following 4 nights of sleep extension (Carskadon & Dement, 1979) might more accurately be described as a reduction in the likelihood of sleep during an MSLT trial rather than any real difference in sleep latency; sleep onset occurring in approx. 50% of all baseline trials compared with less than 20% following 4 nights of baseline sleep.

A major problem with advocating a shortened sleep onset criteria is that this method is largely incongruous with standardized guidelines for the measurement of sleep, which have been widely adopted since the late 1960s (Rechtschaffen & Kales, 1968). Whilst in this study, a 5sec epoch of sleep has been shown to be more sensitive in detecting change in sleepiness levels in relatively alert subjects by effectively increasing the frequency of recorded sleep onset, it might be argued that such a considerably shortened epoch duration is inappropriate for the detection of
sleep onset. In particular, the reliability of detecting 5sec epochs of sleep has not been determined. However, the feasibility of detecting short-lasting changes in the EEG has already been established with respect to momentary arousals from sleep. For this purpose, it has been shown to be possible to detect a change of arousal state in the EEG lasting as little as 3sec. As for the significance of 5sec of sleep in the waking EEG, there is no reason to suggest that this has any less significance for the purpose of measuring a tendency for sleep than a 90sec period of sleep. Furthermore, there are many circumstances throughout the day in which the occurrence of 5sec of sleep would have dramatic consequences for the individual, e.g. driving a vehicle, operating equipment.

In relation to non sleep-deprived subjects, there is also evidence to suggest that, at marginal level of sleepiness, a 5sec epoch of sleep more accurately reflects the dynamics of the sleep onset process Evans (1993). Therefore, to assume a smooth progression from wake to sustained sleep would be misleading.

Carskadon & Dement (1979) outlined four factors relevant to the selection of sleep onset criteria for the MSLT. These include i) interference with sleep propensity for subsequent trials unless sleep during trials is minimised, ii) the MSLT measures only a tendency to fall asleep, for which a stage 1 criterion is sufficient, iii) the end of wakefulness is signalled by the first epoch of stage 1 sleep, and iv) consideration for certain groups unable to sustain sleep for long periods. With the relatively recent extension of the MSLT to cover normal subjects experiencing low levels of sleepiness the outcome of this investigation is considered with particular reference to these factors.

The first factor is concerned with restricting the potential for prolonged sleep during trials in order to minimise interference with subsequent trials, and is primarily offered as a counter-argument to the suggestion that stage 2 is the more appropriate stage for the detection of sleep onset. It has been shown that the transition from wake to sleep is unlikely to be a smooth and continuous process (Evans, 1993). Furthermore, following the first indications of sleep, interruptions leading to spontaneous re-awakenings were both frequent and prolonged for our subjects. During this study the amount of actual sleep gained throughout a number of trials prior to the satisfaction of a 90s threshold was considerable, (Figures 4.5a, b).

The second factor also relates to the question of whether stage 2 sleep would be the better marker of sleep onset? We are reminded that sufficient sleep for the
measurement of sleep tendency alone, rather than sustained sleep, is required during the MSLT (Carskadon & Dement, 1979). However, for the experimental version of the MSLT, the replacement of the 60s threshold offered by Carskadon and Dement (1979) with a 90s threshold of stage I sleep by Carskadon et al, (1986) without detailed qualification highlights the arbitrariness of a reliance on sustained sleep in support of sleep tendency.

The third factor, that an epoch of stage I sleep clearly signals the end of wakefulness, has been undermined by recent developments in our knowledge of the sleep onset process. In behavioural terms there is little justification for an arbitrary threshold of 90s of sustained stage I sleep as a defining feature of sleep onset. Ogilvie et al (1988) reported increased reaction time to auditory stimuli throughout stage I and 2 sleep compared to wakefulness. This would seem to indicate a relationship between EEG defined sleep states and levels of conscious awareness, for which stage 2 represents a final curtailment in response capacity. However, idiosyncratic thresholds of behavioural response suggest considerable variability between individuals in their ability to perform cognitive functioning during EEG defined 'sleep'. Individual variability in the perception of sleep is also reported to be high. As a means of detecting differences between sleep and wake states therefore, visual analysis of the EEG can provide an operational definition of the sleep onset process which at times will be more or less in agreement with observable behavioural data and subjective experience.

The fourth factor discussed by Carskadon & Dement (1979) in setting a minimum threshold for the detection of sleep onset during the MSLT, covers the inability to sustain sleep. For this purpose the example of sleep apnoea is given as a condition for which frequent arousals might hinder the satisfaction of a minimum threshold of continuous sleep. However, the dynamics of the sleep onset process for normal, healthy regular sleepers suggest that during attempts to sleep throughout the day under conditions of low sleepiness there is a protracted period of stage I sleep characterised by frequent re-awakenings. Without evidence of qualitative differences between sleep states following either 5sec, 30sec or 90sec of stage I sleep the determination of a minimum threshold of sustained sleep in support of sleep tendency has essentially to be an arbitrary judgement. For these individuals, actual sleep onset was relatively infrequent during the MSLT. Change following experimental manipulation was relatively small and the implementation of 5sec or 30sec of sleep onset criteria had little overall effect on the experimental outcome. It could be argued therefore that at the two extremes of
the MSLT scale, reduced sensitivity to change represents not only a 'floor' effect, in the case of excessive sleepiness e.g. narcolepsy (Carskadon & Dement, 1982) but also a 'ceiling' effect limiting the appropriate administration of this test to certain subjects groups. Any discrepancy resulting from the use of different temporal thresholds for the detection of sleep onset only becomes an issue at marginal levels of sleepiness, where the possibility for sleep is reduced.

However, despite the recommendations of Carskadon et al (1986) requiring a minimum of 3 consecutive epochs of stage 1 sleep as evidence of 'unequivocal' sleep, in practice there is considerable variation in the scoring of sleep latency during the experimental version of the MSLT. Johnson, Freeman, Spinweber & Gomez, (1991) advocate a conservative approach as latency to stage 2 sleep. Roehrs & Roth (1992) similarly advise the researcher to wait for 'clearer signs of sleep', i.e. sleep spindles and K complexes, should the detection of three consecutive epochs of stage 1 sleep remain ambiguous. On the other hand, Levine, Roehrs, Stepanski, Zorick & Roth (1987) and Levine et al (1988) measured sleep latency as the first of only two consecutive epochs of stage 1 sleep.

It should also be noted that a number of researchers vary the application of sleep latency criteria between their own studies. For example, whilst a threshold of three 30s epochs of stage 1 sleep was enforced for MSLTs following various degrees of sleep reduction (Lumley, Roehrs, Asker et al, 1987; Rosenthal, Roehrs, Rosen et al, 1993), MSLTs following regular 8h sleep (Zwyghuizen-Doorenbos, Roehrs, Schafer et al, 1988) and both 8h and 10h sleep (Roehrs et al, 1989) were scored using a less cautious approach of only two consecutive epochs of stage 1 sleep as evidence of unambiguous sleep.

This study has shown that, in the event of applying different scoring criteria, the comparison of normative data across studies and the significance attached to actual levels of sleepiness are undermined. In particular, deviations from standardised protocol without detailed qualification may compromise the value of diagnostic and descriptive 'bands' of sleepiness such as the three broad categories recommended by ASDA, 1992.

Perhaps more importantly, because of the high levels of 20 min scores routinely measured for normal subjects following between 8-10 h sleep each night, the current experimental version of the MSLT may be an inappropriate tool for the measurement of daytime sleepiness at this level. The adoption of less restrictive
sleep onset criteria in this study suggested some benefit to be gained from increasing the frequency of scored sleep onsets during trials, but also highlighted the probability of a considerable delay between the first sign of sleep and sustained sleep. As such, waiting for 3 consecutive epochs of sleep in these types of subjects, particularly during the morning, is largely unproductive. On the other hand, it has been shown in this study that measurable amounts of sleep do occur throughout these trials despite an eventual sleepiness score of 20min. Whether or not this has any functional significance throughout wakefulness has yet to be established.

In conclusion, by maintaining a conservative approach to the scoring of sleep onset, many instances of sleep are likely to be overlooked during testing for daytime sleepiness in healthy, sleep satiated young adults. Because of this, a preference for this approach has been shown to be intuively unsound as it does not take into account the dynamics of the sleep onset process peculiar to this level of sleepiness. In effect, because so many of these subjects are off the scale of the MSLT following a regular or extended pattern of sleep, the MSLT score contributes little towards an understanding of the nature of sleepiness under study.
4.8 Experiment 2: Can MSLT scores be reduced by increasing 'effort' to sleep?

4.9 INTRODUCTION

As an objective measure of daytime sleepiness, the MSLT is assumed to be free from the potentially distorting effects of subject motivation, unlike many performance or subjective measures which can be influenced to a great extent by effort and attitude (Carskadon et al, 1986; Reohrs and Roth, 1992). It has been argued that the role of subject motivation during the MSLT is both minimal and, as with all major external distractions, controllable. Only factors likely to prevent sleep are seen as potential sources of error during the MSLT (Roehrs & Roth, 1992). This reflects a commonly held belief that the conditions of the MSLT and the subjective states of the individual will not contribute to a state of sleepiness beyond pre-existing levels (Carskadon & Dement, 1982).

However, there have been a number of attempts to influence the onset of sleep in humans and in animals: Wilcox (1975) separated the sleeping process into three basic components - sleep onset, sleep accumulation and waking from sleep. Wilcox (1975) attempted to show that each of these components was amenable to behavioural conditioning within a response-reward context. Wilcox (1975) was able to show that the extent of operant control over the awakening response was considerable in laboratory rats in that clear indications of a conditioned response to frequent awakenings was possible. This was achieved by rewarding rats when waking occurred within 10 sec of sleep onset, and not at other times. Consequently the sleep patterns of these rats displayed extremely shortened and frequent bursts of sleep, separated by regular, short bursts of wake.

Wilcox (1975) also explored the possibility of sleep onset as a conditioned response in these rats by offering a reward for successful attempts to initiate sleep. He found that although the rats engaged in more sleep preparation type behaviours during the conditioning period, i.e. quiet immobility with reduced muscular tone, there was no increased likelihood of actual sleep onset. Wilcox (1975) nevertheless interpreted these findings in terms of a shortcoming in the response-reward design. It was argued that as it was only possible to administer the reward (food) during wake, the waking response was more amenable to response conditioning because of the temporal contiguity between response and reward. On the other hand, sleep onset
necessitated a delayed reward which may have accounted for a failure to elicit sleep onset as a conditioned response in these animals. On the other hand, Wilcox (1975) did interpret signs of increased sleep preparatory behaviour as a positive indication of the volitional nature of sleep onset.

Caruso, Zozula, Davis, Goldfinger, Glovinsky, Anderson et al (1988) had more success with their attempt to produce sleep onset as a classically conditioned response in humans. For 8 to 10 weeks, 5 human subjects underwent weekly testing sessions of MSLTs. Throughout this period nocturnal sleep at night was held constant. Following adaptation and baseline weeks, a hypnotic drug was used to elicit an unconditioned response in terms of shortened sleep latency. As the weeks progressed a combination of drug and placebo was used to reinforce the conditioning. During the final phase, subjects were assessed in terms of a conditioned response when the drug (unconditioned stimuli) was no longer used. It was found that in 4 out of 5 of the subjects sleep latency during this 10th assessment week was statistically shortened compared with the baseline period. A reduction in mean sleep latency at baseline of 25% by post-conditioning assessment was interpreted as clear signs of the considerable effect of classical conditioning on the process of sleep onset.

Using a variant of the MSLT, Alexander, Horne & Blagrove (1991) found that, under increased and comparable pressure for sleep, subjects offered a financial incentive to stay awake, were able to do this longer than subjects without financial incentive. If resisting sleep is at least partially under volitional control then the question of whether subjects can be successful in influencing the onset of sleep through increased effort is also of importance. This was a possibility advocated by Caruso et al (1988) although in their study, the reduction in MSLT scores may have been due to long-term practice effects which, although important, are not so central to the question of subjectivity in the sleep onset process, or the validity of the MSLT as most experimental designs are relatively short and therefore avoid the opportunity for long-term adaptation. This study questioned the role of subjective effort in facilitating the onset of sleep in a more immediate context. By offering subjects a financial incentive to go to sleep during a MSLT, it was expected that any volitional component of the sleep onset process would be maximised.
4.10 METHOD

Subjects, 14 females (age 18-28y), had responded to an advertising poster displayed around the University. They were all slept regularly, between 7-8h/night, did not sleep during the day, and had no sleep complaints. All subjects were undergraduate students and, as the experiment took place during college term time, it was possible to ensure a considerable degree of regularity in daytime activity throughout their participation.

Actigraphs were worn each night for 10 nights to ensure a regular sleep schedule. In conjunction with this, subjects also completed sleep diaries daily, giving details of all episodes of sleep. Again this was to ensure that each subjects slept between 7-8h per night, with little variation in timing or duration of sleep between nights. Subjects underwent two testing days of the trial MSLTs performed at 2 hour intervals commencing 1100h. For all subjects, the first MSLT testing day took place following three nights of actigraphically monitored sleep. The first and second testing days were separated by 7 nights of regular night-time sleep at home thus ensuring that the day of testing was held constant for each week.

For week 1, subjects were allocated to a testing day on an ad hoc basis. This day provided BASELINE MSLTs for which each subject was subjected to an identical protocol as follows: a single channel of EEG (C3-A2) and 2 channels of EOG were recorded according to the guidelines for the administration of the MSLT provided by Carskadon et al (1986). Subjects were asked to lie on a bed in a quiet, darkened room for a period of up to 20 min. They were instructed to "lie down, and with your eyes closed try to go to sleep". EEGs were printed on-line with a Grass Polygraph machine. Paper speed was set at 1cm/sec with gain equivalent to 25pV/cm. Trials were terminated following the observation of 3 consecutive 30 sec epochs of stage 1 sleep, or a single 30 sec epoch of another sleep stage. Other than this, trials were terminated following 20 minutes after lights out. Between trials subjects engaged in light study, avoiding heavy meals, caffeinated drinks and vigorous exercise.

At the first trial of the second testing day (week 2) subjects were allocated equally on the toss of a coin to two groups: CONTROL (C) and INCENTIVE (I) groups. For groups C MSLTs were repeated using a protocol identical to the previous week. Group I were additionally instructed before each trial that "If you can sleep
faster than you did this time last week then you will be given an extra £1.00". Thus it was possible for group I to increase their financial gain by 50%.

At each MSLT trial subjects completed two subjective measures of sleepiness: a 10cm visual analogue scale (VAS) comprising a single horizontal line 10cm in length, see Appendix 5. The ends of each line were marked with vertical delimiters and the expressions i) very sleepy ii) not at all sleepy. Above this line was the instruction "Please indicate (by placing a mark across the line) the point which most closely represents how sleepy you are feeling at this moment". In addition to this subjects also completed the Karolinska Sleepiness Scale (KSS - Åkerstedt & Gillman, 1990). This is a nine point scale with sleepiness-related descriptors placed at alternate points. Subjects are requested to mark their position along the scale in relation to how sleep they feel at that exact moment. Both scales were completed by all subjects immediately before and immediately after each MSLT trial.

4.11 RESULTS

Data for one subject taken from group C were excluded from further analysis due to a technical fault in recording EEG during the trials. The following analyses includes data from the remaining 13 subjects.

4.11.1 MSLT scores

For each MSLT trial a sleep latency score was determined as the time in half minutes to the first of three consecutive 30 sec epochs of stage 1 sleep or 30 sec of another sleep stage, according to the guidelines for scoring the MSLT (Carskadon et al, 1986). The accumulation of sleep as the day progressed and the potentially distorting effect of this on subsequent trials was avoided by terminating the test at this point. Failure to satisfy these requirements was recorded as a score of 20 min and the test terminated. A daily MSLT score for each subjects was calculated as the mean sleep latency across trials for each testing day. Mean daily MSLT scores were calculated for each group (see Table 4.3).
Table 4.3 Mean MSLT scores (SD) across trials for each subject group.

A multivariate analysis of variance with two within factors and one between subjects factor was calculated. These were: a within subject factor of WEEK (2 levels), a within subject factor of TRIAL (3 levels) and a between subject factor of INSTRUCTION (2 levels). To avoid the problem of missing data due to the removal of a single subjects MSLT scores in the C group, the mean of the latency scores of the remaining subjects for corresponding trials were substituted in the empty cells. Prior to the MANOVA, scores were log transformed to reduced the distortion of non-normal distribution of MSLT scores.

The results of the MANOVA were:

(i) no significant main effect of WEEK
(ii) no significant main effect of INSTRUCTION
(iii) a significant main effect of TRIAL (df=2,24 ; f=3.64; p=0.04)
(iv) no significant interaction between WEEK x INSTRUCTION x TRIAL
(v) no significant interaction between WEEK x TRIAL
(vi) a near-significant interaction between WEEK and INSTRUCTION (df=1,12 ; f=3.91; p=0.07)
(vii) a near significant interaction between TRIAL and INSTRUCTION (df =2,24 ; f=3.08; p=0.06)

This final interaction is illustrated in Figure 4.6 as mean change from the corresponding trial of the first week. Change in mean sleep latency for the incentive group is most marked during the third trial of the day (1500h).

To explore simple main effects of the significant differences between TRIALS paired t-tests were calculated to reveal:
(i) no significant difference between TRIAL 1 and TRIAL 2
(ii) a significant difference between TRIAL 1 and TRIAL 3 (df=13, t=2.20, p=0.037)
(ii) a significant difference between TRIAL 2 and TRIAL 3 (df=13, t=2.71, p=0.012)

Paired t-tests were also used as a post-hoc exploration of change within individual INSTRUCTION groups during individual TRIALS. As figure 4.6 suggests, TRIAL 3 was the only trial for which a significant change in sleep latency (from the first week) occurred: a significant difference between weeks for mean sleep latency during TRIAL 3 was found for group I (df=6, t=3.03, p=0.23) but not for group C.
4.11.2 Subjective sleepiness

Subjective sleepiness ratings (on KSS and VAS) taken before and after each MSLT trial were transformed to give a measure of change in subjective sleepiness throughout the trial. This was achieved by subtracting the pre-trial score from the post-trial score. On both scales, the higher the score the greater the sleepiness, i.e. if subtracting pre-trial from post-trial ratings produced a positive score this indicated an increase in subjective sleepiness throughout the duration of the latency trial. For each subject group, a mean difference score was calculated for individual trials. Visual analysis of the means suggested that subjective sleepiness was increased throughout the trial for both groups, but that during the second week the incentive group had experienced a greater degree of change in subjective sleepiness throughout each of the three latency trials. Change according to group for week 2 is illustrated in Fig 4.7a (KSS) and 4.7b (VAS).

![Graph](image)

**Fig 4.7a Change in subjective sleepiness during the course of the MSLT trial, using the KSS (n=14)**

Differences in group subjective responses to latency trials were further examined using the Mann Whitney U test to compare the average change across the day for individual subjects. A significant finding would indicate that, when ranked in order
of degree of change, more individuals showing large differences between pre- and post-trial levels of subjective sleepiness were confined to a single group than might be expected by chance, i.e. rather than being spread evenly across the two groups.

![Graph showing change in subjective sleepiness during the MSLT trial.](image)

**Fig 4.7b Change in subjective sleepiness during the course of the MSLT trial, using the VAS (n=14).**

For the VAS, no significant findings between groups Incentive and Control were found when average daily change for each subject were compared. However, this may have been due to the small number of subjects (n=7) in each group. There was a significant difference between groups for change in KSS scores (U=9.5; n=10; p<0.05; two-tailed). This indicated that for group I subjects there was a greater degree of change in subjective sleepiness than for group C subjects. Control subjects rated a mean change (increased sleepiness) following latency tests throughout the second week of 1.16 on the 9-point KSS compared with a mean change for the incentive group of 2.14.
4.12 DISCUSSION

For three trial MSLTs a relatively high consistency between MSLT scores on repeated testing days throughout a period of regular night-time sleep would be expected (Zwyguizen-Doorenbos et al, 1988 reported a reliability of $r=0.85$ for 3 trials). As interest was primarily focused on change in sleep latency in comparison with baseline scores, an attempt was made to prevent any inadvertent distortion of baseline scores through experimenter bias by postponing group allocation until the first trial of the second week.

However, an interaction between type of instruction and time of day such that an increased reduction in sleep latency was observed for the Incentive group during the third trial of the day suggests that, when coupled with a sufficiently high propensity for sleep, sleep onset during the MSLT may be subject to interference from motivational influences. In this case, it appears that the combination of a circadian related reduction in arousal and a financial incentive to increase motivation to sleep is sufficient to distort MSLT scores. This highlights the difficulties involved with using the MSLT to assess daytime sleepiness.

Whilst a relationship between night-time sleep duration and subsequent daytime sleepiness has been demonstrated following sleep reduction and sleep extension (Carskadon & Dement, 1977; 1979), the sensitivity of the MSLT in subjects experiencing mild levels of sleepiness and the influence of factors other than a physiological need for sleep remains questionable. A recent report by Kribbs, Packe & Dinges (1994) has also addressed the issue of contextual effects on sleep tendency. In their study, significantly shortened sleep latencies were found when latency tests followed immediately from long, monotonous vigilance tasks (Kribbs et al, 1994).

The findings of this study replicate previous findings of a reduction in latency scores on repeated MSLT testing (Caruso et al, 1988) and are also consistent with Alexander et al's (1991) findings of a distortion in latency times due to increased motivation. For this study we can think of this as increased 'effort' to sleep. Although Wilcox, (1975) found that rats were only able to imitate sleep onset by increasing their sleep preparatory behaviour and adopting the appropriate sleep position, the human subjects in this study may have benefitted from an ability to rationalise. That is, in the task situation imposed by this study they were able to 'let go' and relax, as opposed to attempting to 'force' sleep.
It is also of interest that incentive subjects perceived a greater change in sleepiness following the MSLT trial than control group subjects. This is despite the fact that subjects in group I spent less actual time in bed on the whole (due to the test being terminated at sleep onset). If this is the effect of giving an alternative instruction then it seems that subjective sleepiness is also subject to external factors i.e. by the suggestions made by the experimenter that they should be more likely to have experienced sleepiness. Alternatively this increase in sleepiness for the incentive group subjects might reflect actual success in their attempts to reduce their arousal levels and actually fall asleep.

In conclusion, it should be noted that for the subjects involved in this study, a mean level of sleepiness at baseline of around 13-15min was found for both groups. This represents a fairly typical level of sleepiness for young, regular sleeping normal subjects and is indicative of only mild sleepiness throughout the day. Nevertheless, with the added incentive of extra money, subjects in the experimental group were able to significantly reduce the interval between lights out and sleep onset during latency trials from baseline levels. This raises the issue of different forms of sleepiness for different reasons. By effectively increasing a propensity for sleep, this study suggests that, under certain conditions, the mechanisms for achieving sleep are influenced by volition. Consequently, the position that all sleep is purposeful in terms of a physiological requirement is no longer tenable in the light of these findings.
4.13 An unsuitable subject for the MSLT:
Rapid sleep onset during the MSLT with no obvious sleep need

4.14 INTRODUCTION

Johnson (1992) asked whether physiological sleepiness as measured by the MSLT had the same significance for different people, or even for the same person on separate occasions. This will be the topic under discussion. For Roehrs & Roth (1992) the MSLT provides the opportunity to place all individuals along a continuum of normality in terms of their physiological sleepiness. They offered normative values in line with the American Sleep Disorders association (1992) which suggested that normal, non-sleep deprived subjects would be expected to have a daily MSLT score of greater than 10 min. In their view, MSLT scores of less than 10 min, and certainly less than 5 min, are indicative of excessive daytime sleepiness (EDS). Sleep apnea, narcolepsy, or chronic insufficient sleep are given as common causes of this (Roehrs & Roth, 1992). However, Johnson (1992) found no reason to support this view in his observations of normal, healthy, non-complaining young adults, who regularly slept around 7.5 h per night, but who nevertheless were found to have a daily mean MSLT score of around 5 min.

It is therefore important to be able to establish the exact consequences of a shortened sleep latency in terms of performance outside of the laboratory. Are there situations, such as driving, operating machinery etc, in which there is particular risk for these individuals? Roehrs & Roth (1992) and ASDA (1992) imply that this might be the case. However, in Chapter 1, the disassociation between MSLT scores and performance was discussed. It was pointed out that, given sufficient motivation, prolonged sleep reduction for about 2 hours per night could be sustained with no overall effect on performance or subjective sleepiness (Rutenfranz et al, 1972; Noles et al, 1976; Friedman et al, 1977; Horne & Wilkinson, 1985; Blagrove et al, 1995). Even so, in the short term, a reduction of as little as 2.5 h per night has been shown to reduce latency to sleep onset during the MSLT by approx. 6 min from 14 min at baseline to approx 8 min following the 7th night of reduced sleep (Carskadon & Dement, 1982). In this study, although the final MSLT score was presented as indicative of a detrimental effect of sleep reduction, no additional evidence of impaired performance was given (Carskadon & Dement, 1982).

Clearly, there is a systematic relationship between sleep at night and MSLT scores, and this has been shown for both sleep reduction (Carskadon & Dement, 1981) and sleep
extension (1979). In experiment 1, despite only moderate change, latency to sleep onset during the MSLT was delayed by approx 1 min in comparison with baseline testing days. The relationship between this change in the speed of falling asleep and performance in both the laboratory and a real-world setting has yet to be confirmed. Furthermore, the paradoxical finding of shortened MSLT scores despite high performance levels in young adults (Levine et al, 1988; Manni et al, 1992) poses the question of how to interpret findings of rapid sleep onset during latency trials for these individuals. Should we, as Roehrs & Roth (1992) might argue, suspect some underlying pathological or behavioural cause? Or do these individuals simply exhibit an efficient, yet normal, mechanism for initiating sleep?

At the other extreme, Seidel et al (1984) and Stepanski et al (1988) report that, despite impaired sleep at night, as determined by EEG monitoring, individuals suffering from insomnia and reporting excessive sleepiness throughout the day, were not found to have pathological levels of EDS during MSLT testing. We might speculate that for these individuals, the mechanisms for initiating sleep are broken down thereby preventing sleep despite extreme sleepiness. Consequently, normal access to the measurement of underlying physiological sleepiness (i.e. the MSLT) is denied. This suggests that the MSLT measures both a tendency for sleep (as determined by recent sleep behaviour) and an inherent ability to initiate sleep.

Levine et al (1988) examined two large samples of healthy adults with no sleep-wake complaints: young (18-29y; n=129), and older (30-80y; n=47). About half of the former and about one third of the latter had MSLT scores below 10 min, and significant proportions of each group could be classified as 'severe'. None of these subjects complained of excessive daytime sleepiness or had any significant sleep disturbance, but Levine et al (1988) concluded that their subjects were probably suffering from partial sleep loss. They also interpreted in support of this claim the finding of an enhanced efficiency of nocturnal sleep, in comparison with less sleepy individuals.

More recently, Manni et al (1991) obtained MSLT scores from 18 healthy young adults. Thirty percent had scores between 5 and 10 min, and there were no indications of sleepiness from other measures. Nevertheless, the authors concluded that these subjects must have been drowsy, and suffered from excessive daytime sleepiness, probably due to chronic sleep loss.
An alternative view is that many healthy normal sleepers can indeed sleep during their normal waking period if they so desire. The attribution of being a 'good sleeper' may well indicate an ability to fall asleep easily through volition rather than through real sleepiness.

This study focusses on the issue of rapid sleep onset during the MSLT with no obvious sleep need. Details of three subjects who were identified as having 'high sleepability no sleepiness' (HSNS) are given. These subjects were identified as such from a total of 16 (8m, 8f) healthy adult subjects.

4.15 METHOD

Subjects 1 and 2

The first 2 subjects to be examined took part in Experiment 1. As detailed throughout Chapter 3, this experiment examined the ability to extend night-time sleep with particular interest in whether this could be maintained on a long-term basis, and whether daytime alertness improved. Subjects were encouraged to take as much extra sleep as they could, for 14 nights. Night-time sleep was recorded by EEGs and actimetry. All subjects were able to extend their sleep by 1-2h throughout the study. Despite the extra sleep there was little overall improvement in MSLT scores, daytime vigilance performance as determined by one hour sessions at the Wilkinson Auditory Vigilance Task (WAVT - Wilkinson, 1968 - this is generally regarded to be the performance test most sensitive to sleepiness/declining alertness), or subjective daytime sleepiness, determined by the Karolinska Sleepiness Scale (KSS - Åkerstedt and Gillberg, 1990). Two subjects were identified as HSNS.

Subject 3

This was an investigation into the effects of acute sleep restriction on daytime sleepiness throughout the following day. Six males subjects (age range 18-26), all undergraduate students, were recruited for the study on the basis of regularly sleeping 7-8h/night with little variation from night to night. Potential subjects were screened to exclude those taking medication or experiencing sleep difficulties, a subjective sense of excessive daytime sleepiness, or regular napping. Regularity in daytime work, physical activity and periods of relaxation throughout their participation was facilitated by the enforcement of a regular term-time schedule. Throughout this time subjects
completed sleep diaries each morning and wore actimeters each night in order to
monitor and enforce the required night-time sleep conditions.

Each subject participated in three experimental conditions which took place over three
consecutive weeks. For each week, sleep at night was limited to 3 nights of habitual
sleep (BASE), for which a compulsory time in bed of 2330-0800h was enforced, 1
night of restricted sleep (RES), followed by a further 3 nights of BASE sleep. During
each restricted sleep night subjects were instructed to restrict their sleep to either 2h
(RESP2), 4h (RESH) or 8h (RESH). This was achieved by limiting the time in bed
during this night to each of these durations, plus an additional 30 min period for the
purpose of settling down and falling asleep. It was expected that this would maximise
the likelihood of each sleep duration condition being achieved without leading to an
excessive amount of additional sleep.

Order of presentation of RES conditions was counterbalanced across subjects.
Subjects maintained a constant rise time of 0800h each morning throughout the study.
During the RES nights bedtimes were adjusted in accord with sleep duration, i.e. for
RESH bedtime = 2330h; RESH bedtime = 0330h; RESSH bedtime = 0530h.

On the day following each night of sleep restriction subjects came into the sleep
laboratory at 1315h. Actigraphs were read in order to ensure that instructions
concerning the previous night's sleep duration had been met. At 1400h and 1600h
sleep latency trials were performed according to the guidelines prepared for the
administration of the MSLT (Carskadon et al, 1986). The Karolinska Sleepiness Scale
(KSS) was administered as a measure of subjective sleepiness immediately prior to each
test.

A single channel of EEG (C3-A2) and 2 channels of EOG were recorded according to
Rechtschaffen & Kales (1968) guidelines for the detection of sleep onset. Subjects
were told to "Lie still, and with your eyes closed try to go to sleep". Between trials
subjects were monitored by the experimenter whilst they participated in light study to
ensure that no further sleep was accumulated. Heavy meals, caffeinated drinks and
vigorous exercise were prohibited throughout the whole of the testing day period until
the termination of the second test.

On-line analysis and termination of the test following the conventional criteria for sleep
onset (3 consecutive epochs of stage 1 or a single epoch of a 'deeper' stage of sleep)
was achieved by producing a paper trace of the EEG and EOG channels using a Grass
Polygraph machine. Paper speed was set at 1 cm/sec with gain equivalent to 25 μV/cm. In addition to this, on-line display and recording of the EEG channel, digitised at a sampling rate of 128 Hz, was performed directly onto an Olivetti PCS 386 microcomputer using software specific to EEG acquisition and analysis.

One subject who claimed not to suffer from daytime sleepiness had very short latency scores (average 5 min) under all conditions. Sleep restriction made no difference to his MSLT scores. We examined the MSLT EEG data in depth. Spectral analyses (4 sec epochs - using the RHYTHM software package - Stella Systems, Ottawa) were performed on selected minutes during MSLTs prior to sleep onset.

4.16 RESULTS

All three HSNS subjects were 20-24 y men.

Subjects 1 and 2 - Two of the ten subjects (S1 and S2) had particularly low MSLT scores, an average of 5.6 and 7.3 min, regardless of how much sleep they obtained at night. Compared with the other 8 subjects, rapid sleep onset was observed for these subjects throughout the day following a baseline schedule of sleep (Figure 4.8). Despite the opportunity for up to 10 h sleep at night S1 continued to have a relatively shortened sleep latency on MSLT trials, whilst for S2 there was some indication of small change following longer sleep at night, in the direction of reduced sleepiness, but scores were still also relatively low for this subject (Table 4.4). Neither subjects was otherwise sleepy, had any sleep disorders, and night-time sleep EEGs were normal (Table 4.4).

<table>
<thead>
<tr>
<th>Subject 1</th>
<th>BASE7</th>
<th>EXT4</th>
<th>EXT11</th>
<th>EXT14</th>
<th>REC4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep Period Time (SPT) - min</td>
<td>450</td>
<td>475</td>
<td>564</td>
<td>524</td>
<td>397</td>
</tr>
<tr>
<td>Sleep Onset Latency (SOL) - min</td>
<td>14.5</td>
<td>24.5</td>
<td>37</td>
<td>29.5</td>
<td>6.5</td>
</tr>
<tr>
<td>Sleep efficiency - (i) (total sleep time as % of SPT)</td>
<td>93.1</td>
<td>91.5</td>
<td>90.2</td>
<td>90.6</td>
<td>90.9</td>
</tr>
<tr>
<td>Sleep efficiency - (ii) (total sleep time as % of total time in bed)</td>
<td>90.4</td>
<td>87.1</td>
<td>84.5</td>
<td>86.4</td>
<td>89.5</td>
</tr>
<tr>
<td>daily MSLT score (min)</td>
<td>5.6</td>
<td>5.9</td>
<td>4.9</td>
<td>6.5</td>
<td>5.8</td>
</tr>
</tbody>
</table>


Ch 4: measuring sleepiness

<table>
<thead>
<tr>
<th>Subject 2</th>
<th>BASE7 8h TIB</th>
<th>EXT4 10h TIB</th>
<th>EXT11 10h TIB</th>
<th>EXT14 10h TIB</th>
<th>REC4 8h TIB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep Period Time (SPT) - min</td>
<td>368</td>
<td>549</td>
<td>526</td>
<td>474</td>
<td>397</td>
</tr>
<tr>
<td>Sleep Onset Latency (SOL) - min</td>
<td>27.5</td>
<td>34</td>
<td>23.5</td>
<td>17</td>
<td>24.5</td>
</tr>
<tr>
<td>Sleep efficiency - (i) (total sleep time as % of SPT)</td>
<td>97.7</td>
<td>89.9</td>
<td>89.9</td>
<td>91.2</td>
<td>91.8</td>
</tr>
<tr>
<td>Sleep efficiency - (ii) (total sleep time as % of total time in bed)</td>
<td>90.9</td>
<td>83.8</td>
<td>85.7</td>
<td>87.9</td>
<td>86.6</td>
</tr>
<tr>
<td>daily MSLT score (min)</td>
<td>7.3</td>
<td>6.3</td>
<td>8.1</td>
<td>8.5</td>
<td>6.8</td>
</tr>
</tbody>
</table>

Table 4.4 - Sleep duration and disturbance recorded throughout 8h and 10h time in bed (TIB) conditions for two subjects: S1 and S2.

Fig 4.8 Sleep Latency during MSLT trials following 8h sleep at night (n=10).
Sleep latency is shortened for all trials for 2 HSNS subjects (black) in comparison with the remaining 8 more 'alert' subjects (white).
Two measures of sleep efficiency (as the percentage of both the sleep period and the time in bed period actually asleep) were shown to be impaired with increased nighttime sleep. MSLT scores are comparable with Roehr et al's (1989) 'sleepy' subjects and the subjects identified as suffering from sleep restriction by Levine et al (1988) and Manni et al (1991), but by the discerning vigilance tests their scores were similar to those of the other subjects. (Figure 4.9)

**Figure 4.9 Performance throughout 55 min Wilkinson Auditory Vigilance Task (n=10)**

Two subjects scored as 'sleepy' on the MSLT (black) perform at least as well as more 'alert' (mean MSLT = 17.6) subjects (white).

**Subject 3 -**

The mean MSLT scores for the non-HSNS subjects (n=5) were: baseline = 18.0 min, 4h sleep restriction = 12.4 min, 2h sleep restriction = 12.2 min. For the HSNS subject (S3) these scores were 7.0, 5.2, and 6 min respectively (see Figure 4.10). All subjects had their eyes closed throughout the MSLT testing. Power densities were calculated
for each 4 sec epoch of EEG in 0.25Hz bins. These values were then averaged across periods of one minute. For EEGs following 4h and 2h sleep, these values were then subtracted from corresponding baseline (8h) values. This is illustrated in Figure 4.11(a,b) as the power densities averaged for the first minute after lights out during the MSLT relative to power during the same period in baseline trials. Levels of absolute power in the MSLT EEGs showed in the non-HSNS subjects an increase in absolute levels of both alpha and theta with sleep restriction (Figure 4.11a), but for the HSNS subject there was no difference between trials (Figure 4.11b). The HSNS subject displayed a noticeable increase in alpha activity relative to other subjects during this period of the trial. This was apparent in all sleep RES conditions. High alpha power compared with the group mean levels is illustrated for the baseline trial (RES8 - 1400h), Figure 4.12.

![Bar chart showing change in daytime sleepiness following restricted and baseline sleep (n=6)](image)

*Fig 4.10 Change in daytime sleepiness following restricted and baseline sleep (n=6)*

*Sleep latencies for S3 did not vary with night-time sleep duration*
Figs 4.11a and 4.11b Change in average EEG power from baseline (8h) during 1min after lights out, for 1400h MSLT trial following restricted (2h, 4h) sleep.
It has been shown (Chapter 4, part 4.3) that the scoring of sleep onset during the MSLT can be problematic for relatively alert subjects. Periods of sleep are typically short (5 sec), and separated by substantial episodes of wakefulness. This results in an often drawn out process, whereby the eventual sleep latency score bears little relation to the initial signs of sleep. As the scoring of sleep onset using MSLT guidelines (Carskadon et al, 1986) provides an arbitrary cut-off point for the detection of "unequivocal' sleep, differences between the sleep onset process for the same person under conditions of increased sleep need are limited to latency scores. However it has been shown that for subjects 1 & 2 these latency scores are largely unaffected when sleep need is ostensibly reduced by extending the nocturnal sleep period. In addition, for subject 3, increasing a need for sleep by restricting sleep at night did not lead to corresponding reductions in daytime sleep latencies.
The EEG recordings for this subject were further analysed visually to compare the individual features of each occurrence of sleep. This was achieved by scoring the recorded EEG & EOG in 5 sec epochs throughout the trial. Details of the method used were given in Chapter 4, part 4.5 p94. Because the guidelines for the MSLT offer only 'all or nothing' criteria for establishing sleep onset, it is possible that sleep without an obvious sleep need for individuals displaying HSNS characteristics is more directly related to the question of scoring criteria rather than assumptions underlying the nature of physiological sleepiness. For example, it is possible that these (HSNS) subjects display certain idiosyncratic EEG forms which render them unsuitable for the MSLT. This might be a relatively slow dominant EEG frequency similar to the slow (2-7Hz) waveforms assumed to coincide with Stage I sleep for most people (Rechtschaffen & Kales, 1968). We can think of this as a kind of pseudo-sleep whereby features of the EEG sufficient to satisfy MSLT criteria are present yet substantially less convincing than under conditions of increased, or 'true' sleep need.

However, when profiles of the six trials including sleep were made for subject 3 in terms of the six identifiable features of the EEG/EOG during the sleep onset process (Wake, 2-7Hz low voltage activity, slow rolling eye movements, vertex sharp waves, K complexes, sleep spindles - Rechtschaffen & Kales, 1968) there were no discernible differences between trials. Under conditions of reduced sleep (RES2 and RES4) the development of each of these measures from the start of the latency trial showed considerable similarity with the baseline (RES8) conditions throughout both 1400h (a) and 1600h (b) trials.

This was true for episodes of wake, Fig 4.13a. Despite 8h sleep the previous night, wakefulness ended abruptly, with only infrequent bouts throughout the rest of the trial. This was similar to the pattern of change shown during RES2 and RES4 trials.

Similarities in the development of 2-7Hz activity (assumed to indicate Stage 1 sleep for most individuals - Rechtschaffen & Kales, 1968) were also found between baseline and restricted sleep trials, Figure 4.13b.
Fig 4.13a Change in wakefulness throughout latency trials under conditions of baseline and restricted sleep for HSNS subject.

Fig 4.13b Change in 2-7Hz low voltage activity throughout latency trials under conditions of baseline and restricted sleep for HSNS subject.
Despite 8 h of sleep during the previous night, for this subject the first signs of sleep occurred approx 3 minutes into the latency trial. In all cases, following the transition from wake to sleep, 2-7Hz activity was sustained, relatively uninterrupted by long periods of wakefulness, until termination of the trial.

Slow rolling eye movements (SEMs) have been identified by Rechtschaffen & Kales (1968) as a precursor to sleep onset. For this subject, the appearance of SEMs occurred early throughout all trials, Figure 4.13c. Despite 8 h sleep the previous night, for baseline trials SEMs began to appear before 2 min of the trial had elapsed.

Fig 4.13c The appearance of slow rolling eye movements throughout latency trials under conditions of baseline and restricted sleep for HSNS subject.

Finally, the appearance of transient EEG features, such as vertex sharp waves, (VSW) K complexes, and sleep spindles are assumed to provide important clues to the physiological state of arousal. Rechtschaffen & Kales (1968) use VSWs to distinguish between early and later Stage 1 sleep, and K complexes and sleep spindles to distinguish between Stage 1 and Stage 2 sleep. According to Carskadon et al (1986)
and Roehrs & Roth (1992) the appearance of each of these features provides unambiguous evidence of sleep.

For this reason, the obvious occurrence of at least one of these features throughout baseline trials, i.e. following 8h sleep the previous night, provides clear evidence of the unambiguous nature of sleep onset for this subject, see Figs 4.13d - f. That is, given the possibility that this individual is unlikely to be in need of sleep to the same extent during RES8, RES4 and RES2 trials the nature of the sleep onset process, in particular the occurrence of clear signs of 'deeper' sleep was remarkably similar for all conditions. Actimeter readings confirmed actual differences in sleep length prior to each MSLT testing day, see Appendix 6. To sum, breaking the EEG down in this way has provided clear evidence of the occurrence of genuine sleep without obvious sleep need for this subject.

Tab 4.13d  The incidence of vertex sharp waves throughout latency trials under conditions of baseline and restricted sleep for HSNS subject
Fig 4.13e The incidence of K complexes throughout latency trials under conditions of baseline and restricted sleep for HSNS subject.

Fig 4.13f The incidence of sleep spindles during latency trials throughout conditions of baseline and restricted sleep for HSNS subject.
4.17 DISCUSSION

Current thinking amongst most sleep researchers is that one can only fall asleep if sleepy. These three HSNS subjects run counter to this maxim. However, such subjects are by no means unusual, and the approximately 20% prevalence found for these people is the same as that found by other authors, for example, Levine et al (1988) and Manni et al (1991), who were assumed by these authors to have excessive sleepiness, and be suffering from chronic sleep deprivation.

Roehrs et al (1990) differentiated between 'sleepy' subjects who they defined as having a baseline MSLT score of less than 6 min, and 'alert' subjects with a baseline MSLT score of more than 16 min. Differences in sleepiness levels for these subjects, despite comparable times spent asleep at night, were explained as actual differences in a biological sleep requirement. So 'sleepy' subjects had a greater sleep deficit following 7-8h sleep at night than 'alert' subjects. In support of this Roehrs et al (1989) reported that 'sleepy' subjects experienced a greater benefit from sleeping longer at night than a group of 'alert' subjects, i.e. change in MSLT scores was greater for this group. However, this apparent difference between the two groups may have been misleading as for 'alert' subjects, baseline MSLT scores were within 4 min of the MSLT 'ceiling' (20 min termination) and this acted as a limiting factor in the magnitude of any measurable improvement following sleep extension. As some 'alert' subjects reached this ceiling following just one night of sleep extension, this may account for an apparently greater change in MSLT scores for 'sleepy' subjects than for 'alert' subjects.

In this study, the two HSNS subjects (S1 & S2) were no more likely to show reduced levels of sleepiness following extended sleep than the other less 'sleepy' subjects.

It seems likely that overnight EEG recordings and performance measures for these subjects indicate that the HSNS subjects identified are not unusually sleepy in comparison with the remaining subjects despite scoring higher on the MSLT. The ability to initiate rapid sleep onset during the MSLT trial is likely to be influenced by motivation (Alexander, Horne & Blagrove, 1988) and previous activity (Kribbs, Pack & Dinges, 1994). The ability to influence MSLT scores by increasing the incentive to fall asleep was demonstrated in Experiment 2. As such the influence of individual characteristics on the onset of sleep cannot be ruled out.

The MSLT EEG findings for S3 suggest that high alpha producers fall asleep more readily, because, for example, high alpha levels are a sign of occult sleepiness. Given that alpha tends to signify relaxation, it is perhaps more likely that these subjects are
able to relax and prepare themselves more rapidly to fall asleep if they so desire. Gastaut & Bert (1960) observed that subjects able to maintain high levels of alpha activity were more liable to fall asleep during monotonous, yet distracting conditions. Specifically, these were subjects who quickly habituated to alpha blocking in response to external distraction. This might suggest that these subjects were particularly adept at distancing themselves from immediate environmental events.

More recently, Åkerstedt and Gillberg (1990) found increased theta and alpha activities in the waking EEG as sleepiness increased. This correlated with subjective sleepiness and was significantly increased over regular short periods of quiet immobility with eyes open and closed throughout a prolonged period of acute sleep loss in comparison with initial levels. In this study, findings of increase theta and alpha activity with increased sleep loss during the early stages of an MSLT trial (1 min after lights out) are similar to those of Åkerstedt and Gillberg (1990) in that subjects were still awake and in a state of relaxed preparation for sleep at this point. That the HSNS subject (S3) did not show increased levels of theta or alpha activity with sleep restriction may be due to an apparent ease in achieving a relaxed level of arousal which transcends an actual need for sleep. Further observations of shortened sleep latency irrespective of the amount of sleep taken on the previous night suggest that this might be the case.

The possibility of quantifiable differences in sleep episodes which occur either in response to a physiological need or for some alternative motivation has been put forward by Evans, Cook, Cohen et al (1977). In this study, a large group of college students (n=430) were surveyed as to their daytime napping behaviour. Over 60% of this group were found to take regular naps throughout the day. In order to determine the functional significance of such widespread napping behaviour, Evans et al (1977) further separated those identified as nappers into two groups of either 'appetitive' or 'replacement' nappers. Replacement nappers were defined as those subjects who responded 'no' to the question 'Do you nap even when you do not feel tired?'. By implication, appetitive nappers (responding 'yes' or 'maybe' to the above question) might be assumed to sleep without prior sleep need.

Of the original 60% of all subjects surveyed who were found to take regular naps throughout the day, over 20% were subsequently identified as appetitive nappers. A comparison was made between groups of replacement nappers, appetitive nappers and non-nappers. Subjects were given the opportunity for a single sleep episode (up to 60 min) throughout the afternoon under similar conditions to MSLT trials. It was
particularly interesting that, although all self-defined nappers were able to fall asleep as might be expected, 75% of a control group of non-nappers (subjects who reported never or rarely sleeping during the day) did in fact fall asleep throughout the trial.

Sleep latencies were similar for the two groups of nappers. On average, nappers (both replacement and appetitive) were found to have reduced sleep latencies compared with non-nappers. However, sleep stage analysis of the EEGs revealed differences in nap characteristics for appetitive nappers. Specifically, this was shown as frequent stage changes between drowsiness, stage 1 and stage 2 compared with replacement nappers and non-nappers who showed less fluctuation in sleep stage following sleep onset. Appetive nappers also spent significantly more time throughout the nap in stage 1 sleep than both replacement and non-nappers.

Because of the 'rules' for applying the MSLT the subjects in this study did not sleep for more than 90 sec, and so it is not clear whether, for example, the HSNS subjects would have shown a similar trend towards prolonged drowsiness and light sleep. Or, also in relation to EEG characteristics, whether HSNS subjects demonstrated more delta activity, or what may be considered to be a greater build up of Process S (e.g. Dijk et al, 1987). Dijk et al (1987) found delta activity increased throughout short naps placed at regular intervals throughout the day. This was taken to indicate a gradual increase in physiological sleepiness as the interval between the most recent sustained sleep episode was increased.

Evans et al (1977) concluded from their study that there are at least two different kinds of naps which have emerged to satisfy different functions, and that some individuals "use the nap primarily for psychologically restorative functions seemingly unrelated to sleep need". The idea that sleep onset can be facilitated by both voluntary and involuntary components presents a fundamental challenge to the assumptions of the MSLT. The characteristics of the HSNS subjects reported throughout this study suggest that we should at least be cautious in attributing the condition of extreme sleepiness to individuals who are quick to fall asleep. Whether or not this ability to rapidly initiate sleep represents the active suppression of arousal or, more likely, the ability to 'let go' of consciousness has important implications for the study of sleep onset mechanisms and their breakdown.

HSNS has practical ramifications, not only for the MSLT, but for example, it is the converse of insomnia and may offer insight into the treatment of this disorder. Whereas these HSNS subjects can relax and 'switch off' very efficiently and simply go to sleep
without seeming to need to, for insomniacs, being able to sleep is often problematic despite an obvious sleep need (Stepanski et al, 1988).

To summarise, there is evidence from this study and others (Levine et al, 1988, Mannie et al, 1992) that about 20% of normal and healthy, regular sleeping young adults are pathologically sleepy by MSLT criteria. However, by other equally discerning psychological tests of vigilance and sleepiness, sleepiness levels for these subjects are not extreme. They show no other symptoms of excessive sleepiness, and are otherwise healthy, good sleepers, and do not complain of daytime sleepiness. They are not unusual, as the scientific literature suggests, but unrecognised as such by many sleep researchers who maintain that these subjects must be sleepy. Nor is the concept of HSNS particularly new - Evans et al (1977) reported that approx 20% of all nappers were not satisfying a physiological requirement for sleep. It is only more recently, with the adoption of latency to sleep onset for the quantification of sleepiness on a large-scale that rapid sleep onset has been identified as a problem. An alternative explanation is that rapid sleep onset for these people is normal, rather than a result of chronic sleep deprivation as it has been suggested. To date, it has not been demonstrated that they are at an increased level of risk from performance failure in dangerous situations due to their increased sleep propensities throughout MSLT trials.
5.

Sleep satiation in the 'average' sleeper
5.1 Conclusions

The main findings of these studies are summarised in relation to a number of key issues and then by addressing the specific question "Should we be taking more sleep?".

The studies presented here have demonstrated:

- No obvious improvement in terms of physiological, subjective or performance criteria when sleep is extended by approx 2 hours at night.

- Extending sleep at night is possible in the home environment, most of this extra sleep is made up of additional stage 1 & 2 sleep, and REM sleep.

- Conventional guidelines for the measurement of physiological sleepiness using the MSLT are not sensitive to the level of sleepiness commonly found in young, healthy regular 7.5h sleepers: alternative criteria for the scoring of MSLT trials indicate that sleep, in the form of short 5s bursts (or microsleeps) occurs frequently and relatively early in the trial. However, the satisfaction of a 90sec criteria for unambiguous sleep is problematic.

- Motivational factors influence the onset of sleep for this type of subject.

- Within this group approx 20% show signs of being able to fall asleep quickly throughout the day without an obvious need for sleep.

5.2 How much is enough sleep for the 'average' sleeper?

Available survey data suggests that most people endorse a common sense belief that sufficient sleep is somewhere in the region of 8h each night. Exceptions to this (long and short sleepers) have been studied at length although no clear distinctions between average and extreme sleepers have been found. Subjectively, the actual duration of sleep at night does not seem to be a defining feature of self-reported 'good' or 'poor' sleepers. Johnson and Spinweber (1983) found little variation in sleep length between a large sample of self-defined good and poor sleepers and
concluded that prevailing situational and psychological factors were most important in determining perceived sleep quality.

However, there has been considerable support for recent objective evidence to suggest that the average individual has a sleep requirement in excess of 8h (Carskadon & Dement, 1979; 1986, Roehrs et al, 1989, 1994). Prior to this the question of how much sleep is required by normal, healthy individuals appeared to be largely resolved when studies of chronic sleep reduction had been able to show that, on the whole, there were no obvious detrimental effects of a gradual, long-term reduction of habitual sleep by approx 2 hours (Rutenfranz et al, 1972; Noles et al, 1976; Friedman et al, 1977; Horne & Wilkinson, 1988). One important factor in establishing renewed concern for individual sleep habits lies with a widespread belief in a unidimensional approach to the measurement of sleepiness. This has been embodied in the development of the MSLT, perhaps the most widely used of all sleepiness measures in clinical situations, and its transference to the study of normal, healthy individuals in an experimental setting. The MSLT offers the opportunity to locate all persons along a continuum of normality in terms of their level of sleepiness, and to provide descriptive and categorical knowledge of sleepiness levels within the general population.

Because the MSLT offers an intuitive approach to the question of sleepiness, i.e. the speed with which an individual falls asleep is indicative of the urgency, or otherwise, for sleep, the assumptions underlying this approach are rarely questioned. However, I have shown that, at least for healthy, normal subjects, the belief that all sleep is purposeful cannot be upheld in all circumstances. In particular, by studying alert, subjects with relatively low MSLT scores I have been able to highlight a number of situations in which sleep can occur other than in response to a physiological need: (i) as the ability to extend sleep at night without obvious recuperative value, (ii) as the high incidence of short bursts (or microsleeps) throughout non-arousing situations such as the MSLT, (iii) in response to a motivational incentive, and (iv) as the relatively common finding of high sleepiness scores in individuals who otherwise have no apparent sleep need. A prevalence of approx 20% of HSNS individuals in the general population can be inferred from their incidence throughout these studies, and in recent reported literature (Levine et al, 1988; Manni et al, 1992).

Consequently, by showing that not all sleep occurs in response to a physiological requirement, these findings present a substantial obstacle to the assessment of a need for sleep dependent on such a unidimensional view of sleepiness. Furthermore,
in measuring sleepiness it is clearly important to be able to take into account the reasons for sleep in a given situation. Such was the case for the subjects in this study who were able to tolerate the imposition of an extended sleep period at night, despite their own belief in the adequacy of their previous sleep habits. The reasons for this apparent flexibility in sleep duration are explored in more depth by asking "Should we be taking more sleep?", below.

Finally, I have shown that falling asleep can also satisfy a motivational rather than physiological drive, and that this is more highly developed for some individuals. Johnson (1992) asked whether we can be confident that the levels of sleepiness measured using the MSLT have the same significance for different individuals, or even for the same individual on different occasions. The prevalence of HSNS subjects suggests that this is unlikely. Inconsistencies between performance and subjective measures of sleepiness and MSLT scores, such as those found throughout these studies, imply that fundamental differences exist between the types of sleepiness accessed using these tests. The key question is therefore which of these tests offers a more complete representation of likely capacity, or even risk, in a real-life situation.

For that purpose, I suggest that the high levels of performance throughout prolonged vigilance testing, which were maintained during both 8 h and 10 h of sleep at night, offer a more ecologically valid index of the capacity to perform outside of the laboratory. I also suggest that the likelihood of sleep should be considered separately from the ability to perform, and is highly dependent on individual contexts. The reason for this is that, as with studies of sleep reduction, I believe that even at this relatively low level of sleepiness it is a person's willingness to remain alert, rather than a capacity to do so, which determines levels of performance throughout sleepiness related performance testing. Furthermore, I have shown that sleep, or at least substantially reduced arousal, is easily achieved in otherwise apparently alert individuals who might normally be assumed to be sleep satiated. This has been illustrated as the ability to initiate sustained (90 s) sleep after only approx 15-16 min, or even shorter bursts after only 5-6 min, throughout MSLT's trials, whilst in similar conditions being able to perform vigilance tasks to a high level for an uninterrupted period of approx 1 hour.

Horne (1991) proposed at least two types of sleepiness resulting from a need for core or optional sleep. We can think of optional sleepiness as more readily overcome by motivational influences to maintain performance, whereas core
sleepiness results in the more profound and overwhelming urge for sleep. However, the findings of reduced sleep latencies with increased motivation and HSNS subjects (Chapter 4) suggest that, without sufficient alternatives or incentive to remain awake, optional sleepiness will also lead to the onset of sleep. Consequently, if sleep onset is at times facilitated by a will to sleep rather than a need, then it would be misleading to take this as an indication of a physiological requirement for sleep.

5.3 Should we be taking more sleep?

Over the past decade there has been renewed interest and debate about how much sleep we need, whether the "average" 7.5 hours daily sleep is enough, and whether daytime functioning can be improved by taking more daily sleep (Carskadon & Dement, 1979; Carskadon et al, 1986; Roehrs, Timms, Zwyghuizen-Doorenbos et al, 1989; Roehrs et al, 1994). Whereas current opinion seems to moving towards "take more sleep and be less sleepy when awake" (Dement & Mitler, 1993), there is also convincing evidence to suggest that: (i) on a cost (loss of wakefulness) benefit (improved waking functioning) analysis, sleeping beyond ones "norm" produces, at best, only marginal benefits for the majority of people; (ii) the ability to extend one's sleep is not evidence of a need for this extra sleep; (iii) the social and environmental contexts of sleep allows for considerable variation in both its duration and structure; (iv) without there being appreciable improvements in subjective well-being throughout the day, many people are unlikely to be persuaded easily about the benefits of changing their daily sleep/wake patterns in order to take more sleep.

In 1975, Webb & Agnew produced their influential paper, "Are we chronically sleep deprived?" implying that this indeed was the case, and outlining many of the arguments that have since been echoed extensively. The thrust of these arguments falls under the main headings: the ability to take extra sleep both as part of an experimental requirement and spontaneously at home; all of sleep fulfils a physiological need; historical accounts of self-reported increased sleep duration (particularly at the turn of the century); and the inability to terminate sleep spontaneously or feel well rested immediately on waking. For each of these headings substantial counter-arguments exist which are not in keeping with a view of widespread chronic sleep deprivation.
Laboratory Studies of Extended Sleep and Subsequent Daytime Alertness

Interest in this issue began to take shape about 15 years ago, coinciding with advances in the measurement of daytime alertness, in particular the development of the Multiple Sleep Latency Test (MSLT). This device offered an objective and practical approach to measuring the relationship between sleep and daytime functioning, and in particular, provided an opportunity to monitor change following the manipulation of night-time sleep. Reports of benefits in extending the duration of sleep at night (e.g. 4 nights sleep extension [Carskadon & Dement, 1979]; 6 nights sleep extension [Reohrs et al, 1989]) helped both to emphasise the disadvantages of insufficient sleep, and to secure a role for the MSLT in determining change in daytime sleepiness.

However, in the following summary of the contribution made by experimental studies of change in alertness/sleepiness throughout the day following more sleep at night, it can be seen that the advantages of extending sleep may not be so apparent:

i) For the regular sleeping, healthy, young individual with a relatively low level of daytime sleepiness following a preferred sleep schedule, we can expect an improvement in daily MSLT scores of between about 1-4 min when the time spent in bed is increased from 8h to 10h (Carskadon & Dement, 1979; Roehr et al, 1989). A similar finding was made for experiment 1.

ii) Throughout experiment 1, the main effect on MSLT trials was been found to occur in the afternoon. During morning trials, the likelihood of sleep occurring was relatively low both before and following extended sleep. This is consistent with Roehrs et al (1989) who found a worsening of MSLT scores during 1000h MSLT trials following the initial nights of extended sleep. In those subjects these investigators (Reohrs et al, 1989) termed "alert" (as opposed to "sleepy" the small (<2 min) but significant improvements to MSLT scores only became apparent after 6 nights of extended sleep, and were confined to afternoon trials. It might be argued that these "alert" subjects do not represent an extreme or uncommon level of alertness as Roehrs et al suggest, because with baseline scores of around 16 min, these subjects typify the normal individual. Hence their subsequent, limited improvement to their MSLT scores following extended sleep, are also probably typical.
Alternative measures of waking alertness following extended sleep have provided unimpressive and inconsistent results - e.g. whilst Roehrs et al (1989) found sleep extension to improve reaction time significantly, these effects were small (<10% reduction in RT), and of little behavioural significance or of real practical relevance especially when considering the overall cost to the subject in lost productive wakefulness. It should also be noted that Roehrs et al (1989) observed that this performance testing may have been affected by practice. In experiment 1 a very small (12%) but statistically significant improvement to the Wilkinson Auditory Vigilance Test (WAVT) during a 14 day regimen of sleep extension was also found. On the other hand, Taub et al (1971) reported decrements in both WAVT and a complex motor task following two nights of extended sleep (TST = 9.1 h). In another study Taub (1981) found reduced vigilance performance and increased subjective sleepiness following one night of extended sleep.

Although it might be argued that performance improvements of this magnitude have little established ecological validity, for other investigators they are seen as an indication of a need for extra sleep (Carskadon & Dement, 1979; Carskadon et al, 1986; Roehrs et al, 1989). However, it should also be noted that extended sleep studies frequently include individuals without complaints of excessive daytime sleepiness, and with relatively low levels of objectively defined daytime sleepiness. Thus the perceived benefits of taking more sleep might well seem unconvincing to these individuals. For example, in this study, in order to gain about one minute of improvement in MSLT score, subjects had to invest an extra two hours of their time in bed, of which only approximately 60 min was actual sleep.

Apparent ambiguities between MSLT and performance findings in normal healthy subjects are further exemplified by Manni, Ratti, Barzaghi et al (1991), who concluded that MSLT scores obtained from such subjects (young adults) were indicative of a "fairly marked objective drowsiness" despite no additional signs of this using sleep-sensitive performance tasks.

Changes in subjective measures following the opportunity to extend sleep have been reported. Wehr, Moul, Barbato et al (1993), who compared the effects of 28 consecutive "long" nights [14h bed-rest in darkness] with 7 "short" nights [8h bed-rest in darkness]. Increased total sleep time (TST) and improvements in self-reported vigor and daytime fatigue were viewed by the authors to reflect a pre-existing sleep deficit. However, Totterdell, Reynolds, Parkinson et al (1994) found that enhanced mood (cheerfulness, alertness) throughout the day was related to an
earlier than normal sleep onset during the previous night, and not to TST. As the advancement of lights out tends to be adopted as a convenient method of extending sleep (Carskadon & Dement, 1979; Roehrs et al, 1989), the subjective consequences attributed to a night's extended sleep are likely to be confounded by factors other than sleep duration. This is further illustrated by Hawkins & Shaw (1992) in a repeated measures study of sleep duration and subjective sleep quality in college students. Differences in sleep duration on weekday nights and at weekends were found to coincide with an improvement in subjective sleep quality during extended sleep on weekends. However, a corresponding reduction in sleep quality was not found when subjects elected to reduce their sleep across the semester as their work-loads increased.

"Extra" Sleep - A Physiological Need or a Luxury?

The spontaneous lengthening of sleep at weekends has been well documented for both young (Webb & Agnew, 1975) and older adults (Lavie, 1981). Additionally, the ability to extend sleep in the laboratory is commonly observed and interpreted with reference to an underlying sleep debt (Carskadson & Dement, 1979; Roehrs et al, 1989; Roehrs et al, 1994).

However, several areas of research covered by both human and animal studies lend themselves to an alternative explanation for the capacity to take extra sleep, such that in situations where there is every opportunity for sleep, and few incentives to remain awake, sleep can occur for reasons other than in response to a physiological need. For example, Lucas (1979) found that laboratory cats spend more time asleep or in a drowsy state following a schedule involving long, uninterrupted periods of isolation and darkness (12h dark/12h light) than during an alternative schedule of shortened light/dark cycles (79 min dark/27 min light) for an equivalent period of time. Cats fed more than twice their normal quantity of food also showed an increased TST (Tagney, 1973) and baboons kept in field conditions have shorter TSTs (more stage 1 and less stages 3,4 & REM sleep) than in laboratory conditions (Bert, 1975). Ruckebusch (1976) reported a 30% increase in TST when ponies previously allowed to graze in an open field were continuously stabled. A similar pattern of increased TST, particularly REM sleep, was found when cows were transferred from an open field to continuous stabling. Ruckebusch (1976) considered this extra sleep to be in excess of the physiological requirements of the animal, and suggested instead that it represented a 'luxury form' of sleep, referred to more recently by Horne (1988) as "optional sleep".
From this view, wakefulness, like sleep, is dependent on cues from the immediate physical and social environment. For animals these cues include the need to maintain adequate food supply, shelter and physical security. As Ruckebusch (1976) pointed out, the stabled animal is no longer able or required to perform these functions and experiences less involvement with the immediate surroundings. For humans, cues to promote wakefulness are likely to be more complex. Other than a requirement for food and personal safety, most individuals conform to a large number of social activities which operate as powerful social zeitgebers in the organisation of individual work, free-time and sleep schedules. As for the spontaneous lengthening of sleep, this can be seen to coincide with a loosening of these zeitgebers, e.g. at weekends and holidays. We can perhaps include a tendency to oversleep throughout periods of withdrawal or alienation from social involvement, e.g. during a depressive episode (Shimuzu, Hiyama, Yagasaki et al, 1979; Hawkins, Taub & Van de Castle, 1985). From these perspectives, extended sleep observed in self-report studies or more contrived experimental situations in which there is every opportunity to sleep and few benefits to remaining awake, might be understood in terms of a volitional act rather than a physiological requirement.

As Webb & Agnew (1975) indicated, given the opportunity for unlimited food, many people will eat more than they need, and the same may apply to sleep. Aserinsky (1969,1973) demonstrated this latter capacity throughout long periods of enforced bed-rest in his subjects, such that 20h out of 30h bed-rest(1969) and 32h our of 54h bed-rest were spent asleep (1973). Gagnon et al (1985) reported significant increases in TST throughout 15h enforced bed-rest in darkness following a normal bedtime, and under both novel and habituated phase-delay conditions. Campbell (1984) noted that subjects slept for almost half (28h) of a 60h bed-rest period. Wehr et al (1993) reported increased TST throughout 28 "long" night (14h bed-rest in darkness) in comparison with "short" nights (8h baseline nights). Under all these circumstances individuals would be highly motivated to sleep in order to avoid lengthy periods of boredom, which might explain why Roehrs et al (1989) and throughout this study subjects were able to sleep significantly longer during 6 and 14 nights, respectively, despite relatively low baseline measures of daytime sleepiness determined by the MSLT (i.e. about 16 min).
Historical Accounts of Reductions in Self-Reported Sleep Duration

Webb & Agnew (1975) reported a reduction of about 1.5h in the average sleep duration of children aged 8-17y between the periods 1910-11 and 1963, and asked whether this is symptomatic of a relatively recent increased pressure to economise on the time allotted to sleep? It is conceivable that such a change has occurred given the rapid development of social, economic and technological advances between these periods. However, inconsistencies between the surveys suggest that this is a comparison based largely on errors of protocol, and sampling (over 2000 respondents in the 1910-11 survey opposed to 311 in the 1963 survey detailed by Webb [1969]).

Ironically, Terman & Hocking (1913), the authors of the original study, went to some length to explain why, at that time, they had found an actual increase in average sleep length (described by them as a "striking excess of sleep") compared with other contemporary studies originating in Germany and the U.K. Furthermore, a survey of >5000 Japanese children (Hayashi, 1927) during the spring of 1923 reported an average of 1h less sleep than for the children of the same age group in the 1913 study (Terman & Hocking, 1913). When Weissbluth, Poncher, Given et al (1981) compared the sleep of 8-17y U.S. children studied throughout 1980, with those in the 1910-1911 survey, no differences in average TSTs were found. These apparent inconsistencies between studies highlight the difficulties inherent in such approaches and undermine Webb & Agnew's implication that a genuine reduction in TST over time has occurred.

Trends in TST over the long-term between different subjects groups are likely to be confounded by changes: in subject compliance and experimental protocol (especially when relying on subjective estimates that are liable to be more inaccurate with poorer sleepers - Bixler, Kales, Leo et al, 1981); mood (Bliwise, Friedman, Yesavage, 1993); individual differences in economic status, home environment etc. (Terman & Hocking, 1913, MacGregor, & Balding, 1988); seasonal, climatic and geographical differences (Rosen & Rosenthal, 1991).

More recently, apparent changes in the subjective reports of sleep-habits amongst college students have been interpreted to suggest that this group is particularly at risk from peer and work-related pressures to take less sleep than necessary. For example, Hicks, Mistry, Lucero et al (1989) reported a 30 min reduction in the
average self-reported TST of students of the San Jose University between the years 1978 and 1988. However, such changes may be linked with transient changes in workload (Hawkins & Shaw, 1992), and season and time spent at college (Carskadon & Davis, 1988, Acebo, Davis, Herman et al, 1991). For example, Carskadon & Davis (1988) found a reduction in self-reported TSTs of about 20 min during the first term of college, compared with TSTs 6 months prior to entering college. Although this period coincides with a major life change towards independence, the effect on sleep is short lived, as follow-up surveys for a further two years at college suggest some seasonal change (longer TSTs during the fall), with a gradual overall increase in TST by the 3rd year of college, and similar to pre-college entry levels (Acebo et al, 1991). Thus, short-term factors leading to change in sleep-habits (which tend not to be controlled for) are likely to undermine claims of a progressive reduction in TST over recent decades.

There have been several recent large-scale surveys directed towards sleeping habits. A summary of the well-known surveys of this type is presented in Table 5.1. For adults, support for the notion of an 8h typical sleep at night is clear, in that the most popular estimated TST falls between 7-9 h per night. A number of interesting findings relating to the points raised earlier by Webb & Agnew (1975) emerge: Lavie (1981) found spontaneous extensions of sleep duration at weekends, with over half the respondent choosing to wake unaided. Billiard Alpervotich, Perot & Jammes (1987) found that daytime sleep episodes, as an indication of daytime sleepiness, were more likely to occur in habitual short and long sleepers rather than in the 7-9h sleeper. Concerning long-term adverse health effects, sleeping longer than the average 7-9h has been associated with increased mortality rates, as shown by a 9y follow-up of 4713 Alameda County residents (Wingard & Berkman, 1983), and a 6y follow-up of 1 million respondents to the American Cancer Society survey (Kripke, Simons, Garfinkel & Hammon, 1979).
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Failure to End Sleep Spontaneously or Feel Well-Rested on Waking

Webb & Agnew (1975) suggested that the failure to end sleep spontaneously might result in the premature termination of sleep prior to sleep satiation. They referred to a 1937 study in which only 30% of subjects awakened spontaneously in the morning as further indication of an incomplete fulfilment of a sleep requirement. Presumably the remainder of subjects relied on alarm clocks or the intervention of co-habitants for early morning awakening. However, as contemporary societies rely on the coordination of activity, punctuality is a highly valued attribute. It has just been argued that the satiation of sleep may precede the end of sleep, and it is not surprising that without complete reliability in the regularity of sleep offset mechanisms, most people prefer to not leave waking up to chance.

Bell (1980) tested the accuracy with which individuals spontaneously awaken at a chosen time. For well-rested, healthy, adult subjects, accuracy to within 15 min of a target time was 53% (a similar finding was reported by Hawkins, 1989). Bell (1980) also reported that accuracy increased with general overall sleep disturbance, i.e. increased awakening. Thus for the individual prone to frequent intermittent awakenings during the latter part of the night, the ability to wake up at a preselected time is more likely to be due to the increased number of involuntary awakenings coinciding with a target time, rather than to judgement.

There is no scientific evidence to suggest that feeling well rested immediately on waking is a necessary or even likely indication of sleep satiation. For example, Naitoh, Kelly & Babkoff (1993) describe a period of sleep inertia following the termination of sleep as part of the awakening process, which lasts an average of 5 min. Åkerstedt, Hume, Minors & Waterhouse (1994) investigated intra-individual differences in subjective measures of sleep quality following sleep episodes which were irregular in length, and which occurred at different phases of the circadian cycle. The timing of the end of sleep in relation to circadian phase was found to be an important predictor of subjective measures of sleep quality, feeling refreshed, and ease of awakening. In addition, whereas subjects were more likely to feel refreshed on waking following highly efficient sleep, the ease of awakening was found to be improved with reduced sleep efficiency, and was also negatively correlated with TST. Åkerstedt et al (1994) suggest that reduced sleep inertia following poorer, or more superficial sleep, might account for this. Hence, there is no reason to assume that the ability to wake and feel refreshed in an instant will improve with increased
prior sleep. Conversely, the process of going from wake to sleep at bed-time is not instantaneous. In fact, as the ability to switch rapidly from a waking to a sleep state might be considered symptomatic of an underlying sleep disorder (ASDA, 1992) the alternative might be seen to be, at least, unusual.

There have also been reports of a worsening of the morning transition between sleep and wake following extended sleep, exemplified by the "worn-out syndrome", which is a protracted period of thick-headedness and lethargy (Globus, 1969).

**Rapid Sleep Onset and High Sleep Efficiency - Signs of Sleep Debt?**

Contemporary sleep patterns tend to be artificially constrained and context dependent. When normal restrictions on sleep were removed Campbell (1984) found that subjects adopted a polyphasic sleep pattern, i.e. frequent, shortened episodes of sleep throughout a 60h bed-rest period. Wehr et al (1993) examined the proposition that modern lifestyles, especially the advent of artificial lighting, mask natural sleep patterns by separating the 24h period into an extended day and a shortened night. The opportunity for sleep is thus believed to be artificially compressed into a single 8h period. Throughout 28 consecutive "long" nights of 14h bed-rest in darkness there was an increased sleep period time characterised by more discrete, yet shortened sleep episodes, and increased amounts of stages 1, 2 and REM sleep, and an increase in wake after sleep onset (WASO). However, the question remains whether the imposition of a socially determined sleep/wake pattern resulting in consolidated sleep throughout the night is incongruous with a physiological sleep need?

Some researchers have assumed a negative association between sleep efficiency and sleep satiation, whereby a shortfall in sleep leads to a higher sleep efficiency (Levine et al, 1988; Roehrs et al, 1990). That is, the natural state of sleep is for a longer sleep onset latency (SOL), more WASO and less sleep efficiency (Levine et al, 1988; Roehrs et al, 1990; 1994; Wehr et al, 1993). Hence the converse, of short SOL and less WASO are signs of greater sleep need. An association between high sleep efficiency throughout the night and a high level of sleepiness on MSLT trials throughout the following day in young, healthy adults has been presented as further evidence of repeated failures to satisfy a sleep need (Levine et al, 1988). However, if we consider individual differences in SOL during both the nocturnal period and throughout MSLT trials to be, at least partially, dependent on the efficiency of sleep
onset mechanisms, it is not surprising to find that those subjects who experienced the most difficulty initiating sleep at night were those least likely to fall asleep quickly throughout MSLT trials during the following day.

An association between sleepiness levels throughout the day and nocturnal sleep efficiency in healthy, young regular sleepers was also found by Lavie & Zvuluni (1992). This study utilised a shortened polyphasic sleep wake cycle (7min sleep/13min wake) over two separate 48 h periods to examine the development of sleepiness across the day. For each 48 h testing period this involved a total of 144 latency trials of 7 min in length. Throughout the first period, subjects were instructed to attempt sleep at each latency trial. Following a break of at least 10 days a second condition in which subjects were instructed to resist sleep throughout latency trials was performed by all subjects.

Although the instruction to resist sleep throughout trials led to the accumulation of significantly less sleep than during the conventional (attempt) sleep latency trials, within subject correlations between the first and second testing periods were high. That is, those subjects least likely to fall asleep quickly in the attempt sleep condition were more likely to be able to resist sleep under alternative instructions. In addition to this, an association was also found between increased sleep propensity throughout latency trials and increased sleep efficiency throughout recordings of nocturnal sleep performed under baseline conditions.

Such consistency in within-subject sleepiness between testing days led Lavie & Zvuluni (1992) to emphasis the importance of individual traits in sleep propensity. In their view, for sleep propensity to be accountable solely in terms of circadian phase and prior sleep behaviour, the acute sleep deprivation imposed by the experimental schedule throughout this study would have (i) substantially reduced any between subject discrepancy in sleepiness levels due to prior sleep habits, and (ii) reduced within subject consistency in sleep propensity between the first and second 24 hour period of each testing day as the effects of sleep deprivation built up. However, as this was not the case, Lavie & Zvuluni (1992) argued in favour of relatively stable individual traits of sleepiness/alertness which are 'to a certain extent independent of previous sleep history'. In their view, such traits separate the 'alert' individual (unlikely to sleep throughout latency trials and reduced sleep efficiency at night) from the 'sleepy' individual (sleeps readily and efficiently) rather than actual differences in habitual sleep duration.
It is also important to consider the subjective consequences of sleep efficiency. In their study, Totterdell et al (1994) found a shorter SOL and less awakenings throughout the night (but not TST) to be associated with improved subjective sleep quality and enhanced mood throughout the following day. Åkerstedt et al (1994) also found subjective sleep quality to be improved with increased sleep efficiency. In effect, the enhancement of sleep efficiency represents for most people a valued aspect of sleep behaviour. Furthermore, as increased SOL and WASO are common features of extended sleep, it would be difficult to persuade most individuals of the benefits of sleeping longer.

Conclusions

An increasing concern that many people may not be getting enough sleep has led to a number of sleep researchers calling for increased efforts towards raising popular and scientific awareness of risks associated with this problem (Dement & Mittler, 1993). A recent report by the National Commission on Sleep Disorders Research referred to the self-imposed restriction of sleep below acceptable limits and called for a "radical change in the way society deals with sleep".

Various major accidents have been reportedly attributed to sleep-related human error (Mitler, Carskadon, Czeisler et al, 1988). Researchers have also advocated a causal relationship between inadequate night-time sleep and: increased sleep-related traffic accidents (Dement & Mitler, 1993); increased daytime sleepiness (Carskadon & Dement, 1979; Carskadon et al, 1986); impaired performance (Roehrs et al, 1989, Timms et al, 1988); and mood (Wehr et al, 1993) throughout the day. These findings imply that many individuals forego essential sleep to maximise time spent in waking activity, and at a cost to both their personal safety and their potential for self-fulfilment. But rather than sleep length per se being the underlying cause, the more direct involvement of total sleep loss and circadian influences on arousal/sleepiness are the most important features of many incidences in which individuals are overwhelmed by the urge to sleep. This is clearly evident in the temporal distribution of traffic accidents across the 24h period, in which the prime period of increased risk is early morning (Horne & Reyner, 1995; Langlois, Smolensky, Hsi et al, 1985; Mitler et al, 1988), when the victims have had no recent sleep. That is, they were vulnerable because of the inappropriate timing of sleep. It does not suggest that they should be taking more than their usual 7.5h daily sleep. Extended night-time sleep has yet to be shown to improve alertness during a subsequent night of no sleep. On the other hand, the afternoon dip in alertness might
be partially avoided after taking extended night-time sleep (Roehrs et al, 1989; Experiment 1 - this study), and the relevance of this to performance during the afternoon in real-life situations requires further investigation.

However, as the most promising positive aspect of extending sleep at night, it is nevertheless possible for improved alertness during the afternoon to be achieved equally successfully by other methods, and with less disruption to habitual daily patterns. For those individuals particularly prone to a period of reduced alertness during the afternoon, an effective solution would be to take a short nap at a suitable point during the day (Naitoh, 1992; Gillberg et al, 1994).

Webb & Agnew (1975) suggested that people have experienced a relatively recent change in attitude towards dedicating time to sleep at night. For Wehr et al (1993) it is the advent of artificial lighting which has proved most influential in these respects. Social impositions may have also had an impact as withdrawal from social influence leads to dramatic modifications to sleep patterns (Campbell, 1984; Wehr et al, 1993). Indeed, the duration and structure of sleep is context dependent, influenced by internal and external sources such as the prevailing social, environmental, physiological, and psychological factors.

Webb & Agnew (1975) asked whether, given the vicissitudes of modern lifestyles, sleep has fallen below the levels of individual necessity? In experiment 1, an experimental remedy in the form of extended sleep has shown consistently that the marginal benefits are out of proportion with the actual costs to the individuals in terms of reduced active wakefulness and deterioration in sleep efficiency (e.g. 2h more time in bed= 1h more sleep= worse sleep efficiency=small improvement in MSLT). Thus, the real-life significance of extended sleep needs to be put more clearly by proponents of the 'take more sleep' view.

A number of observations which run counter-intuitive to a view of widespread chronic sleep deprivation have also been highlighted. Studies of extended sleep have reported benefits to MSLT scores using subjects reporting to be free from daytime sleepiness, who do not nap in the day, and are regular, healthy sleepers (Carskadon & Dement, 1979; Carskadon et al, 1986). Furthermore, MSLTs in subjects tested at baseline show low levels of daytime sleepiness (e.g. Carskadon & Dement, 1979 - subjects did not sleep during 50% of baseline MSLT trials). Even without obvious signs of sleepiness during the day, subjects are still able to extend their sleep at night, and this situation can be maintained over the long-term (Experiment 1 - this
study, Wehr et al, 1993), suggesting that this extra sleep is gained for reasons other than a deficit of sleep.

Finally, to return to my original question "how much sleep do we need?", it is perhaps tempting to assume that systematic differences in an objective measure of sleepiness following the manipulation of sleep at night allows greater sensitivity for the assessment of a sleep need than that provided by earlier measures. This approach offers, in principle, the means to determine the point of sleep satiation for every individual, and to locate those individuals who are not sleeping enough. By satisfying a commitment towards objective measures, the production of normative values makes feasible the management of sleep within the wider population. However, the mechanisms involved in the initiation of sleep are still not fully understood, such that it is not possible to state the full effects of a change, either increase or reduction, in daytime sleep propensity on daytime functioning. As with the case of taking more sleep - the feasibility of extending sleep over the long-term is questionable as the incentives are limited and studies have failed to provide consistent evidence of improvements in subjective well-being or performance ability. Consequently, without knowing exactly what an increase in daytime sleep latency of around 1-4 min offers for the average, healthy young adult, there is no justification for prompting a change in current social norms of sleep patterns.
6.

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APPENDIX 1

Subject Selecting Criteria

The following procedures were employed to select subjects for the study. The following procedures were employed to select subjects for the study. The following procedures were employed to select subjects for the study. The following procedures were employed to select subjects for the study. The following procedures were employed to select subjects for the study.

Appendices

SLEEP QUESTIONNAIRE

Please try to give full and honest answers to the following questions, using the space provided and continuing onto the next page when necessary.

1. Are you experiencing any difficulties with your sleep at the moment?
APPENDIX I

Subject Selection Criteria

The following application form was completed by each prospective subject. Subject selection was dependent on a regular, 7.5 - 8h sleep duration each night, with little variation between nights. Applicants were rejected if they experienced a prolonged sleep latency (greater than 30 min), frequent awakenings throughout the night, difficulty returning to sleep if wakened, a regular final wake-time outside of the 07:30 - 08:30h period, or if they were receiving current or recent medical treatment. Those drinking excessive or regular amounts of alcohol (more than 10 units per week) or caffeinated drinks were also excluded as abstinence was required throughout the participation of all studies. In addition, it was also essential to establish that the subjects involved in these studies did not rely on supplementing night-time sleep throughout the day, therefore regular daytime nappers were not included.

SLEEP QUESTIONNAIRE

Please try to give full and honest answers to the following questions, using the space provided and continuing over the page when necessary.

1. Are you experiencing any difficulties with your sleep at the moment?
2. During the past 4 weeks at which of these times have you most often gone to bed?
   (a) before 10.00pm
   (b) between 10.00-11.00pm
   (c) between 11.00-midnight
   (d) between midnight-1.00am
   (e) after 1.00am

3. After going to bed and settling down for the night, how long would you normally expect to take to actually fall asleep?

4. On average, how many hours of sleep do you normally get each night?

5. Over the past week, roughly how many nights have you slept straight throughout without waking?

6. If you do wake, do you often have a problem getting back to sleep again?

7. During the past 4 weeks, which of these times are you most likely to wake up in the morning?
   (a) before 5.30am
   (b) between 5.30-6.30am
   (c) between 6.30-7.30am
   (d) between 7.30-8.30am
   (e) after 8.30am

8. Have you ever taken sleeping pills to help you sleep?
   If so, please state how recently this was.

9. Throughout a normal week, how many units of alcohol would you normally drink? (unit = 1 measure of spirit, glass of wine, pint of beer etc.)

10. How many cups of coffee or tea do you normally drink each day?

11. Would you mind going without alcohol completely throughout the study?

12. Do you sleep in a bed alone?
13. Do you share a bedroom with anybody?

14. How regular are you in your sleeping habits? i.e. are your sleeping patterns:
(a) the same every night
(b) the same most nights
(c) slightly unpredictable
(d) very irregular

15. In terms of how much sleep you might actually need, do you feel that the amount of sleep you are getting is:
(a) adequate
(b) inadequate and would feel better with more sleep
(c) excessive and would feel better with less sleep

16. Do you sleep during the day?
(a) occasionally
(b) regularly
(c) never

Finally, use the space below to include any information concerning your normal patterns of sleep which you feel might be relevant to this study ..................
APPENDIX 2

Sleep Diary

The following Sleep Diary was kept close to the subjects' bed and completed each night and morning throughout their participation in a study.

SLEEP DIARY

SUBJECT NAME ....................................................................
DATE ........................................................................................

Please remember to treat your actimeter with care. Keep it close to your bed with this form and a pen so that it will be easy to remember each day. Put the actimeter on your dominant wrist about 10-15 minutes before getting into bed and take it off each morning when you are ready to get up for the day.

AT NIGHT :

1. Just before you turn out the lights to sleep please fill in the time:

   1.

THE NEXT MORNING :

2. What time did you wake up this morning ?

   2.

3. How many times did you wake up throughout the night ?

   3.

4. Roughly how long did it take you to get to sleep last night ?

   4.
5. On a scale of 1 to 10 how well would you say you slept last night? (1 = slept extremely badly; 10 = slept extremely well)

Each morning subjects were also asked to complete the Profile of Mood States subjective scales for VIGOR and FATIGUE (see Appendix 4.)
APPENDIX 3

The Karolinska Sleepiness Scale

Listed below are a set of feelings which reflect various degrees of alertness and drowsiness. They are graded from 1 to 9. Read them carefully and indicate your present state by placing a tick in the appropriate box.

- [ ] 1 Extremely Alert
- [ ] 2
- [ ] 3 Alert
- [ ] 4
- [ ] 5 Neither Alert nor Sleepy
- [ ] 6
- [ ] 7 Sleepy, but not fighting Sleep
- [ ] 8
- [ ] 9 Extremely Sleepy, Fighting Sleep, effort to stay awake
APPENDIX 4

POMS mood scales for VIGOR and FATIGUE

The following words describe feelings that people have. For EACH word try to indicate how you are feeling AT THIS MOMENT.

Do you feel .................

<table>
<thead>
<tr>
<th>not at all</th>
<th>a little</th>
<th>moderately</th>
<th>quite a bit</th>
<th>extremely</th>
<th>[MOOD]</th>
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</thead>
<tbody>
<tr>
<td>1. Active</td>
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<td>V</td>
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<td>2. Alert</td>
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<td>3. Bushed</td>
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<td>F</td>
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<td>4. Sluggish</td>
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<td>5. Lively</td>
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<td>6. Energetic</td>
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<td>V</td>
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<tr>
<td>7. Fatigued</td>
<td></td>
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<td>F</td>
</tr>
<tr>
<td>8. Carefree</td>
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<td>V</td>
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<tr>
<td>9. Weary</td>
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<td>F</td>
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<tr>
<td>10. Worn out</td>
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<td></td>
<td>F</td>
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<tr>
<td>11. Listless</td>
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<td>F</td>
</tr>
<tr>
<td>12. Cheerful</td>
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<td>V</td>
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<tr>
<td>13. Full of pep</td>
<td></td>
<td></td>
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<td></td>
<td>V</td>
</tr>
<tr>
<td>14. Exhausted</td>
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<td></td>
<td>F</td>
</tr>
<tr>
<td>15. Vigorous</td>
<td></td>
<td></td>
<td></td>
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<td>V</td>
</tr>
</tbody>
</table>
APPENDIX 5

Visual Analogue Scale (VAS) of Subjective Sleepiness

Please indicate (by placing a mark across the line) the point which most closely represents how sleepy you are feeling AT THIS MOMENT.

VERY SLEEPY

|-----------------|

NOT AT ALL SLEEPY
APPENDIX 6

Actimeter readings throughout RES nights for HSNS subject 3.

The following actimeter readings were obtained for the nights requiring sleep restriction, and immediately preceding each MSLT testing day.

(i) RES2

Subject reported wearing actimeter from approx 1 am. Actimeter recordings confirmed that subject remained active throughout the night until an eventual bedtime of around 05:30h. Dramatically reduced activity at this point indicates that subject slept until around 08:00h. At this point there is a burst of activity, the subject was awakened by an alarm clock, dressed for the day, and took off the actimeter.
Subject reported wearing actimeter from approx midnight. Actimeter recordings confirmed that subject remained active until approx. 03:30h. From this point, dramatic reduction of activity indicated that, with the exception of an arousal lasting approx 5 min around 05:00h, subject slept through until 08:00h. At this point subject got out of bed and dressed for the day.
Subject reported wearing actimeter from around 23:45h. He went to bed around midnight. Very little activity occurred throughout the night, until final awakening shortly before 0800h. Actimeter recording therefore confirmed that subject remained inactive throughout the night, and very likely slept for approx 8 hours.