SLEEP DURATION AND MOOD

by

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Abstract

It is widely believed that sleep and mood are interrelated and that prolongation of sleep may have beneficial effects on subsequent mood and general well-being. In the present investigation, it is hypothesised that excess sleep is in fact, detrimental to mood and is associated with a 'Wornout Syndrome', characterised by feelings of fatigue and lethargy, that can persist for up to 5 hours.

The studies to be presented here compare the differential effects of Sleep Extension and Sleep Restriction on mood in healthy adults. The experimental design required subjects to undergo one night of Sleep Extension [+2h] and, following an interval of one-week, one night of Sleep Reduction [-2h]. The conditions were counterbalanced. Subjective assessments were conducted hourly on mood states and sleepiness using an adapted Profile of Mood States Questionnaire and the Stanford Sleepiness Scale. Actometers were worn throughout the experimental days and nights. In the first study of 10 subjects results indicated that four subjects were adversely affected by oversleep. Study 2 investigated the effects of sleep duration on mood in 20 healthy adults. Personality factors were assessed using Cattell's 16PF Questionnaire. Subjects maintaining regular sleep schedules reported negative effects of oversleep on subsequent mood. Results indicated that certain personality types were predisposed to the 'Wornout Syndrome' following Sleep Extension. In Study 3, thirty-four subjects were selected on the basis of personality type. It was hypothesised that Introverts, Morning types, Emotionally Tenderminded and Low Impulsives would report symptoms characteristic of the 'Wornout Syndrome' following one night of Sleep Extension. This was confirmed by reports of increased fatigue, diminished vigor, and increased confusion following Sleep Extension. Oversleeping produced greater detrimental effects on mood than a comparable reduction in sleep duration.

There are many similarities in symptomatology between the 'Wornout Syndrome' and Chronic Fatigue Syndrome (CFS), specifically, intense fatigue and impaired concentration. Interestingly, chronically fatigued patients often complain of sleep disturbance, and spend much of their time resting in bed. It was hypothesised that the 'Wornout Syndrome' may be a confounding factor in the symptomatology of CFS. As a clinical dimension, twelve subjects were investigated polysomnographically [six were CFS patients]. Findings indicated that CFS patients acquired sleep of longer duration than controls. In addition to excess nocturnal sleep, CFS patients were taking daytime naps. EEG data indicated that these individuals obtained twice the normal amount of slow wave sleep. CFS sufferers may be better advised to regulate their sleep habits and reduce their total sleep time to avoid the confounding effects of the 'Wornout Syndrome'.

Acknowledgements

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Mood and Sleep:
A Review

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Mood

1. Mood

1.1 Introduction to Mood.

Moods are basic expressions of the conscious human state and are believed to be influenced by any number of significant events, favourable or unfavourable. They reflect the general well-being of the individual. One factor that is popularly assumed to affect mood is sleep. It is thought that oversleeping is beneficial to mood and reduces daytime sleepiness. Feelings of lethargy and fatigue are frequently associated with sleep reduction and it is commonly believed that a morning 'lie-in' will counteract such feelings. However, following a survey of healthy young adults, Globus [1969] described a 'wornout syndrome', particularly when sleep exceeded ten hours and was not replacing a prior sleep deficit. This syndrome, characterised by terms such as 'wornout', 'lethargic' and 'sleepy' was reported to persist for up to five hours after awakening.

Whilst it is generally agreed that sleep is a biological necessity, the quantity and timing of sleep is largely dependent on socio-cultural factors. Adults in most cultures do however confine the major part of their sleep to the night-time and tend to maintain schedules of retiring and awakening that are determined primarily by external factors. People often judge the quality of their day to day living by the quality of their sleep. One frequently hears individuals explaining their current mood state by referring to how well they slept the previous night.

1.2 Mood: A problem of definition

Despite the undeniable importance of the study of mood, psychologists until quite recently, had not advanced far in its comprehensive formulation. Early mood theorists e.g. Nowlis [1956], Jacobsen [1957] and more recently Isen [1984] recognised moods as distinctive affective states deserving of special attention. There was a general consensus that the effects of mood are widespread and pervasive and that moods are capable of influencing a broad array of potential responses, whereas the effects of emotions are relatively specific. Compared to emotions, mood, it is generally agreed, represents a less intense affective state and is thought to be involved in the instigation of self-regulatory processes. For example, as the discrepancy between the desired or normal state and actual state increases, mood

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may be more and more likely to cross the awareness threshold, attract attention and as a result, generate self-regulatory behaviour. Furthermore, whilst the antecedents of emotions are often identifiable, the same is not always true in the case of moods. Nowlis [1956] regards moods as subtle background states, more resilient to change than emotions. He adopts a behaviouristic approach and suggests that:

"Mood may be defined as an intervening variable or predispositional factor that is a source of information , or discriminable stimuli to the organism, about the current functioning characteristics of the organism."

Nowlis views conscious mood as consisting of the perceptual and cognitive responses to this information. Mood as an intervening variable is regarded as having a direct (unconscious) effect on the probability of occurrence of certain responses in a given situation, as in expressive behaviour and action. Meddis [1969] suggests that mood acts as "an economic regulator of behaviour", and that current mood state is determined primarily by; (i) the physiological well-being of the individual, (ii) the degree of success recently experienced and (iii) the perception of the external environment. Mood is thus intimately linked to a variety of activities, both physiological and psychological.

1.3   Dimensions of Mood

Although the affective lives of individuals seem incredibly complex and rich [Wessman and Ricks, 1966] they can be successfully submitted to dimensional analysis, thereby enabling a search for individual differences. Mood is primarily a psychological manifestation and much of what we know about it comes from self-report measures. Though there is little controversy associated with the attempt to identify basic dimensions of self-reported mood, there is disagreement as to how many dimensions exist and the way in which they should be labelled. Some investigators e.g. Watson and Tellegen [1985] assert that a basic two-dimensional structure of mood is required. They suggest that it is possible to conceptualise affect as positive or negative, and claim this relationship is orthogonal. Diener et al. [1985] similarly propose that positive and negative affect are bipolar and stress that there is an inverse relationship between the two. Others argue that there are at least 5 to 11 monopolar and discrete factors [Nowlis, 1965; Thayer, 1987; McNair et al., 1971]. The mood items listed by Nowlis includes aggression, social affection, surgency, elation, sadness, vigor, fatigue, anxiety, concentration, egotism, scepticism and nonchalance. Nowlis suggests that mood is a multidimensional set of temporary reversible dispositions.

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Thayer's Mood Scale (Activation-Deactivation Adjective Check List: AD ACL) is a multidimensional measure of mood. He asserts that most mood states may be subsumed under two bipolar dimensions, associated with positive and negative affect. Thayer discusses mood in terms of energetic and tense arousal states and proposes that from low to moderate levels, energetic and tense arousal are positively correlated, but from moderate to high levels, the two dimensions are negatively correlated and appear to be bipolar. This, according to Thayer, provides evidence of mood dimensionality. Mc.Nair et al. [1971] advocate that moods are sensitive to and reflect the general well-being of the individual. A good mood is one in which the person can be satisfied with the achievement of current goals and desires to achieve new ones. A bad mood is one in which the person has failed to meet set targets. Mc.Nair et al. have devised the Profile of Mood States (POMS), comprising of 6 mood factors: (i) vigor-activity (ii) fatigue-inertia (iii) confusion-bewilderment (iv) tension-anxiety (v) anger-hostility and (vi) depression-dejection. Nowlis' Mood Adjective Check List (MACL) and Mc.Nair's POMS portray mood as multidimensional, without describing it as primarily positive or negative in character. It is argued that multifactorial outcomes retain a degree of articulation that more adequately reflects the phenomenology of mood.

1.4   Measurement of Mood

There is disagreement as to the validity of self-report measures of mood [Wilson, 1982]. Nevertheless, the majority of research studies still use some form of self-report measure and supportive research evidence justifying its use continues to be published [e.g.Watson, 1988]. Nowlis [1965] argues that validating self-reports of mood is unnecessary because the use of self-descriptive mood labels is a *direct* measure of mood when one conceives of it as a source of information to the organism about its current state. Blaney [1986] asserts that the problem with mood research is that single measurements are made of a process that is dynamic and which should be measured repeatedly over time.

Nowlis's pioneering work into the measurement of mood was so exhaustive that those who followed [e.g. Thayer, 1987; Mc.Nair et al., 1971] automatically adopted both his procedure and his rating scale. Nowlis used a four-point rating scale containing two categories of acceptance [definitely and slightly], one of rejection [definitely not] and one of confusion [can't decide]. Meddis [1972] criticised Nowlis's rating scale arguing that the scale used was asymmetrical and more sensitive to degrees of acceptance rather than to degrees of rejection. An

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explanation was offered by Meddis to account for the lack of bipolarity in Nowlis's factors. Meddis proposed that Nowlis's conclusion that mood dimensions are monopolar was merely an artefact of the rating scales used. Meddis [1972] adopted a symmetrical scale relying on a forced-choice procedure, thereby abolishing the neutral or 'cannot decide' alternative. Meddis disagrees with earlier researchers [e.g Nowlis] who, using factor analysis, claim that a large number of mood dimensions exist along which individuals vary independently. Instead, Meddis used Cattell's incremental R-technique and proposed the existence of two bipolar factors of mood, tentatively named vigor and hedonic tone. Meddis used cluster analysis techniques and criticised Nowlis for not applying Cattell's P-technique and R-technique to the MACL. Cattell's P-technique involves taking repeated measures on a single individual, and the R-technique involves the use of mood change scores rather than simple mood report scores. Meddis concluded that the asymmetrical scales used by Nowlis and others were suppressing negative correlations and prejudicing the factor analysis against the discovery of bipolar factors.

The Profile of Mood States is one of the best known self-rating mood scales. It contains six factors which are independent of each other [see above] and encompasses a series of adjectives that the subject rates on a five-point scale (from "not at all" to "extremely"). The scale is easily administered and completed within five minutes. In addition to a total mood disturbance score, it is possible to evaluate subjects' scores on particular dimensions, thereby enabling finer comparisons to be made across conditions.

1.5 Mood and Personality

In order to adequately conceptualise mood, it is important to understand the theoretical relationship between mood states and personality traits. Studies of the relationship usually hold that the broad personality types, traits or dispositions, act as parameters of the more limited mood states. Eysenck, for example related high neuroticism to negative mood [see Williams, 1981]. This relationship was confirmed by Thayer [1988] who found that tense arousal correlated significantly with the Eysenck Personality Inventory (EPI) assessed neuroticism. In general terms though, it is often assumed that individuals of a particular personality type are more prone to experiencing certain moods than individuals of a different personality type. Wessman and Ricks [1966] were among the first mood theorists to remark on this relationship. They proposed that it was essential to understand the affective state of an individual before proceeding to investigate his personality. Costa and

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McCrae [1980] found that negative affect was strongly related to neuroticism but not to extroversion, whereas positive affect was more highly related to extroversion, thus linking these two basic mood factors with two important and pervasive dimensions of personality. McCrae and Costa [1991] proposed that personality is not merely associated with emotion because personality characteristics lead individuals to obtain different experiences. Rather, there appears to be something about personality characteristics which is associated with the manner in which individuals respond to mood-eliciting events.

Cattell [1961] was originally concerned with determining dimensions of individual differences. His concern lay primarily in the true nature of moods and their relationship to minor changes in personality and stress or anxiety responses rather than the behaviour of an adjective checklist presumed to measure mood. He found that individuals differed in their responses to the various dimensions from day to day and developed the P-technique from his research into intra-individual differences. He postulated the existence of a number of independent mood states which are free to vary in intensity independently. Cattell holds moods to be "patterns of response common to most people in our culture, each operating in response to a variety of stimuli."

Cattell's P-technique is ideally suited to the study of mood because of its sensitivity to change within the individual. Mood may be considered a dimension of change in a given person rather than differences between individuals. Moods, or state changes were assessed using P and R-techniques and a list of thirteen mood state patterns was developed [Cattell and Scheier, 1961] thus providing support for the multidimensionality of mood states. Although Nowlis [1965] has attempted to compare his mood adjective checklist to Cattell's factors, Meddis [1972] has indicated that any such comparisons must be viewed with caution, due to the absence of any systematic exploration of introspectively reported mood.

Cattell [1972] has produced a powerful technique for studying moods from the personality aspect. He used the Sixteen Personality Factor Questionnaire (16PF) and a range of objective (RT, verbal fluency, memory) and physiological (pulse rate) tests. Cattell's 16PF is one of the most extensively used personality questionnaires. It identifies 16 primary and 4 secondary order factors that enables one to isolate particular individuals presenting specific personality characteristics, e.g. low impulsives, introverts and emotionally tender minded individuals are easily identified. These 16 dimensions are essentially independent so that each scale

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provides some new piece of information about the person being tested. The high level of differentiation obtained by using this personality test was a major factor that determined its incorporation into the present study.

1.6 Circadian Rhythms, Individual Differences and Mood.

Virtually every system of the body which has been investigated exhibits its own rhythmicity, usually circadian or ultradian. The classic unidimensional arousal theory of circadian rhythms [Kleitman, 1963] has been supplanted by the view that circadian rhythms are controlled by several independent internal 'clocks' or oscillators. Wever's [1984] temporal isolation studies demonstrated the independence of the body temperature rhythm from the sleep-wake cycle. Further evidence that circadian changes in alertness may be affected by more than one endogenous rhythm was presented by Folkard et al. [1985] who has consistently argued for the adoption of a multidimensional approach to arousal. Folkard [1990] suggested that alertness increases rapidly in the morning and reaches its peak between 1100 and 1400 hours, followed by a gradual decline. He suggested that it was in this way that the course of subjective alertness corresponded with psychomotor and memory tasks. Monk et al. [1983] suggested that circadian rhythms exist for a wide range of human physiological and psychological functions including mood. They proposed that these rhythms are self-sustaining in conditions of temporal isolation, indicating that they are under the control of internal oscillators or biological 'clocks'.

Inter-individual differences have been observed in the phase relationship between subjective alertness and body temperature rhythms. The morningness-eveningness dimension appears to be a contributory factor to these differences [Kerkhof, 1985]. During wakefulness, subjective alertness reaches its peak much earlier in morning types than in evening types. Individual differences are particularly pronounced at the beginning and end of the day and are generally accompanied by differences in sleep schedule. Kerkhof demonstrated that the difference in alertness between morning and evening types was far greater than the difference in sleep/wake schedules.

Thayer's [1987] model of mood posits two bipolar, orthogonal dimensions:
(i) Energetic Arousal - contrasts feelings of vigor and energy against tiredness and fatigue and (ii) Tense Arousal - contrasts tension and anxiety against relaxation and calmness. Matthew's' inventory incorporates a third dimension of hedonic tone or

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pleasure-displeasure, which is a product of the rotation of Thayer's two dimensions through 45 degrees. According to Thayer, endogenous rhythms influence our basic moods: alertness, tiredness, fatigue and energy are believed to be affected by circadian rhythms. He demonstrated a circadian rhythm in energetic arousal, which was found to peak by late morning, before dropping in the late afternoon, and climbing towards a secondary peak by early evening. Thayer observed differences between morning and evening type individuals and even components of extroversion such as impulsivity, appeared to affect these mood variations. Thayer was to conclude that the general concept of 'larks' and 'owls' was possibly related to differing levels of 'energetic arousal'.

Takahashi et al. [1985] reported that morning types tended to be higher in energetic arousal in the morning and lower in energetic arousal in the evening, whilst evening types demonstrated opposite trends. Matthews [1984] found that evening type subjects reported elevated levels of energetic arousal in the evening compared to the morning, whereas morning types displayed a trend towards higher energetic arousal in the morning.

Clodore et al. [1986] studied the diurnal evolution of alertness as a function of degree of morningness. They concluded that morning and evening types differed only during the morning: evening types fell asleep more frequently at 1000 hours and 1200 hours (MSLT) and rated lower self-alertness on arising than did morning types. Following a 2 hour reduction (either delayed bedtime or early arising) morning types rated their alertness lower when they had just awoken than when they had been awake for 2 hours. In contrast, evening types had the same low levels of alertness at 0800 hours, independent of the time elapsed since arising. Clodore et al. also concluded that the advanced rising time condition affected the general pattern of alertness to a greater extent than delayed bedtime.

Adan and Guardia [1993] demonstrated that the effect of the time of day in the scores of the mood scales had not been analysed in any depth as most studies had previously explored only two daily measurements (morning / afternoon). They investigated diurnal variations in mood, specifically in energetic arousal, tense arousal and hedonic tone. Adan and Guardia measured mood four times per day (at 0900, 1300, 1700 and 2100 hours). In addition, they examined individual differences such as morningness-eveningness and personality, since earlier studies had provided conflicting data [e.g. Monk, 1983; Stephan et al., 1985; Kerkhof, 1985]. Results from their study indicated the significance of morningness in

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energetic arousal. Morning types rated an energetical level escalating rapidly in the morning and peaking around 1300 hours before declining dramatically. Evening types detailed an increment in energy throughout the day, culminating at 2100 hours. Vigor in morning types was highest at 0900 hours and diminished throughout the day. This correlated negatively with tension, which showed a monotonic rise, peaking at 2100 hours. In terms of hedonic tone, morning types felt best in the morning and the sensation of well-being subsided in the evening, whilst the evening type presented the opposite trend. It was concluded that morningness was the most relevant individual trait when diurnal self-reported activation and positive mood were evaluated.

Adan and Guardia suggested that it would be more beneficial to study mood hourly, throughout the day, and to investigate the sub dimension of extroversion, namely impulsivity, which they proposed accounted for the daily functions of activation. In summary, their subjects reported a decrease in energetic arousal and hedonic tone from morning to evening, whilst tense arousal increased throughout the day. When individual differences were considered, only morningness influenced energetic arousal and hedonic tone. Morning types disclosed an earlier peak time than evening types in both scales. A phase advance was observed in morning types compared to evening types, oscillating between 2-12 hours depending on the test chosen. Introverts also demonstrated a phase-advance compared to extroverts; the difference again depending on the test parameter involved.

Horne and Ostberg's [1976] Morningness-Eveningness Questionnaire (MEQ) measures individual differences in circadian arousal or activity rhythms. The majority of people are encompassed in the 'intermediate' dimension - and around 10 percent of the population fall into the extreme categories. Patkai [1971] highlighted a significant positive correlation between 'morningness' and introversion and also between 'eveningness' and extroversion. Folkard [1975] demonstrated that the circadian rhythm in subjective alertness differed for introverts and extroverts in a manner similar to that observed by Patkai for morning and evening types. Webb and Bonnet [1978] examined sleep patterns in extreme morning and evening types and ascertained that in addition to retiring earlier and awakening earlier than evening types, morning types had a less variable sleep length and awakening time and took shorter naps than evening types. In relation to personality, Kerkhof suggested that correlations between extroversion and morningness-eveningness may have been a secondary result of personality differences in sleep-wake regulation. He recognised that introverted subjects presented an advance in activation in relation to extroverts of between one and four hours.

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Eysenck and Eysenck [1963] demonstrated that the association between morningness and personality lay in the extroversion sub factors of impulsivity and sociability. Revelle [1980] suggested that the interactive effects of time of day, arousal and impulsivity resulted from a correlation between impulsivity and the circadian arousal rhythm phase, with high impulsives reaching peak arousal later in the day than low impulsives. This hypothesis predicted that high impulsivity - but not high sociability - would be associated with eveningness. Larsen [1985] tested Revelle's hypothesis directly and found that the MEQ was significantly negatively correlated with the EPI assessed sociability, but not with EPI assessed impulsivity. Larsen accounts for the predictive failure by suggesting that Revelle's studies showed effects of state arousal while the MEQ is related to circadian rhythms in a trait of activity level.

Matthews [1988] investigated correlations between Horne and Ostberg's MEQ and personality traits. He administered the 16PF, the EPI and the MEQ to three groups of subjects. Although a large number of significant correlations were reported, these were partly attributed to intercorrelations between the 16PF primary factors. For all subjects, Matthews found that the strongest predictors of MEQ score were the primary factors Q1 [Radicalism] and Q3 [Self-sentiment]. Additionally, the MEQ and Extroversion were significantly negatively correlated in females only [Pearson correlation = -0.22, p < 0.05]. A direct comparison of the extroversion subfactors Impulsivity and Sociability revealed similar findings, both factors were significantly negatively correlated in females. Matthews conducted a test to check that the sex differences in correlations were not an artefactual result of sex differences in personality. He found that regressions were linear thus suggesting that gender differences in correlations were not artefactual. Matthews concluded that no significant correlations were observed between extroversion and MEQ in males but were evident in females, suggesting that extroversion appears to be linked to eveningness in females. This gender difference may be explained partly through differences in socialisation in the sexes. For example, Block, [1973] suggested that social contact, support and unity are more emphasized in the socialisation of females than males and any deviations from the sleep-wake cycle would be more threatening to females than to males. Hence, Matthews concluded that personality appeared to be linked to morningness-eveningness through exogenous rather than endogenous factors. Correlations between the MEQ and Extroversion tended to be negative but small, and were observed only in females. He suggested that personality may be related to social and cognitive factors determining the entrainment of circadian rhythms to the sleep-wake cycle.

In relation to sex differences, Wever [1984] has shown that men have a longer free-running sleep-wake cycle than women. Sex differences can also be expected in the action of 'zeitgebers' which entrain arousal rhythms to the sleep-wake cycle. Social cues are capable of acting as zeitgebers [Minors and Waterhouse, 1985], and the sexes differ in socialisation and response to social signals [Aubert, 1976].

Mecacci et al. [1986] examined other personality traits in relation to the MEQ and remarked that when extreme groups were compared, morning types were higher in neuroticism than evening types and higher psychoticism was associated with eveningness. Matthews [1986] observed that anxious subjects tended to be evening types. Wehr [1982] reported that depressive patients, who tended to be high in neuroticism [Eysenck and Eysenck, 1964], felt better in the evening than in the morning. With the exception of the morningness of neurotic subjects in Mecacci et al.'s study, the above data suggests an association between eveningness and personality traits associated with psychopathology.

It is argued that the 'wornout syndrome' may be due primarily to a disruption of the circadian mechanisms underlying sleep, rather than to mechanisms associated with the functions of sleep [Globus, 1969; Taub, 1981; Broughton, 1982]. Because the 'wornout syndrome' is not just a propensity to take more sleep, it is unlike the excessive daytime sleepiness that is associated with many sleep disorders, e.g. hypersomnia. Interestingly, the syndrome does bear some similarity to myalgic encephalomyelitis or the 'chronic fatigue syndrome' where the most common complaint is of chronic fatigue that may last for months or even years. My research into the sleep habits of chronic fatigue sufferers will be presented later in this thesis.

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Sleep

2 Sleep

2.1 Sleep Duration

Sleep had been, until quite recently, the dark one-third of our lives, and like the far side of the moon, a persistent mystery for all humanity. Many factors, ranging from: physical (external conditions), psychological (personality types and levels of stress) and physiological (diet, physical activity) are known to impact on the nature of sleep. Rosen and Rosenthal [1991] remarked on the association of self-reported sleep duration and season, whilst Rugg-Gunn et al. [1984] observed a link between a relatively shortened sleep duration and lower social class. Sleep is considered by most to be a coherent and organised system that sometimes resembles a 'need state'. We have all, at one time or another, experienced an overwhelming desire for immediate sleep, a sensation which can only be relieved by a sound slumber.

With regard to sleep duration, human infants spend about 50 percent of their time in sleep while adults devote one third of their day to sleep, even less in the case of the elderly (about 6 hours a day). Carskadon, Vieira and Acebo [1993] investigated the changes in sleep duration throughout the transition from childhood to adulthood. In addition to a reduction in overall sleep time, these researchers observed a shift in the timing of sleep towards later bedtimes and awakening times throughout this period. This change in sleep duration with maturation is affected by prevailing social and environmental factors, in addition to changing physiological requirements for sleep.

Normal sleep periods in the adult range from the reported five hours per night for Napoleon to the more than nine hours per night of Einstein. There is considerable intra- and inter-individual variability in the duration of sleep. This variability has important implications with respect to sleep related health issues. A survey relating lifestyle to health and mortality was conducted in California. Belloc and Breslow [1972] disclosed that 5,290 individuals documented sleeping between 7-8 hours per night. These individuals were reported to be healthier than those sleeping more or less than 7-8 hours per night. Wingard and Berkman [1983] observed that habitual sleep duration of 6 hours or less or 9 hours or more was associated with an increased rate of mortality.

An earlier survey conducted between 1959 and 1960 in America found a mean self-reported habitual sleep duration of between 8-9 hours per night [Kripke et al., 1979]. A six-year follow up suggested that men sleeping less than 4 hours per night were 1.3 times more likely to have died, whilst 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 were 1.8 times more likely to have died. Kripke et al. advocated an 8 hour sleep duration for optimum performance.

A more recent study of sleep habits in 50-65 year old men and women enquired into sleep pathology (e.g. snoring and excessive daytime sleepiness) in addition to sleep duration [Bliwise, King and Harris, 1994]. They exposed a greater association between sleep pathology and ill-health compared to sleep duration and ill-health. This was more pronounced in the case of long sleepers. Billiard [1987] investigated the sleeping habits of 58,000 male French army recruits between the ages of 17-22. He observed that over 60 percent of the respondents reported sleeping between 7-8 hours per night. Moreover, 18.3 percent reported sleeping more than 9 hours and 20.7 percent were sleeping less than 7 hours each night.

Horne [1988] hypothesised that only the first 4-6 hours of a normal night's sleep (the segment which contains the major portion of stage 4 or slow wave sleep, SWS) is necessary for the maintenance of a normal state of health. This is referred to as 'core sleep' as opposed to the last hours of sleep, termed 'optional sleep'. According to Horne, core sleep is necessary for cerebral restitution whilst the optional component of sleep is a behavioural immobiliser and varies in response to environmental factors; the latter is very much under a circadian influence, is more adaptable, and given time, can be reduced or extended without affecting daytime sleepiness. In terms of the structure of nocturnal sleep, SWS is confined to the first half of the sleep period, in contrast with REM (rapid eye movement) sleep which is more apparent in the second half. Unlike much of REM sleep, SWS seems to be obligatory; following sleep deprivation, most of SWS is regained compared to only half of REM sleep and little or none of stages 1 and 2 [Horne, 1983].

Investigations conducted by Broughton [1992] into the nature of sleepiness revealed that sleep deprivation and sleep satiation produced qualitatively different forms of sleepiness. He cites Globus' [1969] analysis into the effects of sleep deprivation and oversleeping in which Globus observed that sleep reduction produced mood states of irritability and over-responsiveness, unlike sleep

extension, which produced a distinctly different type of mood, characterised by under-responsiveness, lethargy and 'thick-headedness'. Broughton argues that separate states of sleepiness occur for a variety of reasons, which in turn can be differentiated in terms of their subjective and behavioural consequences.

Hartmann et al. [1972] examined personality differences in naturally short and long sleepers (less than four hours and greater than nine hours). They recognised that short sleepers tended to deny intrapersonal or interpersonal problems and some exhibited mild compulsive traits. Long sleepers revealed trends towards shyness and some were chronically depressed or anxious and almost all divulged evidence of inhibition in aggressive or sexual functions. Hartmann proposed that compared to long sleepers who exhibited more mild to moderate neurotic pathology, short sleepers had a greater need to appear acceptable or normal; in addition, they tended to be more socially adept and dominant in relationships with others.

In contrast, Webb and Friel [1971] failed to observe significant differences between groups of long and short sleepers on either personality, depressive state or performance measures. These researchers concluded that obtaining either extremely short or long sleep on a regular basis did not result in adversity. Several authors reported findings in accordance with Webb and Friel [e.g. Buela-Casal, 1984 and Buela-Casal, Carlos-Sierra and Caballo, 1992]. Globus et al. suggested that the discrepancy in the findings of the above mentioned studies might be due to errors in sampling. They proposed that individuals had specific requirements for a certain duration of sleep and large deviations from the ideal could prove detrimental to mood and well-being.

Before reviewing studies on sleep length, it is essential to clarify our understanding of what constitutes a 'normal' sleep duration. Most of us believe that we need somewhere between 6 to 8 hours and often refer to a 'shorter than ideal' sleep period as a 'bad night'. Anch et al. [1988] observed that people from diverse cultures sleep on average between 7 to 8 hours. In a historical review, Webb and Agnew [1975] remarked that the sleep of adolescents during the year 1910 averaged 1.5 hour more than a comparable sample surveyed in 1963. Hicks [1989] similarly reported a decline in sleep length of 30 minutes per night comparing students from 1978 to 1988. He concluded that escalating demands of modern lifestyle were to blame for the shortening of sleep duration. Carskadon [1990] proclaimed that although most studies of adolescent sleep habits show a pattern of diminishing total sleep time, a tendency to delay the timing of sleep, and an enhanced level of

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daytime sleepiness, many adolescents do not get enough sleep. Carskadon argued that this increased need for sleep was reflected in daytime sleepiness, mood and behaviour problems in addition to the development of major disorders of the sleep/wake cycle. Carskadon [1991] surveyed 3,000 high school students on sleep satisfaction. Although most averaged between 7 to 8 hours of sleep per night, 58 percent still complained of insufficient sleep. Agnew [1975] noted that less than one third of students awoke feeling 'fresh and rested' and that one third experienced difficulty in waking up every morning.

Webb and Agnew contend that we are 'chronically sleep deprived' (ideally requiring 9 to 10 hours nocturnal sleep) whilst Horne maintains that we only require 6 hours. These controversies on the optimal sleep length inevitably lead to the wider issue of the actual *function* of sleep. Carskadon and Roth [1991] suggested that one function of sleep was to maintain performance capacity and alertness. Alternatively, it was suggested that sleep serves a vital function and that when sleep is insufficient the organism strives for sleep during 'waking' hours — this is reflected by decreased alertness and impaired performance following insufficient sleep.

A review of recent studies examining the effects of sleep duration on performance led Carskadon and Roth [1991] to the following conclusions:

> (i) Acute sleep restriction, for example curtailing sleep by 2 or more hours for one night resulted in deficits in alertness and vigilance performance. However, more gradual sleep restriction over a longer period of time, e.g. reducing sleep by 30 minutes each week for 4 weeks, failed to demonstrate similar deficits.
> (ii) Impairment in performance and alertness occurred with as little as 2 hours of sleep loss.
> (iii) These adverse effects on performance and alertness accumulated with constant levels of sleep restriction.
> (iv) Sleep extension led to improved performance and alertness, and
> (v) Sleep restriction led to changes in nocturnal sleep, indicative of an increased sleep drive.

In studies of acute sleep restriction there are problems in assessing performance tasks because of prolonged practice effects. However, in gradual sleep restriction studies, where performance is not assessed as often, practice effects are not so evident. This could lead to misinterpretations of the findings, particularly if the effects of acute and gradual sleep reduction are to be compared [Horne and Wilkinson, 1985].

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Hawkins and Shaw [1992] conducted a study of sleep duration and subjective sleep quality in college students. They observed differences in sleep duration between weekdays and weekends, which coincided with improvements in subjective sleep quality on weekends. However, when these subjects reduced their sleep duration at a later date, no corresponding reduction in sleep quality was documented.

2.2 Oversleeping

Most individuals will readily seize any opportunity to extend their sleep, this tendency towards having a 'lie-in' is particularly evident on weekends, when external constraints are not as stringent relative to weekdays. Carskadon and Mancuso [1988] observed that the weekend sleep profile was characterised by a delay in bed-time and awakening. This suggests that the weekend sleep indulgence fulfils the individuals requirement for sleep which remains incomplete during weekdays. Webb and Agnew [1975] studied this tendency in 16 male volunteers and realised that they slept on average for 126 more minutes on weekends than on basal nights. This was interpreted as a reflection of the sleep restriction normally imposed upon sleep length. On the basis of their experimental results they advocated that we are 'chronically sleep-deprived' - and claimed that we should be sleeping 9 to 10 hours per night rather than 7 to 8 hours. In further support of this viewpoint, Webb and Agnew indicated that many people do not feel fully refreshed on awakening; rather than awaken naturally when sleep is satiated, they rely on alarm clocks to artificially terminate sleep.

Globus [1969] was the first to remark on the effects of oversleep on the psychological state of individuals. He gathered retrospective information from students about their sleeping habits and from this data, he recognised a particular condition which followed oversleep, which he termed the 'wornout syndrome'. Globus noted that this syndrome was characterised by descriptors such as 'fuzzy', 'wornout', 'lethargic' and 'difficulty in getting going' and remarked that this syndrome was most likely to occur in individuals who had obtained more than 10 hours of sleep and were not making up for any prior sleep loss. He concluded that healthy young adults systematically report depressive affect, anergia, fatigue and impaired cognitive functioning after sleeping longer than 10 hours.

Taub and Berger [1976] studied the effects of changing the phase and duration of sleep on subsequent mood and performance. They investigated the effects of:
(i) sleep extension (+ 3 hours), (ii) sleep reduction (- 3 hours) and (iii) shifts of

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habitual sleep time on mood and performance in 10 subjects who regularly slept 9.5 to 10.5 hours per night. Performance measurements were obtained from the 45-minute Wilkinson auditory vigilance task, and a 5-minute modified version of the Williams and Lubin experimenter-paced addition task. Mood was assessed using two self-report inventories, Thayer's AD-ACL and Lorr et al.'s multifactorial adjective checklist were administered to measure mood 30 minutes after awakening, at midday and in the evening.

Following each 3-hour altered condition Taub observed an equivalent decline in vigilance performance and in subjective arousal as measured by the mood scales. Results supported the hypothesis that acute disruptions of the 24-hour sleep-wake cycle lead to degradations in performance largely independent of total sleep time. The findings implied that maintaining accustomed sleep schedules was of equal or greater importance to the integrity of most waking behavioural functions than obtaining some invariant amount of sleep. Taub and Berger concluded that acute extensions of habitual sleep duration in the healthy young adult produced detrimental effects on subsequent waking behaviour, performance and overall mood. The degree of impairment following oversleep was of a similar level as that typically observed following partial or total sleep deprivation [Wilkinson, 1968].

Vigilance and performance efficiencies are commonly used as indices of fluctuating alertness, and provide information concerning CNS functioning. The psychological performance tasks that are most sensitive to the effects of sleepiness are those that are simple, dull, monotonous, easy to score and not prone to large practice effects, e.g. simple reaction time and auditory vigilance tasks [Horne, 1991]. Researchers at the Walter Reed Army Institute of Research identified tasks sensitive to the effects of even one night's loss of sleep as those which were not self-timed, which lasted for at least ten minutes and which were not intrinsically motivating. At the same time, researchers in Cambridge developed the Lapse Theory, which proposed that the effect of sleep loss was to increase the number of lapses in attention, possibly through microsleeps. When subjects were faced with the five-choice serial reaction task, where the subject responds to one of five lights on a continuous basis, average response speed tended to decrease over a period of twenty minutes in sleep-deprived subjects compared to rested controls. This failure in attention was attributed to a lowering of arousal level, which may be temporarily counteracted by the subjects' motivation to remain awake. Evidence supporting the theory has come from experiments on the interaction of sleep loss with other manipulations known to increase arousal level, such as incentives and noise [Empson, 1989]. Dinges

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[1991] reported that simple reaction time tasks requiring sustained attention are far more sensitive to sleepiness than complex tasks where possible interest may ameliorate any deficit. Wilkinson's auditory vigilance task is well suited to probe sustained attention, in paradigms like sleep deprivation. However, because of its lengthy nature [45-50 minutes] it is of doubtful value in diurnal studies where testing is required at short intervals.

In a later study, Taub [1980] examined behavioural functions in 16 healthy adults following ad-libitum extended-delayed sleep. These subjects regularly slept from midnight to 0800 hours and were required to undergo one control night of habitual sleep and an ad-libitum sleep session, conditions were counterbalanced. Thirty minutes after awakening from each condition measurements were obtained from a 30-minute short-duration auditory vigilance task [Deaton et al., 1971] and a continuous self-paced 20-minute test of visual choice reaction time [Wilkinson et al., 1975] to evaluate sensorimotor performance. Short-term memory was assessed by a free recall task [Taub, 1974]. The Stanford Sleepiness Scale [Hoddes et al., 1973] was used to evaluate relative levels of subjective propensities for activity versus sleep. Following extended sleep, Taub observed a significant decline in performance and significant elevations of subjective sleepiness 90 minutes after awakening. Ad-libitum sleep produced impaired performance on both auditory and visual tasks. These findings led Taub to conclude that behavioural deficits reflect a genuine decline of CNS response capacities rather than habituation or fatigue in peripheral sensory receptors [Williams, Lubin and Goodnow, 1959]. Taub proposed that the decline in performance and memory was attributable to the temporal displacement of extended sleep into the daytime hours.

Taub and Berger [1969, 1973] reported performance decrements after oversleep as well as undersleep. Their studies differentiated between the effects of simple extension and habitual sleep amount. In later studies, Taub and Berger [1976, 1980] varied sleep duration and sleep timing from the habitual hours. They proposed that a change in circadian placement was sufficient to impair performance and that in oversleeping it was just such circadian *desynchronosis* (rather than the increase in sleep duration) that led to performance deterioration.

Investigations of oversleep, whereby normal 7.5 to 8 hour sleepers are encouraged to extend their sleep in the morning, have shown that 2 hours or more of extra sleep is easily obtained [e.g. Feinberg et al., 1980; Gagnon et al., 1985]. This additional sleep does not produce a pro rata sleep reduction the following night but is 'extra'

sleep. There is little evidence to show that daytime alertness and performance is improved to any remarkable extent by oversleep. This does not fit in with Webb and Agnew's 'chronic sleep loss' proposition, as oversleep ought to leave one feeling more refreshed and performance improved. Carskadon [1986] permitted subjects to take up to 10 hours sleep a night for 4 nights. There was a small effect evident on the second day, in the direction of diminishing daytime sleepiness. Roehrs [1989] allowed up to 2 hours of extra sleep for 6 nights in groups of "sleepy" or "alert" subjects. Although MSLT and performance data from the extended sleep days divulged significant improvement, indicative of curtailed daytime sleepiness in both groups, the effects were minor.

Broughton [1985] noted that SWS reappears in sufficiently extended sleep periods. He distinguished a 12.5 hour rhythm of SWS, thus contesting the previously held belief that SWS is a reflection of the amount of prior wakefulness and that it does not decrease with the amount of time spent asleep. Broughton concluded that SWS follows a bimodal 12 hour rhythm which is seen immediately upon extended-delayed sleep and can be fully phase-shifted with habituation. Webb [1986] revealed a similar SWS resurgence around 12 hours after sleep onset in extended sleepers.

The extended sleep paradigm was used by Gagnon and De Koninck [1984] to test the hypothesis of a 12 hour SWS rhythm. The reappearance of SWS around 13.5 hours after sleep onset observed in volunteers extending their sleep to 15 hours supported Broughton's original proposition of a circasemidian rhythm of SWS. The re-emergence of SWS was observed following simple extension from usual sleep patterns and following acute and adapted delayed extended sleep.

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2.3 Sleep Restriction

Studies of the effects of sleep restriction on mood are few and far between; most studies have focused on the effects of altering sleep duration on performance. Webb and Agnew [1974] restricted the sleep of 16 young adults by 2 hours for 60 days and noted the only significant decrement in performance was a steady decline in correct detection in an auditory vigilance task. This degression was attributed to a decline in motivation rather than to the effects of sleep loss. However, it could be argued that the principle effect of sleep loss is on motivation. A confounding effect of this falling motivation is the worsening of performance. This link is evident in the Walter Reed studies which indicated that providing subjects with incentives was sufficient for optimal performance to be maintained. There are impressive findings from sleep loss studies showing how powerful the impact of incentives on performance at these monotonous tasks can be [e.g. Williams, Lubin and Goodnow, 1959].

However, Globus et al. [1977] examined the long term effects of gradual sleep reduction on performance and mood and concluded that 6-8 months of gradual sleep restriction, down to 4.5 to 5.5 hours per night did not prove to be detrimental to performance. They observed that subjective fatigue was the limiting factor in determining tolerability to gradual sleep restriction. In spite of the fatigue encountered, sleep was curtailed by 2.5 to 3.5 hours during the experiment and maintained 1 to 2.5 hours below baseline for at least one year - during which time levels of subjective fatigue returned to baseline. The study implied that sleep length could be adapted to conform to a changed lifestyle - but only to a certain extent. Wilkinson [1963] had earlier examined the effects of recuperative sleep after a period of sleep deprivation and likewise, indicated performance decrements.

2.4 Measures of Sleepiness

Sleepiness is widely regarded as a multi-faceted phenomenon, a transitional state
between wakefulness and sleep. Various objective [e.g. Multiple Sleep Latency Test
(MSLT), Richardson et al., 1978; Carskadon et al., 1986] and subjective [Stanford
Sleepiness Scale (SSS), Hoddes et al., 1973 and Visual Analogue Scales (VAS),
Folstein and Luria, 1973] tests have been devised to assess sleepiness.

Dinges [1989] has reviewed the major assessment measures of sleepiness and notes
a certain degree of discrepancy amongst the different tests. He proposes that this
incongruity has two explanations; the first assumes that sleepiness is a basic
physiological process, but not all evaluations of sleepiness are equally sensitive to
variations in it. The second explanation is predicated on the view that there are
different kinds of sleepiness and that this multidimensionality is reflected in the
different assessments.

Carskadon and Dement [1981, 1982] have suggested that one factor known to alter
daytime sleepiness and alertness is the amount of prior sleep and wakefulness.
Restriction of the usual 8 hour time in bed (TIB) by as little as 2 hours produces
enhanced levels of daytime sleepiness, and extension of TIB produces increased
alertness. Carskadon [1981] maintains that sleep is a unitary physiological state that
can be measured by either objective or subjective means - high correlations were
obtained between the MSLT and the SSS in early studies. In contrast to the unitary
view, Broughton [1992] has proposed that daytime sleepiness consists of
qualitatively distinct states. He has presented data to illustrate that sleepiness from
undersleeping and sleepiness from oversleeping produce qualitative differences in
subjective states and in performance. If we accept Broughton's position, then it
follows that any measure used to assess sleepiness in one context should be
employed with caution in another. In addition, it may not be fruitful to use a single
measure of sleepiness, but more prudent to use a variety of tests.

A study of daytime sleepiness in healthy sleepers failed to establish a relationship
between objective tests (MSLT and reaction time lapses) and subjective assessments
(SSS and VAS), although there were significant correlations between the two
subjective measures and between the two objective measures [Laverne and
Johnson, 1992]. Significant correlations were also disclosed between mood ratings
(POMS) and subjective sleepiness. Individuals with high ratings of sleepiness also
judged their mood as poorer than those with lower ratings and performed

unsatisfactorily in performance tasks. These results support Broughton's view of the existence of different states of sleepiness. Johns [1991] also commented on the discrepancy between the MSLT ratings of sleepiness and those of the SSS, as recorded by Cook et al. [1988]. He suggested that these subjective accounts may be related more to tiredness and fatigue than to actual sleep propensity and devised a new questionnaire, the Epworth Sleepiness Questionnaire (ESS) which, he advocated, measures sleep propensity in a simple standardised way.

Instead of comparing the different measures of sleepiness, Dinges [1988] proposes that it is more important to administer the appropriate form of test that is deemed most relevant at the time. He used the SSS to assess post-sleep inertia following two-hour long naps. Dinges observed a dramatic dissociation between behaviour and subjective self-reports of sleepiness. It was concluded that more work was required on the contextual factors influencing self-reports of sleepiness, particularly in tests like the SSS.

From these studies into sleep extension and restriction, it is clear that subjective measures have been deemed sensitive enough and appropriate to gauge alertness. On this basis, I will employ a battery of subjective measures for the studies to be reported in this thesis. A major concern of this thesis will be to focus on the nature of the SSS and ask: does it actually measure sleepiness or some other condition, such as tiredness? This question will be considered later in the thesis.

3

Mood and Sleepiness

3 Mood and Sleepiness

3.1 Introduction

In the context of mood assessment, 'sleepiness' is an ambiguous term. In earlier versions of the POMS [Mc.Nair et al., 1971] 'sleepiness' was included as an adjective, but had to be deleted as it had significant factor loading for Vigor, Fatigue and Confusion, as well as more minor loading for Depression and Anger. This supports the view that sleepiness should not be considered as a unitary concept, but rather, as one with many dimensions.

Mc.Nair et al. [1971] advocate that sleepiness may have three dimensions, associated respectively with Vigor, Confusion and Fatigue. Horne [1991] suggests that Vigor and Confusion may be relevant to core and optional sleepiness. The third, Fatigue, might be associated with Globus' 'wornout syndrome', which follows two hours or more of oversleep, and may also be a variant of post-sleep inertia.

The POMS adjectives/phrases describing the mood states are listed below:

> (i) Confusion — confused, unable to concentrate, muddled, bewildered, inefficient, forgetful and uncertain about things.
>
> (ii) Vigor — lively, active, energetic, cheerful, alert, full of pep, carefree and vigorous.
>
> (iii) Fatigue — wornout, listless, fatigued, exhausted, sluggish, weary and bushed.

Most of the terms used by Globus to describe the 'wornout syndrome' were the same or similar to the POMS descriptors for the dimension of Fatigue. Importantly, Globus observed that if the oversleep was replacing lost sleep and did not exceed 10 hours, then subjects were most likely to report feeling 'just great'. When this recovery sleep exceeded 10 hours then feelings of 'just great' and 'wornout' were obtained with equal frequency. However, when the extra sleep was oversleep without a prior sleep deficit then subjects were more likely to report feeling 'wornout'. What is particularly interesting about these findings is that the descriptor most frequently used by subjects was 'sleepy' [Globus 1970]; unfortunately, no objective measures of daytime sleepiness were administered.

The work of Globus was followed up in the 1970's by Taub, who observed from his own experiments, that while two hours of sleep extension led to increments in fatigue and sleepiness, more sleep was difficult to achieve, and if obtained, was not refreshing. More recent work into the effects of extended sleep on mood in normal subjects confirm these findings of raised levels of fatigue [Broughton, 1982; Hawkins et al., 1985]. Studies of oversleep using the MSLT have failed to discover any increases in daytime sleepiness. Paradoxically, sleep latencies lengthen a little, seemingly pointing to greater alertness. In contrast with these objective measures, subjective ratings of sleepiness as reflected by the SSS show distinct shifts towards enhanced sleepiness [Carskadon et al., 1986].

3.2 The Stanford Sleepiness Scale

The Stanford Sleepiness Scale (SSS) [Hoddes et al., 1973] is one of the most commonly used self-rating sleepiness scales. It is used to evaluate relative levels of subjective propensities for activity versus sleep. The scale consists of seven ranked statements from:

1. Active, vital, alert, wide awake
2. Functioning at high level, not at peak, able to concentrate
3. Relaxed, awake, not at full alertness, responsive
4. A little foggy, not at peak, let down
5. Fogginess, losing interest in remaining awake, slowed down
6. Sleepiness, fighting sleep, prefer to be lying down, woozy
7. Almost in reverie, sleep onset soon, lost struggle to remain awake

Subjects are asked to place a mark next to the scale value [associated with a series of descriptors] which best describe their levels of sleepiness. The scale has been found sensitive to changes occurring over time intervals as small as fifteen minutes.

Taking a critical look at the individual items on the SSS highlights certain deficiencies. It is clear that the scale is ambiguous; several of the statements are more pertinent to the 'wornout syndrome' and to the POMS Fatigue dimension, than to what is normally encompassed by the more traditional 'alertness-sleepiness' dimension [and to POMS Vigor]. It is clear that the SSS confounds at least two independent factors described in POMS terminology as Vigor and Fatigue. So the reason why oversleeping bias SSS scores in a direction commensurate with greater sleepiness is because participants respond to the 'fatigue' descriptors more than they do to those associated with 'sleepiness'.

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Dement et al. [1976] maintained that sleepiness was an "independent feeling state", and despite some degree of overlap between sleepiness, tiredness, and depression and lack of initiative, they emphasised the distinction between sleepiness and physical tiredness - a distinction which was supported by clinical observation [Moldofsky, 1989]. Carskadon and Dement [1982] distinguished between physiological and manifest sleepiness or sleep tendency. Physiological sleepiness was exposed only in the absence of alerting factors whilst manifest sleepiness was believed to be influenced by many such factors.

The most damaging criticism of the SSS was made by MacLean et al. [1989]. They examined the structure of the SSS as a result of comments made on the lack of relationship between the SSS and objective measures by Dement [1976]. MacLean et al. [1992] focused on two issues; (i) the extent to which the component items of which each level of the SSS is constructed are homogenous and (ii) the factorial structure of these component items. The descriptors for each level of the SSS were reconstituted to form 24 True/False items. These, together with the SSS and four visual analogue scales (calm-irritable, happy-sad, energetic-sluggish, relaxed-tense) were administered to 340 volunteers. Subjects were asked to check items from the reconstituted SSS: at the same time they were also asked to rate items from the original SSS. These two ratings were then checked for any degrees of correspondence. MacLean et al. remarked that on many occasions subjects would also check items from the 24 items (reconstituted SSS) that did not correspond to the original SSS. This particular comparison represented their most stringent criteria. A second method of comparison was employed, in which at least the pertinent items should be checked - 68 percent of the protocols reached this criterion. Considerable heterogeneity was found in the results - individuals checking Level 4 say of the SSS would endorse those items but in addition to items from other levels. A principal components analysis was conducted to explore the dimensionality of the SSS. MacLean et al. proposed that the SSS contained two factors; (i) an activation factor [accounting for 24.2 percent of the variance] characterised at one extreme by the statements "I feel slowed down" and "I am not at full alertness" and at the other by "I feel active" and "I feel wide awake" and (ii) a sleepiness or a loss of control factor (accounting for 20.6 percent of the variance) characterised by the statements "I am losing the struggle to remain awake", "I am beginning to lose interest in remaining awake" on one extreme, and on the other by "I am awake" and "I am able to concentrate". They failed to establish a systematic relationship between the SSS and Factor 1 or 2. MacLean et al. concluded that the descriptors defining each level of the SSS were not

homogenous, and that sleepiness, as defined by these items, is not a unidimensional construct.

Broughton [1982] has emphasised some of the shortcomings of the SSS. He indicated that while the SSS had been shown to predict performance decrements following acute sleep deprivation [Herskovitch and Broughton, 1981], these findings had not been replicated. He concluded that "the assumption that excessive sleepiness is a unitary phenomenon varying only in degree should be abandoned".

MacLean has commented on Horne's [1988] dichotomy of sleepiness into 'core' and 'optional' sleepiness, whereby 'core' sleepiness relates to "increased cerebral impairment .... and the other relates to whatever governs optional sleep". MacLean has speculated that Factor 2 (sleepiness or loss of control) is closely aligned to optional sleepiness, whereas Factor 1 (activation) is aligned to core sleepiness. MacLean suggests that a regimen of partial sleep deprivation that allows for the integrity of core sleep should result in altered scores on Factor 2 (which should also vary in a circadian manner) but not Factor 1. In relation to mood, Horne has suggested that Factor 1 relates to POMS Vigor and Factor 2 has similarities to POMS Fatigue.

3.3 Sleep Manipulation and Mood

Roehrs et al. [1973] investigated mood in individuals prior to sleep and at intervals after awakening. They recognised a consistent change in mood between morning and afternoon, and proposed that sleep may modulate mood changes. Roth et al. [1976] used the Clyde Mood Scale to determine the effects of sleep deprivation on 11 students. The scale was completed upon arising after each of three sleep regimens: normal baseline sleep, sleep deprivation and sleep recovery phase. The sleep deprivation treatment resulted in significantly higher scores on the three scales of sleepy, friendly and aggressive. However, the data were inadequate for determining whether the mood changes were a direct result of lack of sleep or of the protocol used to enforce the deprivation. What the results did indicate was that a single night of sleep deprivation had significant effects on morning mood states. Roth et al. observed a significant relationship between the sleep profiles and indices of mood. They suggested that one of the functions of sleep is the regulation of mood. Moreover, they concluded that mood is a relevant and measurable attribute of wakefulness that varies from day to day and within a day and which might bear some relation to the physiology and psychology of sleep.

Taub and Berger [1974] measured 36 subjects mood state at three different times of the day on a 57 item multiple adjective check list (MACL) derived from the instruments of Mc.Nair et al. [1964] and Raskin et al. [1967]. Five of the mood scales indicated diurnal variation with increased concentration, activity and friendliness as the day progressed, and diminished fatigue and depression (the final measure was taken around 1730 hours). By shifting subjects normal sleep pattern either by advancing or delaying their usual hour of retiring, Taub and Berger perceived less activity, cheerfulness and concentration after displacements of two or four hours.

In a systematic approach, Wehr et al. [1993] compared the effects of 28 consecutive nights of sleep extension [14 hour bedrest] to 7 normal nights [8 hours bedrest] in healthy adults. On baseline nights, subjects slept on average 7.2 hours. However, they slept in excess of 10 hours for 3 nights, and around 9.1 hours for the next 4 nights. Over the next 3 weeks, the average total sleep time was 8.8, 8.9 and 8.4 hours. Subjects reported being more energetic, less fatigued and happier following extended sleep. The researchers viewed these improvements in mood as evidence of a pre-existing sleep deficit. However, Totterdell et al. [1994] suggested that the enhanced mood reported by Wehr et al. was a consequence of the timing of bedtime: they argue that an earlier than usual sleep onset, rather than sleep duration, was more likely to be associated with inflated mood.

A recent study investigated the effects of long-term sleep extension on mood and subjective alertness [Harrison and Horne, 1996]. Extended sleep was monitored [up to 10 hours] for 14 consecutive nights. Findings revealed no beneficial or adverse effects of sleeping an extra hour on mood or subjective alertness. These researchers found little evidence to support the notion that young adults are sleep deprived. In addition, it was argued that the ability to indulge in extra sleep is not necessarily indicative of a physiological need for sleep. Harrison and Horne used an earlier than normal bedtime to increase sleep duration, with normal wake times preserved. Employing this method has failed to show detrimental effects on mood or subjective sleepiness the following day [Roehrs et al., 1989; Wehr et al., 1993]. By contrast, Totterdell et al. [1994] suggested that retiring earlier than usual has a beneficial effect on mood. Akerstedt et al. [1994] proposed that the timing of final awakening, more specifically the closeness to the circadian acrophase of the temperature rhythm, was associated with increased ratings of sleep quality, feeling refreshed and ease of awakening.

3.4 Sleep Inertia

The term 'sleep inertia' refers to the phenomenon of disorientation and/or inferior task performance occurring immediately after awakening from sleep relative to pre-sleep status [Kleitman, 1963]. The duration of sleep inertia varies from 4 to 30 minutes, and apart from producing a dysphoric mood has a negative effect on performance - which is one reason why all tests should be administered at least 30 minutes after awakening [Wilkinson, 1972].

It is important to distinguish post-sleep inertia from the 'wornout syndrome'; the latter can persist for 4 to 5 hours after awakening [Globus, 1969]. Dinges has indicated that the amount of SWS in a nap may contribute to the severity of sleep inertia. It appears that any factor associated with increased sleep depth enhances the severity of sleep inertia at awakening. Sleep inertia is said to be more severe around the circadian nadir (0300 hours) and less severe near the circadian peak (2000 hours) of alertness. Most studies on sleep inertia involve participants taking afternoon naps and have illustrated that mood variables, especially self-reports of sleepiness (using the SSS), fatigue, and activation improve quite consistently after naps [Percival, 1983].

If we accept that SWS is associated with a 12 hour rhythm [Broughton, 1990] and governed by a circadian factor, then this may serve to extend our understanding of sleep inertia. The degree of disorientation experienced upon awakening is directly proportional to the depth of sleep - awakening from sleep stages 1 and 2 results in comparatively less disorientation than awakening from deeper sleep (SWS). The effects of sleep inertia however, are transient and rarely last longer than 30 minutes. It is important to note that sleep inertia is a slow wave sleep dependent phenomenon. However, in both Globus' study and the current study the sleep extensions are still morning awakenings where REM sleep or stage 2 is present.

3.5 Sleep and Depression

Sleep may be defined as a means of detaching the person from the environment. This may serve psychological motives for avoiding painful aspects of the social environment. The idea that sleep may become involved in emotional conflict is a simple extension of known psychogenic factors in fatigue and neurasthenia [Bartley and Chute, 1947]. Horney [1937] says that excessive sleeping is one way of "narcotising" anxiety. Other authors associate it with an obsessive-compulsive

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neurosis and state that it is sometimes observed in depressed patients [Drake, 1949]. The excessive sleeper is identified as being passive, shy and dependent, someone who has learned to use sleep as a defence against aggressive feelings.

Disturbed sleep is one of the main symptoms of depression. Two types of sleep disturbance have been described; some complain of longer sleep latencies, increased intermittent waking time and insomnia associated with early morning awakenings, which leads to a reduction in total sleep time; others, particularly if younger, sleep excessively [Kvist, 1980]. The composition of a night's sleep and the evolution of sleep stages through the night are often modified in depression. Patients frequently exhibit shortened REM latencies, enhanced REM sleep density, and a shift of the distribution of REM sleep toward the first third of the night. The most pervasive abnormality is a decline or absence of delta (SWS) sleep. In contrast, Mendelson et al. [1987] conducted sleep studies on eight patients with major depressive disorder and matched controls. Although total sleep time and sleep efficiency were curtailed in depressed patients, there was no significant difference in REM latency between the two groups. More recently Myriam [1994] has reported that around 90 percent of depressed patients display alterations in their EEG. These changes include low delta sleep and more delta sleep in the second cycle, in addition to an increase in total REM and shortening of the first REM latency. Myriam suggests that there is a total sleep advance, which correlates with parallel circadian shifts in body temperature. However, decrements in both REM sleep and SWS have also been described in various psychiatric and medical conditions and therefore do not appear to be pathognomic of depression.

There is considerable inter-individual variability in the amount of REM sleep and in the latency to REM sleep among depressives. The contradictory electroencephalographic findings in studies of depression may be due to a number of factors. The differing amounts of REM sleep among depressives may be attributed to the type and severity of depression. Not all depressed patients respond to sleep deprivation or circadian manipulation. Wu and Bunney [1990] in a review of the antidepressant response to sleep deprivation observed that 36 percent of patients with endogenous depression showed no antidepressant response to sleep deprivation. They concluded that there may be different subtypes or phases of depression that vary in sensitivity to sleep manipulations. Mendels and Hawkins [1971] reported that the more severe the depression the more sleep was fragmented. Severely depressed patients had less delta and REM sleep and more wakefulness. Hauri and Hawkins [1971] found an inverse correlation between the percentage of

REM and severity of depression as measured by the Beck Depression Inventory [BDI]. Increased REM percentage was associated with decreased scores on the BDI. However, the study was flawed in that a significant number of patients were receiving drugs known to affect REM sleep.

Snyder [1972] postulated that REM sleep was more susceptible to change because of stresses. In the initial phase of the illness, when depression was most severe, there was a deficit of REM sleep. However, when recovery of depression began, REM rebound occurred. This would account for the increase of REM sleep and the reduction in REM latency, commonly observed by various researchers. Therefore, it is proposed that the severity of the illness could explain the contradictory findings observed in the EEG's of depressives. Clearly, if one sample is more severely depressed than another, then, according to Snyder and Hawkins, differences are to be found in sleep structure between depressives.

Another explanation for the differing percentages of sleep stages in studies of depression is the type of depression being examined. For example, Snyder [1972] reported that the sleep of delusional depressives was more severely disrupted than that of non-delusional depressives. Delusional depressives had more wakefulness and longer periods of stage 1 sleep in association with decreased SWS [around one minute per night] and REM sleep. Therefore the inconsistencies in REM activity or density may be due to a lack of adequate, standardised techniques being employed by various researchers. An additional factor known to impact on the sleep structure in depressives is the age range of the sample studied. It is well documented that the structure of sleep changes with increasing age. Sleep becomes more fragmented and SWS diminishes. It is vital that studies examining the sleep structure in depressives compare their findings to a healthy age-matched control group, to avoid such confounding factors.

Myriam [1994] questions the mutual relationship between sleep disorders and depression - do depressive disorders engender sleep disturbances or is it sleep disorders that bring about depression ? Myriam observed that frequent complaints by depressed patients include restlessness, frequent awakening, early morning awakening and a non-restorative type of sleep, i.e. sleep of normal duration but of subnormal quality. This type of non-restorative sleep has also been reported by chronically fatigued patients and will be discussed in more depth later.

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Various investigators have examined a number of manipulations of sleep in order to ameliorate symptoms of depression. Pflug [1976] used total sleep deprivation as treatment for depression. He observed an improvement in about 60 percent of cases; however this was dependent upon continuing wakefulness, the benefits lasted only a few days and were often followed by an exacerbation of symptoms. Schillgen and Tolle [1976] realised that partial sleep deprivation in the second half of the night was as effective as total sleep deprivation in alleviating depressive symptoms. They allowed as much as four hours of sleep prior to 1 or 2 a.m. This finding has important clinical implications in that partial sleep deprivation can be carried out repeatedly, thereby preventing relapses.

Sack et al. [1988] conducted a controlled, balanced order, crossover study in which the same depressed patients were partially sleep deprived in the first and second halves of the night on two different occasions, one-week apart. Partial sleep deprivation in the second half of the night had a significantly greater antidepressant effect than partial sleep deprivation in the first half of the night. Patients had significantly shorter sleep durations and reduced REM sleep in the significant treatment condition. They proclaimed that the timing and duration of sleep was particularly important in partial sleep deprivation therapy. The shorter the sleep duration, and in particular the REM sleep duration, the greater the antidepressant response. However, in the above study, the amount of sleep and sleep structure was different under both conditions. Therefore, to determine whether the timing of sleep is a critical variable in partial sleep deprivation therapy, both sleep duration and sleep stages should be balanced.

Vogel [1975] conducted a parallel design study in which endogenous depressives' responses were assessed to two treatments carried out for three weeks: selective REM sleep deprivation and selective non-REM sleep deprivation. Vogel found that selective REM sleep deprivation was modestly superior to the latter condition [p < 0.03]. However, patients who improved were those who showed evidence of REM sleep rebound during recovery night sleep following the REM sleep deprivations. Unfortunately, the results of this arduous study have not been replicated, perhaps because REM sleep deprivation is technically difficult to do.

In contrast to Sack et al.'s findings, Giedke et al. [1992] compared the effects of early and late partial sleep deprivation on mood in 26 depressed patients. They recognised that both procedures had a significant therapeutic effect, and concluded that the timing of sleep reduction was not a significant factor. Varying rates of

success have been documented in different studies. Myriam [1994] has concluded that the mood elevating effects of sleep manipulations, such as sleep deprivation, depends on the type of depression.

Aserinsky [1969] and more recently Feinberg et al. [1980] studied extended sleep (longer than 6-8 hours) in normal subjects and observed both a high REM density and a tendency for successive REM periods to become progressively shorter. These findings were also recounted by Vogel [1980] in a study comparing endogenously depressed patients to insomniac controls. It was speculated that a circadian rhythm disturbance - such as phase advance of the REM non-REM cycle, or a loss of rhythmicity, might account for this phenomenon. Gillin [1984] aware of Aserinsky's concept that such measures as enhanced REM density might reflect sleep satiety, proposed that depressed patients were in a continuous state of sleep satiety. If this was true then depressed patients might be expected to show increased wakefulness during the day. An MSLT study by Kupfer et al. [1980] confirmed this. They suggested that a disruption of social rhythms could be responsible for triggering the onset of depression in vulnerable subjects. The mood changes seen in depressives were thought to result from the shifting of a critical phase of the REM / temperature cycle, such that it interacts with sleep.

Mendelson [1987] reviewed studies which examined depression as a consequence of disordered rhythms. Most of the work revolved around the REM Phase Advance Hypothesis [see Wever, 1979] which suggests that the circadian rhythm of REM sleep is phase-advanced relative to the sleep-wake rhythm. MacLean et al. [1986] proposed a phase-advance of the circadian rhythm of REM sleep propensity relative to the sleep-wake cycle, to account for the abnormalities of REM sleep commonly found in depressed patients. Wehr [1982] observed that a number of circadian rhythms were phase-advanced relative to sleep in depressed patients. He hypothesised that this might be responsible for the abnormally early occurrence of REM sleep in depression. Wehr predicted that if this were true then a corresponding advance in the timing of the sleep schedule would correct the internal phase disturbance and improve depressive symptoms. A 6 hour phase advance of sleep schedules was indeed recognised and associated with alleviation of depressive symptoms. However, his findings resulted from a series of uncontrolled experiments and thus must be interpreted with caution. Surridge-David [1987] disclosed that it was possible to induce depression in normal subjects by delaying the sleep schedule by 6 hours from the usual pattern. He discovered a modest lowering of mood in all subjects; and two subjects became markedly depressed.

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The fact that relapses occur after recovery sleep suggests that sleep may be depressogenic. This could explain why patients with endogenous depression usually feel worse in the morning after a period of sleep, and feel best in the evening following a period of wakefulness. Gillespie [1979] proposed that the daily degression in symptomatology may be due to a mechanism similar to that responsible for the decline in depressive symptoms that occurs after total sleep deprivation. Some researchers [Van den Hoofdakker et al., 1985] revealed that subjects who show diurnal mood variation have a better chance of responding to sleep deprivation. They suggested that sleep deprivation acts in part by preventing the nocturnal deterioration in mood that often takes place in patients with endogenous depression. Some depressed patients display a different pattern of diurnal mood, contrary to the pattern seen in endogenous depression; they feel better in the morning and worse in the evening (they also feel worse after sleep deprivation). The tendency to display one pattern or another may be explained by differences in personality and propensity towards morningness or eveningness. This is an area that has been grossly overlooked in relation to mood and sleep.

Wu and Bunney [1990] reviewing studies of relapse following a period of sleep after improvement, theorised that sleep was depressogenic, They observed that depressed patients taking medication had a significantly lower tendency to relapse after a night of recovery sleep compared to patients not taking medication. They observed hypomanic symptoms in a significant number of patients the day after sleep deprivation. Wu and Bunney proposed that this modulation of emotion by the sleep-wake cycle may be mediated by a depressogenic process and possibly by a substance released during sleep and metabolised during wakefulness. It has to be acknowledged that if sleep is depressogenic, then depression, once induced, could become self-reinforcing.

More recently, Riemann et al. [1993, 1995] observed that total sleep deprivation exerts beneficial but transient effects on mood in approximately 60 percent of patients with a major depressive disorder. The positive effect of total sleep deprivation is generally reversed after the next night of sleep. Riemann et al. report that even short naps during the period of sleep deprivation are capable of re-inducing depressive mood in subjects responding to total sleep deprivation. Following a one-week phase-advance programme, increments in REM percentage were observed, in spite of clinical improvement [REM latency was still short]. This contradicts the assumption that REM sleep suppression is a necessary pre-requisite for antidepressive therapy. Riemann et al. conclude that there is a 'critical' phase in the morning hours during which sleep can induce depressive mood.

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In a number of the foregoing studies, methods were flawed or results were ambiguous. Therefore it is sometimes difficult to interpret the results. The natural course of depression is characterised by a tendency to remit spontaneously; therefore in studies of treatments it is important to control for the influence of spontaneous improvement on the results. In studies involving sleep-wake interventions for longer than one-week, it is possible that some improvement was due to spontaneous remissions.

In studies of treatments, such as sleep deprivation, it is important to control for the influence of patients' and experimenters' expectations on the results. Double-blind studies are usually used, but in the case of sleep deprivation, or phase-shifts of sleep periods, it is difficult for the subjects not to be aware of the nature of the intervention. In Vogel's study of REM sleep deprivation, subjects were aware of some intervention but were probably unaware of whether they were being deprived from REM or non-REM sleep.

Many of the above studies failed to use control treatments, because of the difficulty in finding a suitable placebo control. Therefore the results obtained could be explained partly by experimenter intervention. Perhaps the antidepressant response could be due to experimental bias expectations. However, it is possible to use any sleep intervention that appears to be inactive as a control. For example, Vogel used selective non-REM sleep deprivation as a control for selective REM sleep deprivation. Similarly, partial sleep deprivation in the first half of the night could be used as a control or placebo for partial sleep deprivation in the second half of the night, particularly since depressives are unlikely to have any expectations as to which treatment would be most effective.

Van den Burg et al. [1992] observed that in the typical depressive, total sleep deprivation produces a beneficial effect. They realised that most explanations were sought in biorhythmic desynchronisations basic to the depressive symptomatology - which were temporarily adjusted by sleep deprivation [Wehr and Wirz-Justice, 1981]. A second approach was based on depressogenic abnormalities per se [Wu and Bunney, 1990]. The concept of behavioural activation or arousal has also been associated with the mechanisms involved in the alleviation of depressive symptoms by total sleep deprivation.

According to the classical view, a continuum of arousal, ranging from deep sleep or coma on one extreme to panic-stricken terror or great excitement on the other, is a basic dimension determining acting and feeling [Duffy, 1962]. In line with this theory, Van den Burg and Van den Hoofdakker [1975] surmised that the depressive is in an unremitting state of 'inner tension' or 'over-arousal', and that total sleep deprivation brings this back to more acceptable levels (the 'over-arousal' hypothesis). In contrast, Post et al. [1976] proposed that the depressive was in a state of hypoarousal and was temporarily 'activated' and mobilised by the stress that total sleep deprivation entails. Post et al.'s hypothesis suggested that stress was causally related to an improvement in depression. As a measure of control, Post et al. informed subjects and raters that the purpose of the sleep deprivation was to assess changes in biochemistry. Mood effects were presented as incidental to the study. Despite this, he reported significant antidepressant effects.

The two arousal explanations differ on two important points. The first is whether total sleep deprivation serves as an activator or a de-activator, and the second is whether the depressed mental state is essentially one of hypo or hyper-arousal. In mild depression, tension and lack of energy tend to co-exist [Thayer, 1989]. This further suggests that a unidimensional concept of arousal is inadequate or insufficient in relation to depression. Sleep deprivation studies have illustrated the complexity of arousal [Broadbent, 1971]. While arousal theorists generally agree that total sleep deprivation is primarily de-arousing, with the potential consequence of some detrimental effects on behaviour, it is also acknowledged that sleep deprived subjects display signs of enhanced rather than low arousal in stressful situations [Eysenck, 1982]. On the basis of such findings, it is conceivable not only that the antidepressant effects of total sleep deprivation are achieved by its common de-arousing influence, possibly associated with a reduction in tension, but also that the effects are sought in an activating process triggered by the efforts of the depressed patient to fight fatigue.

In later studies Van den Burg and Van den Hoofdakker [1992] described the relationship between the antidepressant response to total sleep deprivation and changes in arousal using Thayer's AD ACL. They proposed that depression in arousal terms, was multidimensional, consisting of a 'tension' and a 'non-energy' aspect. They suggested that the hypoarousal explanation was inadequate because it failed to explain the reduction in felt tension and increment in calmness. In addition, the overarousal hypothesis was criticised for failing to explain the increase in subjective energy. To recap, Thayer's model of mood posits that energetic arousal

and tense arousal are positively correlated at low levels but negatively correlated at high levels. The explanation for this could lie in the notion that strong feelings of tension may inhibit feelings of energy while low levels of energy may easily lead to tension. However, it is possible that these two effects reinforce each other leading to a continuing and distressing state of deadlock. Van den Burg and Van den Hoofdakker suggested that when patients respond to total sleep deprivation it is through a process of 'deblocking' or 'disinhibition' - total sleep deprivation loosens the block in feeling and thinking. Wu and Bunney's [1990] findings of hypomanic symptoms in some depressives following total sleep deprivation was interpreted as an overshoot of the 'disinhibition' process. Wehr [1990] remarked that sleep deprivation improved states of inhibition and provoked states of excitement. In an earlier study, Wehr [1987] found that sleep reduction over extended periods produced symptoms of mania.

Van den Burg and Van den Hoofdakker [1992] asserted that tiredness and sleepiness were unrelated to the antidepressant response of total sleep deprivation. In their study they perceived that both responders and non responders indicated more tiredness after total sleep deprivation. This is important since it reflects fatigue of the brain [Horne, 1988]. It is suggested that fatigue plays a key role as a matrix on which the antidepressant effect may develop, whilst subjective fatigue during and after total sleep deprivation shows a monotonic increasing trend. There is also a pronounced 24 hour oscillation with a peak in the small hours and a trough in the late afternoon and early evening [Akerstedt, 1979]. Horne has argued that the circadian component is unrelated to cerebral fatigue.

The above review poses one question: why are the effects of one night's sleep deprivation so significant in depressed patients while not very striking in normal subjects ? Lader et al. [1987] suggested it was due to the 'instability' of the depressed 'brain state'. Gordjin [1990] proposed that the mechanism involved was related to the existence and instability of diurnal variations. Roy-Byrne et al. [1984] observed that patients who usually felt better in the evening were more likely to respond to total sleep deprivation. The relationship between diurnal variation of mood and response to total sleep deprivation was investigated in 131 depressed patients. Reinink et al. [1990] distinguished three types of diurnality: morning type, evening type and a non diurnal type. The results indicate that diurnality predicts the mood response to total sleep deprivation: evening types perceive total sleep deprivation more beneficial than morning or non diurnal types.

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The increased tiredness after total sleep deprivation suggests a cerebral basis for the mechanism involved in the antidepressant response to total sleep deprivation. It has been associated with the dampening of sub cortical arousal systems [Van den Burg and Van den Hoofdakker, 1992]. However, Horne argues that sleep loss has primarily a cortical impact - there is fatigue of the frontal lobes but not of the lower part of the brain that regulates sleep and wakefulness. Therefore the beneficial effect of total sleep deprivation is perhaps related to the reduction of over-inhibitory activity in these regions.

To summarise, total sleep deprivation appears to be simultaneously arousing (increased energy) and de-arousing (decreased tension) - this response occurring against a background of enhanced tiredness and sleepiness. Van den Burg and Van den Hoofdakker have argued that total sleep deprivation sets off a psychological process on the basis of cerebral fatigue. In particular, the prefrontal areas of the cerebral cortex are implicated, possibly in relation to the dampening down of sub cortical arousal systems.

Sleep and mood are clearly interrelated in depression. This is evident via the antidepressant effects of various techniques, such as total sleep deprivation, partial sleep deprivation, REM sleep deprivation and temporal shifts of the sleep period. The question of whether sleep disorders are part of the aetiology of depression remains unanswered. Many of the studies were flawed in that they lacked controls and placebo conditions. As a result, the reliability of findings emerging from uncontrolled studies is questionable. However, the few studies that used controlled balanced designs [e.g. Vogel, 1980; Sack, 1988] have yielded interesting results, which require replications on a larger scale. Still, clinical observation in general, polysomnographic studies and the observed antidepressant effects of sleep deprivation clearly indicate the intimate relationship between sleep and depression.

3.6 Mood changes in the Chronic Fatigue Syndrome.

Fatigue as a symptom is both vague and subjective, often accompanying a wide variety of diseases. It is generally regarded as abnormal exhaustion after performing normal activities, and is one of the most common symptoms in the community at large [Wessely, 1995]. In a British survey, 38 percent of a community sample reported substantial fatigue. In 18 percent, the fatigue had been present for over six months [Pawlikowska et al., 1994]. Fatigue is a phenomenon of concern to health care providers because of its implications for safety in the work setting

[Yoshitake, 1978] and because of its prevalence among persons with physical or emotional illness [Solberg, 1984; Krupp et al. 1989]. Fatigue alone may be associated with a degree of physical impairment comparable to that observed in some chronic disabling medical diseases [Kroenke et al., 1988].

Muncie [1941] first described a chronic fatigue syndrome characterised by a 'variable degree of fatiguability on effort, either mental or physical'. Various aetiologies, viral, endocrinal and emotional were discussed. Fatigue is a central feature of the chronic fatigue syndrome (CFS). Interestingly, the 'Wornout Syndrome' first described by Globus [1969] has many cardinal features that closely resemble the chronic fatigue syndrome, the primary symptom being undue fatigue.

A precise definition of fatigue is required. It has been variously described as malaise, lassitude, tiredness, exhaustion, weakness and myalgia. Osler [1910] described three types of fatigue:

> (i) 'weakness which is experienced by patients who have paralysis or paresis' - which is felt in the muscles of the body.
> (ii) 'general lassitude felt on slight exercise in states of exhaustion, in which slight movements cause palpitation, dyspnoea, perspiration, tremor and faintness' - more commonly indicated in acute viral infections.
> (iii) 'painful weariness' or 'feeling of being knocked out ' - associated with slight exercise in many nervous patients.

This 'feeling of being knocked out' was experienced primarily in the cognitive domain and referred to by Beard [1981] as neurasthenia. The term refers to all kinds of neurotic conditions supposedly originating from mental weakness and nervous over sensitivity. It was typically an affliction of young adults, usually women. Psychological symptoms of neurasthenia included lack of initiative and reduced capacity for mental effort. Many of the symptoms of neurasthenia were similar to those typical of depression.

From a psychiatric perspective, fatigue can be defined as a subjective state of weariness (related to reduced motivation, prolonged mental activity or boredom) that manifests in stressful situations. From a physiological perspective, fatigue can be defined as the end result of excessive energy consumption, depleted hormones or diminished ability of muscles to contract. Anaemia, infection, and impaired oxygenation deplete energy reserves by creating an unrelenting physical demand for

energy expenditure. Physical symptoms of neurasthenia include: lassitude, lack of certainty and precision in movements, loss of tone and body relaxation.

The CFS was more recently defined by Holmes et al.[1988]. It was characterised by severe disabling fatigue and symptoms of impaired concentration and short term memory, sleep disturbance and musculoskeletal pain. However, these symptoms are not specific for the CFS. Moreover, there are no pathognomic, clinical or laboratory findings to help substantiate the diagnosis. New guidelines were laid down by Fukuda et al. [1994]. It was proposed that prolonged fatigue be defined as lasting at least a month, and chronic fatigue as lasting more than six months. The patient could be classified as having CFS if established medical conditions had been ruled out. However, non psychotic psychiatric disorders, e.g. fibromyalgia, anxiety disorders, somatoform disorders or non-melancholic depression were not necessarily reasons for exclusion.

The CFS is also known as Myalgic Encephalomyelitis (ME), Postviral Fatigue Syndrome (PVFS), Akureyri Disease and 'Yuppie' flu. The various names have been used because of the perplexing nature of the illness and unknown aetiology. I have chosen to use the term CFS because it is an accurate clinical description but has no aetiological implications. The CFS is a chronic, persisting or relapsing illness that in some cases follows an acute viral infection and may occur sporadically or in epidemics. The main feature of the syndrome is overwhelming fatigue lasting six months or more. Characteristically, fatigue reduces the patient's pre morbid level of activity by 50 percent or more and is present despite adequate bed-rest. A variable degree of myalgia, especially following physical exertion is usually present and the majority of patients also report autonomic symptoms, e.g. excessive nocturnal sweating, intermittent low grade fever, hot flushes or feeling cold, fluctuations in body weight and changes in appetite or bowel habit.

Lawrie and Pelosi [1994] conducted a community survey in Edinburgh and recognised a prevalence rate of 0.56 percent for CFS. However, prevalence rates vary according to the criteria used. The prevalence of CFS in a Boston primary care clinic varied from 0.3 to 1 percent according to which of 3 national sets of diagnostic criteria was applied [Bates et al., 1993].

It should be recognised that a range of factors may be important in the development and maintenance of CFS. Given that exertion characteristically exacerbates the symptom of fatigue, it has been recommended that patients should limit their

physical activity [Spracklen, 1988; Dawes, 1991]. Others emphasise the dangers of avoiding activity because of the resulting diminished physical and psychological tolerance: as symptoms come to occur at progressively lower levels of exertion, the patients belief in the persistence of factors responsible for the illness is reinforced. This results in a vicious circle of avoidance behaviour, fatigue, helplessness and depression [Butler et al., 1991; Wessely et al., 1991].

Reviewing recent literature regarding symptoms most commonly reported by chronically fatigued patients, sleep disturbance was found to be a common complaint [Krupp et al., 1993; Morriss et al., 1993; Vercoulen et al., 1994; Farmer, 1995]. Sleep and fatigue characteristics were evaluated in 72 CFS patients and compared to 57 Multiple Sclerosis [MS] patients and 40 healthy controls [Krupp et al., 1993]. Chronically fatigued patients had significant elevations in both fatigue and sleep disturbance compared to the MS and healthy control groups. Polysomnography revealed sleep abnormalities, including sleep apnoea, periodic movement disorder, excessive daytime sleepiness and narcolepsy, in 62.5 percent of CFS patients.

In a similar study, Morriss et al. [1993] compared 12 CFS patients to 12 healthy controls. They found that CFS patients spent more time in bed than controls, but slept less efficiently, with longer sleep latencies after retiring. Seven patients with the CFS had a sleep disorder [four had difficulty maintaining sleep, one had difficulty getting to sleep, one had difficulty in both initiating and maintaining sleep, and one had hypersomnia]. The researchers concluded that most chronically fatigued patients had sleep disorders, which were likely to contribute to daytime fatigue. Around 80 percent of subjects seen in fatigue clinics by Farmer [1995] fulfilled criteria for sleep disorders. He concluded that primary sleep disorders are treatable, and are not excluded by a diagnosis of depression or CFS.

Presented here was a brief review of chronic fatigue syndrome. There is considerable overlap in the symptomatology of CFS and the wornout syndrome. Some clinical aspects of sleep disturbance in CFS [a commonly reported symptom] will be examined in some depth later in this thesis.

3.7 AIMS

A major aim of this thesis will be to examine the effects of sleep extension and sleep reduction on mood in an attempt to clarify the ambiguity surrounding this complex relationship between sleep and mood. In the light of early research into the association between sleep and mood [e.g. Globus, 1969; Taub, 1974] individual differences such as personality, morningness / eveningness and circadian rhythms will be examined in relation to mood and sleep. Since the most common complaint of sufferers of the chronic fatigue syndrome is intense fatigue and lethargy (symptoms commonly described as the 'wornout syndrome') I will examine the sleeping habits of this clinical group. More specifically, I will test the hypothesis that oversleeping may be causally related to sensations of intense fatigue upon awakening.

Further investigations into the *nature* of sleepiness will also be conducted in order to contribute to the growing debate on current definitions of sleepiness – what exactly do we mean when we say we are 'sleepy' ? Studies will be carried out to evaluate existing measures of subjective sleepiness, notably the Stanford Sleepiness Scale [Hoddes et al., 1973] in relation to the 'wornout syndrome' – do we say we are 'sleepy' when in fact we are actually 'tired'?

B

Investigations

4

Effects of Sleep Extension and Restriction on Mood

4.   Study 1

The Effects of Sleep Extension and Restriction on Mood.

A 'wornout syndrome' following oversleep was first described by Globus [1969]; persisting for up to 5 hours after awakening. It was most apparent when sleep exceeded 10 hours and was not replacing sleep loss. All subjects in Globus' study extended sleep ad-libitum. Globus differentiated between those individuals sleeping more than 10 hours and those sleeping less than 10 hours and examined whether subjects reported feeling 'wornout' or 'just great'. It was established that subjects sleeping 10 hours or more *with no sleep deficit* experienced the 'wornout syndrome' more frequently than feeling 'just great'. Although the data imply that too much sleep is detrimental to human functioning, one must enquire into the validity of using subjective reports based on individuals memory, since Globus' findings were based on retrospective accounts of mood states following oversleep.

Later studies executed in this field [e.g. Taub, 1973; Roehrs and Roth, 1989] have used various measures of mood and sleepiness and have generally yielded diverse findings. Taub [1979] discovered that a 2.7 hour sleep extension, beyond regular awakening times was unequivocally detrimental to subsequent daytime performance on tasks requiring sensorimotor and memory capacities. He asserted that the reduced performance and alertness following the ad-libitum lengthened sleep regime was not readily explained by the commonly accepted assumption that sleep is simply recuperation from fatigue. Taub maintained that beyond given temporal limits, sleep could become a potentially debilitating and fatiguing process and concluded that optimal levels of psychobiologic-behavioural states are highly contingent on a delicately maintained balance between wakefulness and sleep.

Taub and Berger [1974] assessed diurnal variation in mood using adjective checklists (ACL) three times a day. They established that positive moods were significantly elevated and negative moods significantly diminished at noon and late afternoon compared to morning. These trends corresponded to the pattern of increased efficiency in performance tests 4-8 hours after awakening. Taub and Berger found that certain mood states (activation, concentration, depression and fatigue) were affected to a greater extent by fluctuations in internal physiological processes relative to other moods (anxiety and hostility) which were affected by

environmental events. Although a simplified measure of mood was administered by Taub, the study overlooked mood changes in the first 4 hours following awakening; this, according to Globus, is the most critical in terms of the 'wornout syndrome'.

In an attempt to further our understanding of the relationship between sleep and mood, a preliminary study was designed to investigate subjective mood changes along several independent dimensions following oversleep and sleep restriction. The primary concern of this study was to examine the effects of oversleep in relation to the 'wornout syndrome'. It was hypothesised that following oversleep, individuals would experience a 'wornout syndrome' similar to that described by Globus [1969]. Whereas Globus' study assessed retrospective accounts of mood states, this investigation aimed to examine the individual's on-going mood state by hourly probing. Mood changes following both sleep extension and sleep reduction were compared to test the commonly held view that whilst sleep reduction is detrimental to mood, sleep extension is beneficial. This preliminary study therefore focused on mood states and prior sleep length.

Subjects

Participants were 10 healthy undergraduates, comprising of both sexes (6M, 4F) aged between 21-34. All subjects were normal sleepers routinely obtaining 7-8 hours of sleep per night, with no history of daytime sleepiness, habitual nap taking or treatment with CNS medication. All subjects agreed to adhere to the experimental conditions.

Design

The independent variable under investigation was sleep length. Subjects were required to undergo sleep extension and sleep restriction conditions, and measures of mood and daytime sleepiness (dependent variables) were recorded and compared to baseline values. A repeated measures design was employed: all 10 subjects underwent all the conditions; with the order counterbalanced.

Measures

The measures included: i). Sleep Diary

                            ii). Post-Sleep Questionnaire

                            iii). Mood Scale -Adapted POMS

                            iv). Stanford Sleepiness Scale

                            v). Actigraphy

                    — *see Appendix* —

i). Sleep Diary (completed daily during the assessment week, Phase 1).

This determined:- a. Sleep onset and wake-up times

                       b. Quality of sleep

                       c. A brief summary of the day's activities

ii). Post-Sleep Questionnaire

This was completed 30 minutes after arising in the morning and determined:-

                       a. Times of retiring and awakening

                       b. Quality of sleep

iii). Mood Scale - Adapted POMS

For the present study, it was decided (following extensive review of the major mood scales) that the Profile of Mood States was the most sensitive to the variables under investigation. Mc.Nair et al. [1971] claimed that each of the six dimensions of the POMS, namely; Fatigue, Vigor, Confusion, Tension, Anger and Depression were independent, orthogonal factors. Because the POMS uses a large number of highly specific mood descriptions, it offers considerable advantage over the other global rating scales. In addition, it has been extensively revised and updated and has been shown to be valid and reliable in numerous situations. The POMS was thus deemed a purer scale compared to many other measures of mood currently used in research. Importantly, it was found that several items used to describe the 'wornout syndrome' were the same or similar to items on the POMS "Fatigue" dimension. The POMS scale was modified for this study since the factors most sensitive to the 'wornout' syndrome were classed under the POMS dimensions of fatigue, vigor and confusion. By adapting the scale, to probe only the relevant dimensions, the scale could be shortened and therefore administered at regular intervals throughout

the experimental day. This was important because the nature of the study required one to monitor mood changes at frequent intervals, particularly during the first four hours after awakening. Though the Tension scale was included as a control variable (baseline for the suggestion-compliance effect), it was not relevant to the 'wornout syndrome' and changes in tension were not expected following experimental sleep manipulations. The selection of particular variables was based on the highest factor loading for each dimension in order to utilise those descriptors with very little overlap with other dimensions. The following items were selected from the POMS scale:

Table 4.1 Adapted POMS (after Mc.Nair et al., 1971).

Dimension	Factors
Fatigue	Wornout
	Fatigued
Vigor	Alert
	Energetic
Confusion	Confused
	Unable to Concentrate
Tension	Nervous
	On-Edge

iv). Stanford Sleepiness Scale (SSS).

The Stanford Sleepiness Scale (developed by Hoddes et al., 1973) is one of the most commonly used self-rating sleepiness scales. The SSS measures sleepiness on a 7-point ranked scale from:

1. Active, Vital, Alert and Wide Awake

to

7. Almost Unable To Stay Awake, Struggling To Remain Awake

Subjects were asked to place a mark next to the scale value which best describe their levels of sleepiness.

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v). Actigraphy

Ambulatory activity monitors were worn on the wrist [Type: Z80-32K V1; available from: Gaehwiler Electronic, Eichtalstrasse 20, CH-8634 Hombrechtikon]. These actometers detected the average bodily movement in one-minute epochs during periods of sleep and wakefulness, and thus provided an objective method of monitoring sleep-wake patterns. Actometers were used because the primary purpose of the study was to assess changes in daytime function associated with extended time in bed, and not to document changes in sleep physiology, occurring during extension. Actometers predicted polysomnographically determined sleep or wake with 93 percent accuracy [Levine et. al., 1986] and were deemed to be sensitive to the effects of restriction and extension of time in bed [see Levine et al., 1988].

Procedure

The experimental procedure consisted of two phases: in phase one, subjects were monitored to provide normative sleep data. Phase two was the experimental phase; sleep was increased by 2 hours under the condition of sleep extension and reduced by 2 hours under sleep reduction. The sleep scheduling in phase two was based on each individuals normative (baseline) sleep. For example, under sleep extension, an individual who normally arises at 7.00am would sleep on until 9.00am, whilst another individual who habitually wakes at 8.00am would continue sleeping until 10.00am. Under the Sleep Extension condition, it was decided to ask subjects to sleep on in the morning rather than go to bed earlier in the evening. One reason for this was that most subjects were unable to go to sleep earlier in the evening due to factors such as noise in corridors. In addition, most excess sleep is attained in the form of a lie-in which characteristically involves sleeping on later than usual in the morning rather than retiring earlier. This pattern is particularly evident at weekends.

Phase One

Subjects were asked to keep a sleep diary for a one-week period. This was to determine their usual sleep-wake pattern, from which the duration of sleep on the experimental nights in phase two were calculated (+/- 2 hours from normative sleep). Wrist actometers were worn throughout the week to further monitor and verify the sleep diary information.

Phase Two

During the second phase, subjects were required to extend their normal sleep time by two hours (retiring at the usual bedtime and sleeping late into the morning). On another occasion (separated by an interval of one-week) they were required to reduce their normal sleep time by two hours (waking up earlier in the morning). Two baseline nights (again with an interval of one-week) were also monitored. A repeated measures design was employed, all subjects underwent all conditions with the order counterbalanced. For example, subject 1 complied with the following schedule: Baseline 1, Extension, Reduction, Baseline 2, whilst subject 2 had the following schedule: Reduction, Baseline 1, Baseline 2, Extension.

Upon arising, and at intervals throughout the day, subjects were required to complete the following questionnaires:-

1. Post-Sleep Questionnaire — 30 minutes after arising
2. Stanford Sleepiness Scale — 30 minutes after arising
3. Mood Questionnaire — hourly throughout the day

Results

As the conditions were counterbalanced, data was examined for any order effects or transfer effects between conditions, despite the one-week gap between conditions. There was no evidence of any order effects, and the data was pooled for analysis.

<u>**Results are to be presented thus:**</u>

1. Post-Sleep Questionnaire: (Baseline, Extension, Reduction) — pages 47-50
2. Mood Questionnaire: (Fatigue, Vigor, Confusion)
   A. Comparison of Baseline, Extension and Reduction scores for all the subjects together — pages 51-53
   B. Sleep Extension — pages 53-58
   C. Sleep Reduction — pages 59-60
3. Stanford Sleepiness Scale: (Baseline, Extension, Reduction) — pages 61-62

1. Post-Sleep Questionnaire

The Post-Sleep Questionnaire was administered to monitor subjects sleep onset and awakening times as well as to ensure that there were no reported sleep problems. Table 4.2a displays the mean sleep onset and awakening times across all conditions for the 10 subjects.

Table 4.2a Mean [with se] Sleep Onset and Awakening Times: reports completed 30 minutes after arising.

	Baseline (normative)	Extension (+2hrs)	Reduction (-2hrs)
Sleep Onset	0019h [0.23]	0009h [0.21]	0021h [0.16]
Time of Awakening	0726h [0.13]	0914h [0.11]	0511h [0.12]

All 10 subjects normally averaged 7 hrs and 7 mins of sleep per night. On experimental days the average time of awakening for sleep extension was 0914h and for sleep reduction, 0511h. The variance [se] for both sleep onset and time of awakening were comparable across all 3 conditions.

The Post-Sleep Questionnaire also probed subjective sleep evaluations, with responses gauged on a 5-point scale. One-way Analyses of Variance were computed for each of the 4 questionnaire items enquiring into: 1. Difficulty getting to sleep, 2. Restlessness during sleep, 3. Quality of sleep, and 4. Difficulty getting up. Significant findings were further subjected to Post-hoc analysis using Tukeys HSD Test.

There was a significant difference between all conditions with respect to restlessness during sleep, $F = 4.05$ [2, 27] $p < 0.05$. Post-hoc analysis showed that these differences were principally between baseline and sleep reduction ($p < 0.02$); subjects reported feeling significantly more restless after sleep reduction compared to a night of baseline sleep [see fig 4.1].

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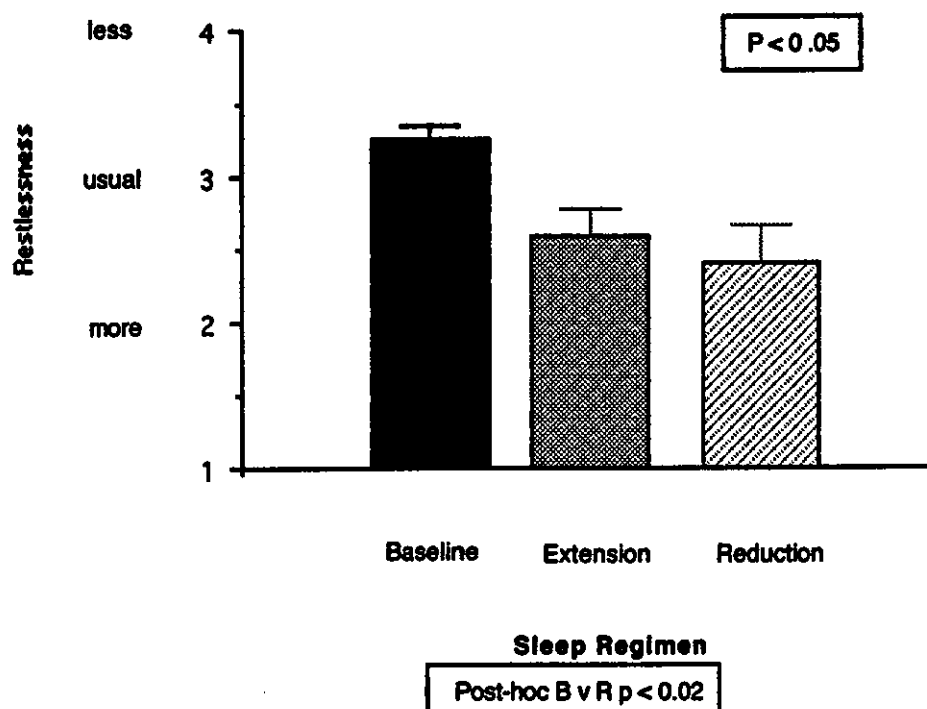
Fig. 4.1 Restlessness During Sleep

A significant difference was observed in subjective reports of 'difficulty in getting up' between the conditions, F = 5.09 [2, 27] p < 0.05. Post hoc analysis revealed these differences to be principally between extension and reduction (p < 0.01). Subjects found it more difficult to get up after a night of curtailed sleep. Compared to baseline, subjects reported less difficulty getting up after a night of Sleep Extension [see fig 4.2].
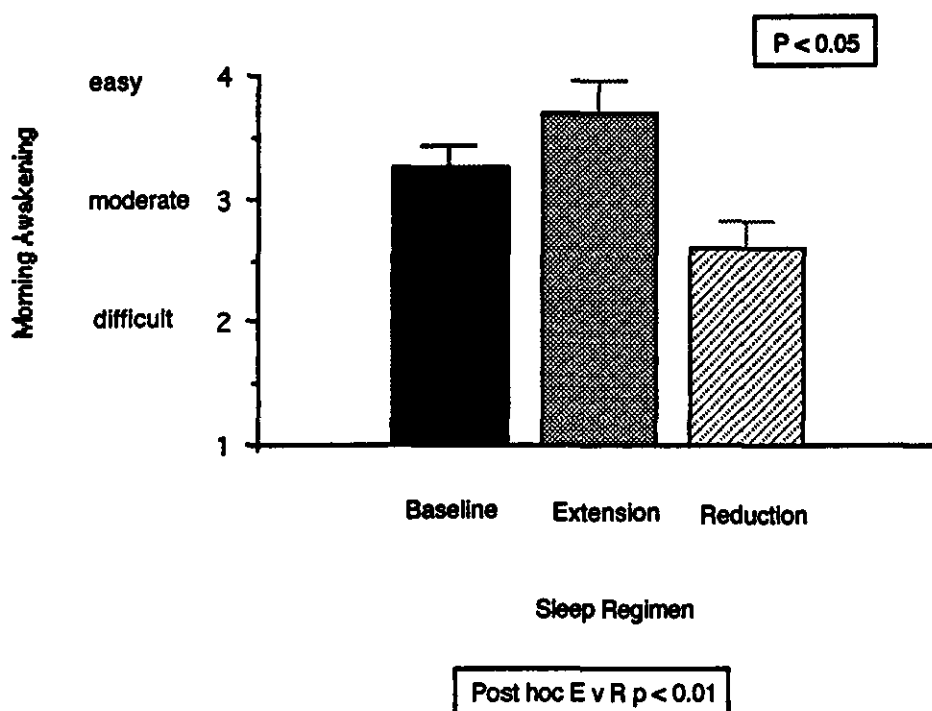
Fig. 4.2 Difficulty in Getting Up.

Table 4.2b. Mean [with se] ratings of difficulty getting to sleep.

Five-point Scale	Baseline	Extension	Reduction	1-way ANOVA
Difficulty in getting to sleep 2=Harder than usual 3=Same as usual 4=Easier than usual	3.06 [0.19]	2.8 [0.2]	2.6 [0.31]	p = 0.66 F = 0.43 (2, 27)

Table 4.2c. Mean [with se] ratings of sleep quality.

Five-point Scale	Baseline	Extension	Reduction	1-way ANOVA
Quality of sleep obtained 2=Better than normal 3=Normal 4=Worse than normal	2.94 [0.17]	2.9 [0.18]	3.0 [0.15]	p = 0.89 F = 0.11 (2, 27)

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When asked about difficulty getting to sleep, subjective responses were similar across all 3 conditions [see Table 4.2b]. This was encouraging, as sleep onset was not a confound. At each of the 3 experimental phases, subjects fell asleep with the same ease. It was clear that there were no significant differences in subjective 'quality of sleep' after baseline, sleep extension or sleep reduction. [see Table 4.2c].

2. Mood Analysis

Mood scores were obtained for the following dimensions: Fatigue, Vigor and Confusion. Two-way ANOVA's were performed separately for each mood dimension comparing the scores after Baseline, Sleep Extension and Sleep Reduction for all subjects together. The Between-Group factor was sleep length [3 levels] and the Within-Group factor was time [14 hours].

Fatigue scores (change from baseline) following Sleep Extension were examined hourly and those subjects with positive fatigue scores (> 0) for the first 4 hours were classified as experiencing the 'wornout syndrome'. Four subjects were placed in this category.

Two-way ANOVA's were performed separately for Sleep Extension and Sleep Reduction, with the 'wornout syndrome' [WOS] / 'non-wornout syndrome' [NWOS] as the between group factor and time as the within group factor. These analyses were conducted for each of the three mood variables: Fatigue, Vigor and Confusion. Significant findings were further analysed with a Post-hoc test using Tukeys H.S.D.

The results were examined in terms of 'hours after arising' rather than 'clock time'. This was done because actual mood scores [e.g. Fatigue, Vigor and Confusion] were subjected to analysis using the change from baseline values rather than actual scores. This allowed comparisons to be made across conditions. However, because subjects in the Sleep Reduction condition arose 2 hours before Baseline, no comparative Baseline data was available for the first 2 hours: as a result this data was superfluous when comparisons were made according to real time [clock time]. Moreover, comparing data as 'hours after arising' meant that all the data were incorporated into the analysis.

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A. Sleep Length and Mood

a) Fatigue

A comparison of fatigue scores across Baseline, Sleep Extension and Sleep Reduction yielded non-significant findings, $F = 1.63$ [2, 27] $p = 0.21$. However, interactions between sleep length and time were highly significant, $F = 3.02$ [26, 351] $p < 0.0001$. Figure 4.3 illustrates the interactions.
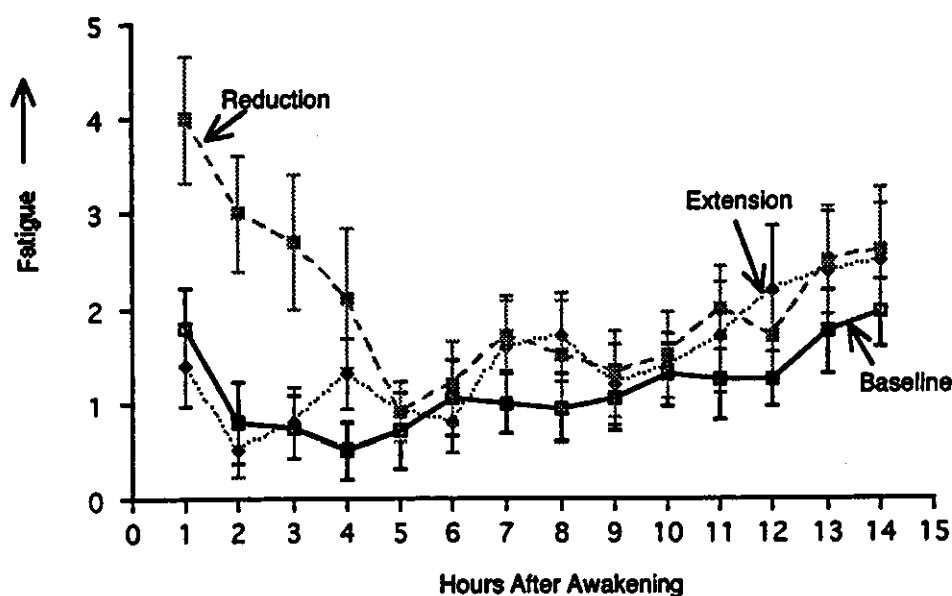
Fig 4.3   Fatigue scores after Baseline, Sleep Extension and Sleep Reduction.

Post hoc analyses revealed significant interactions at hours 1, 2 and 3, $p < 0.05$. Following Sleep Reduction, subjects were significantly more fatigued than either Baseline or Sleep Extension conditions, for the first 3 hours after awakening. Thereafter, fatigue scores were comparable in all 3 groups. Fatigue after Sleep Extension was similar to Baseline throughout the day.

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b) Vigor

All 3 groups described comparable levels of Vigor, $F = 0.17$ [2, 27] $p = 0.84$. Significant interactions were found between sleep length x Time, $F = 3.3$ [26, 351] $p < 0.0001$. Fig 4.4 illustrates the findings, following Sleep Reduction subjects were significantly less energetic than either Baseline or Sleep Extension, for the first 2 hours after awakening. Post-hoc analyses revealed significance at hours 1 and 2, $p < 0.05$.
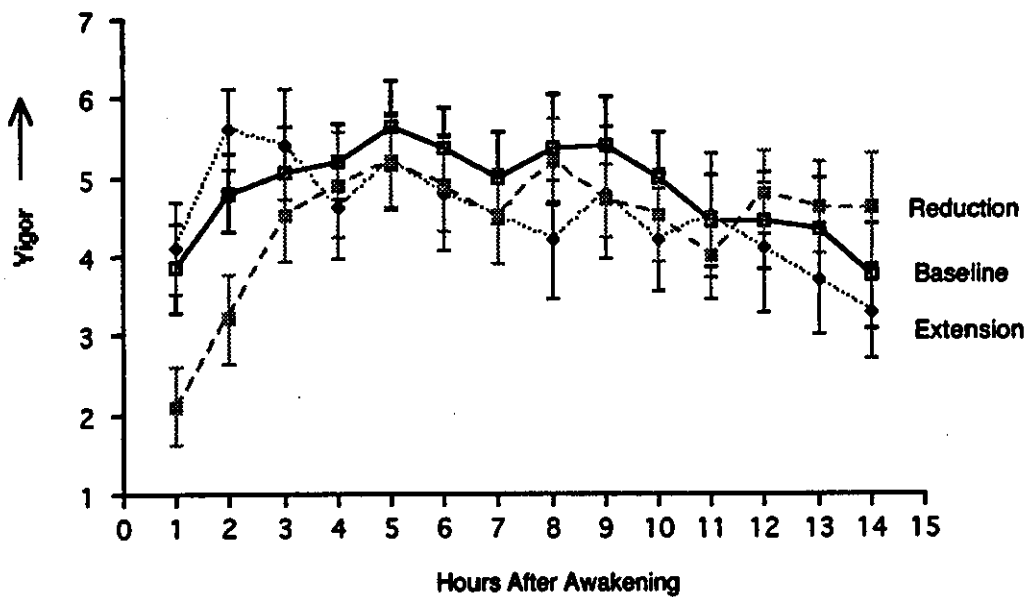
Fig 4.4   Vigor scores after Baseline, Sleep Extension and Sleep Reduction.

c) Confusion

A comparison of confusional state across conditions revealed interesting trends, $F = 2.65$ [2, 27] $p = 0.09$. Although not significant, subjects revealed enhanced confusion following Sleep Reduction. Interactions between sleep length x Time revealed significant findings, $F = 1.7$ [26, 351] $p = 0.02$. Fig. 4.5 highlights the interactions. Post-hoc analyses indicated significant interactions 2, 3 and 11 hours after arising, $p < 0.05$.
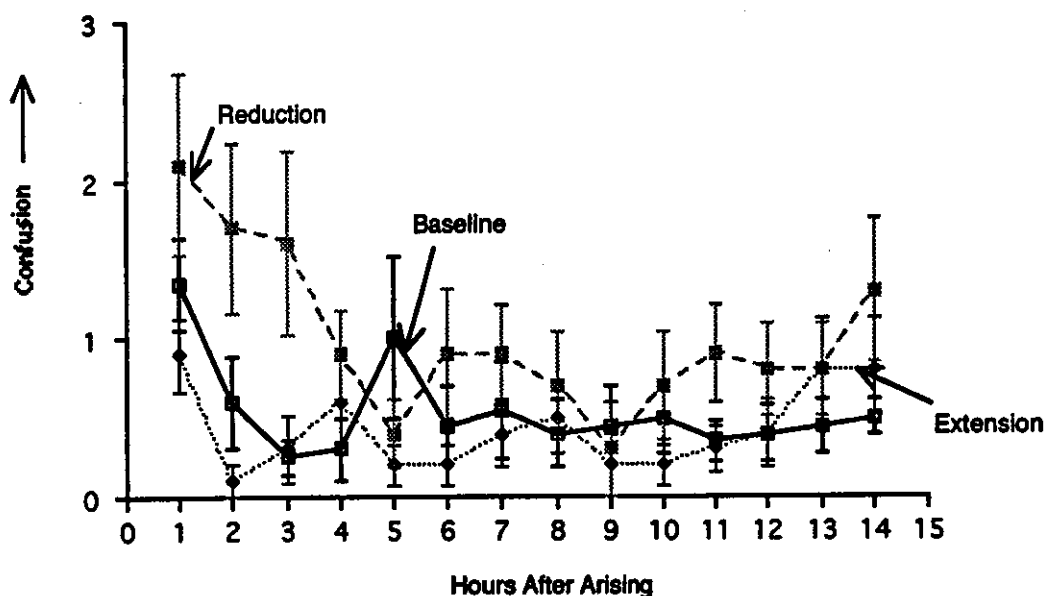
Fig 4.3 . Confusion scores after Baseline, Sleep Extension and Sleep Reduction.

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B. Sleep Extension

a) Fatigue

Main Effects

There was a significant difference in fatigue scores between subjects exhibiting the WOS [n=4] and the NWOS [n=6], $F = 7.94$ [1, 6] $p = 0.03$. Figure 4.6 highlights the divergent trends. Subjects in the WOS group reported significantly more fatigue than those in the NWOS group. These results were not surprising since subjects were categorised as being 'wornout' on the basis of reporting positive fatigue scores (> 0) for the first 4 hours after arising. Due to the small sample size, results have to be interpreted with caution. But clear trends are evident.
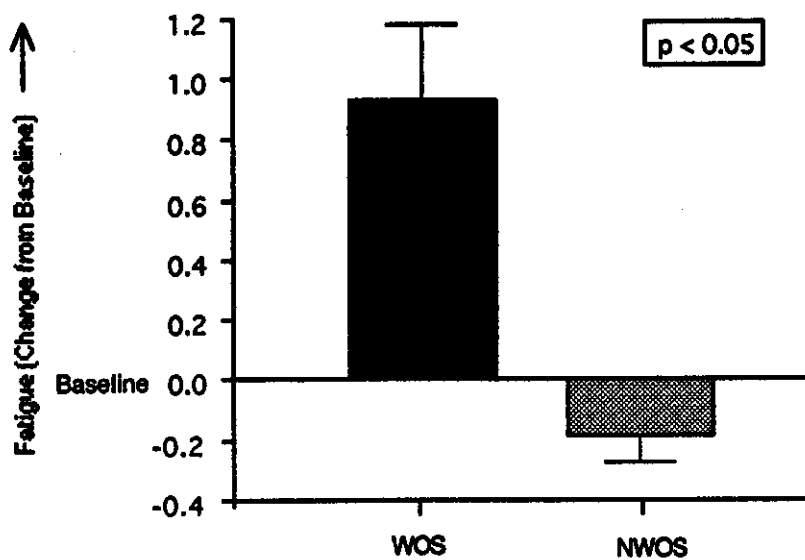
Fig. 4.6 Fatigue after Sleep Extension.

Interactions
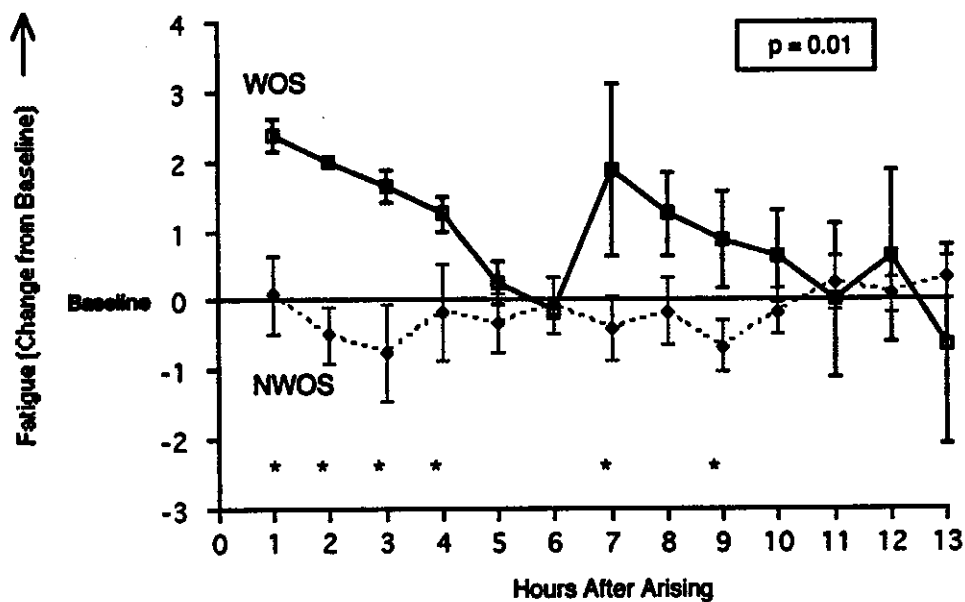
**Fig. 4.4 Fatigue after Sleep Extension in WOS [n=4]
and NWOS [n=6] groups.**

A two-way ANOVA revealed significant differences between the WOS and NWOS groups after Sleep Extension, $F = 2.4$ [12, 72] $p = 0.01$. Post-hoc tests revealed significant differences at the $p = 0.01$ level for the times asterisked in the graph. Figure 4.7 indicates that the NWOS group were more stable — there was less fluctuation in fatigue throughout the day. There was greater variation in fatigue in the WOS group.

b) Vigor

Main Effects
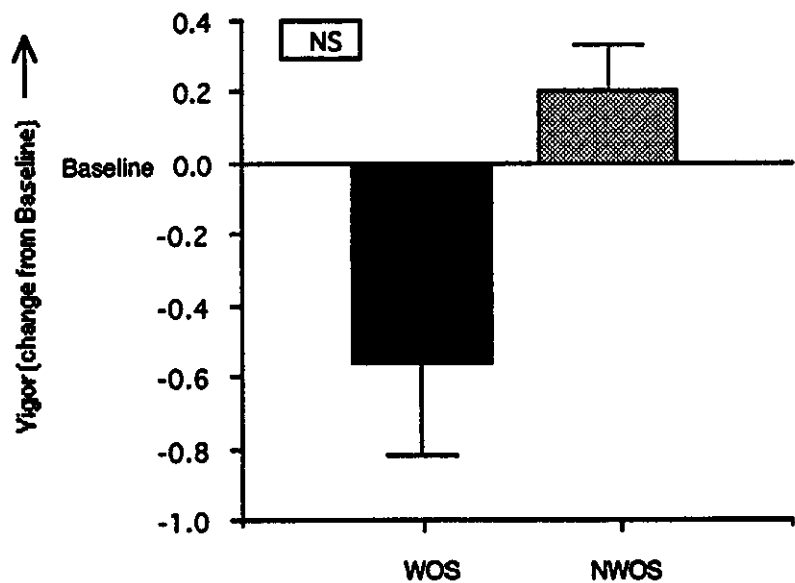
Fig. 4.8 Vigor after Sleep Extension.

There were no significant findings for vigor comparing the symptomatic WOS and NWOS groups, $F = 2.52$ [1, 6] $p = 0.17$. Nevertheless a trend emerged: subjects in the WOS group reported diminished vigor compared to the NWOS group, who by contrast, described slight elevations in vigor following sleep extension [see fig. 4.8].

Within-Factor: Time

Figure 4.9 illustrates fluctuations in vigor in all 10 subjects, $F = 2.38$ [12, 72] $p = 0.01$. One hour after awakening, vigor was below baseline, increasing sharply two hours after awakening but then declined rapidly to below baseline four hours after awakening. The first four hours are most crucial to the 'wornout syndrome'.

Post-hoc tests revealed significant differences in vigor between the 1st and 12th hour after awakening.
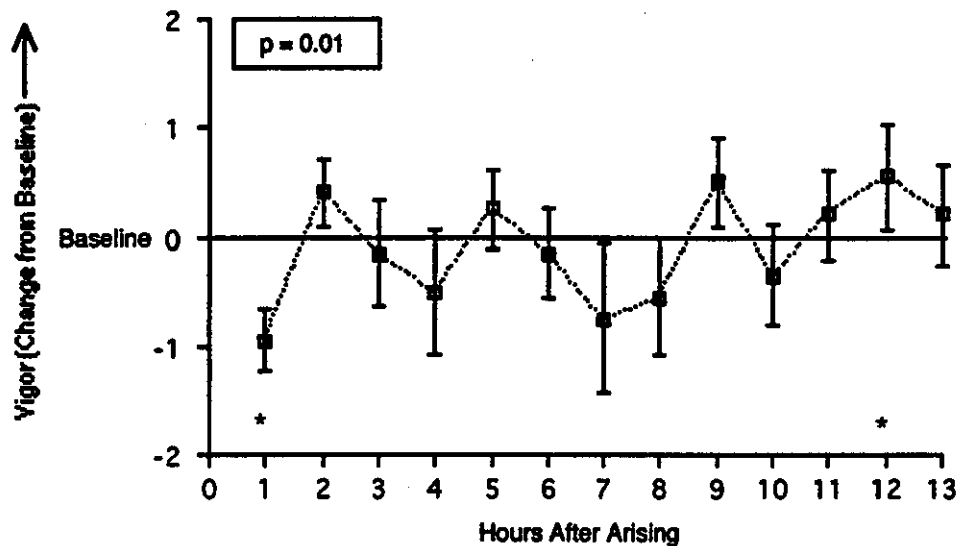
Fig. 4.9 Changes in Vigor after Sleep Extension.
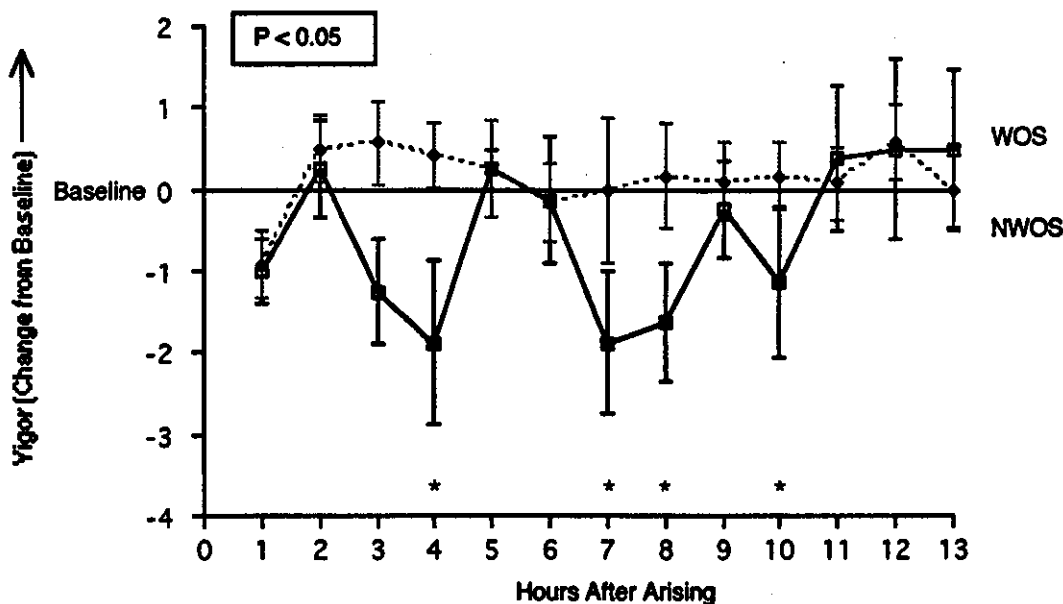
Interactions

Fig. 4.10 Vigor in WOS and NWOS groups after Sleep Extension.

Interactions between WOS x Time were significant, $F = 2.02$ [12, 72] $p = 0.03$. Figure 4.10 illustrates vigor in the WOS and NWOS groups over time. A post-hoc test revealed significant differences between the groups at the times asterisked in the

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graph. Following Sleep Extension, there was greater variation in vigor over time in the WOS group, compared to the NWOS group. Vigor was very similar in both groups for the first 2 hours after awakening; the WOS group showed a rapid decline by hour 4, followed by a return to baseline 5 hours after arising. There was another sharp dip in vigor in the WOS group 7 hours after arising.

c) Confusion

Main Effects

Although no significant differences emerged between the groups, $F = 2.81$ [1, 6] $p = 0.14$ the two-way ANOVA distinctly indicate trends suggesting that the WOS group were more confused than the NWOS group after Sleep Extension.

Within-Factor: Time

There were significant changes to confusion ratings in the first 5 hours, $F = 1.88$ [12, 72] $p = 0.05$. There was a sharp rise in confusion 4 hours after arising. Post hoc tests indicated significant differences in confusion between the 1st hour after awakening and 2, 3 and 5 hours after awakening.
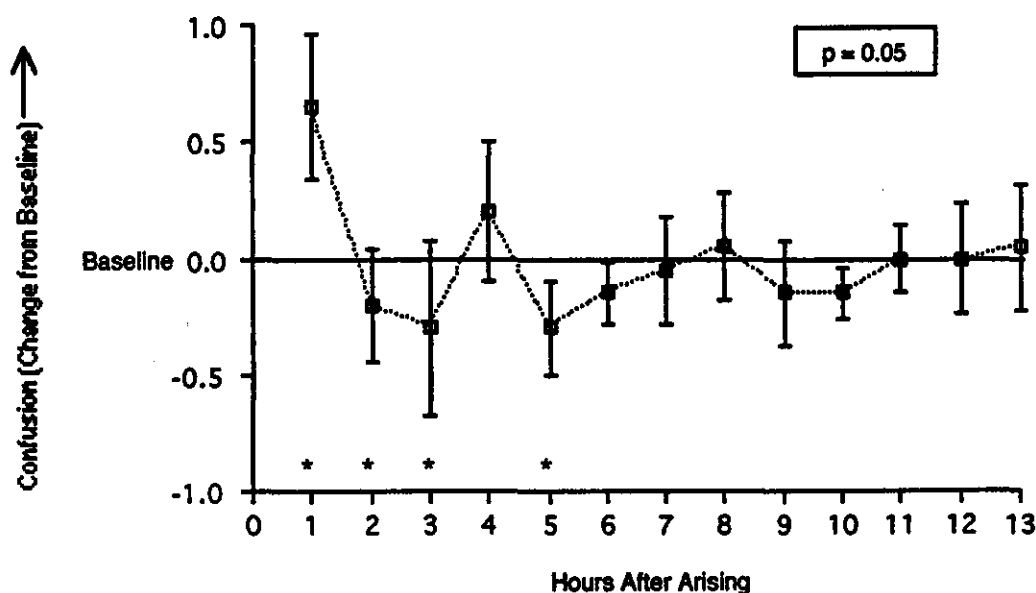
Fig. 4.11 Changes in Confusion after Sleep Extension.
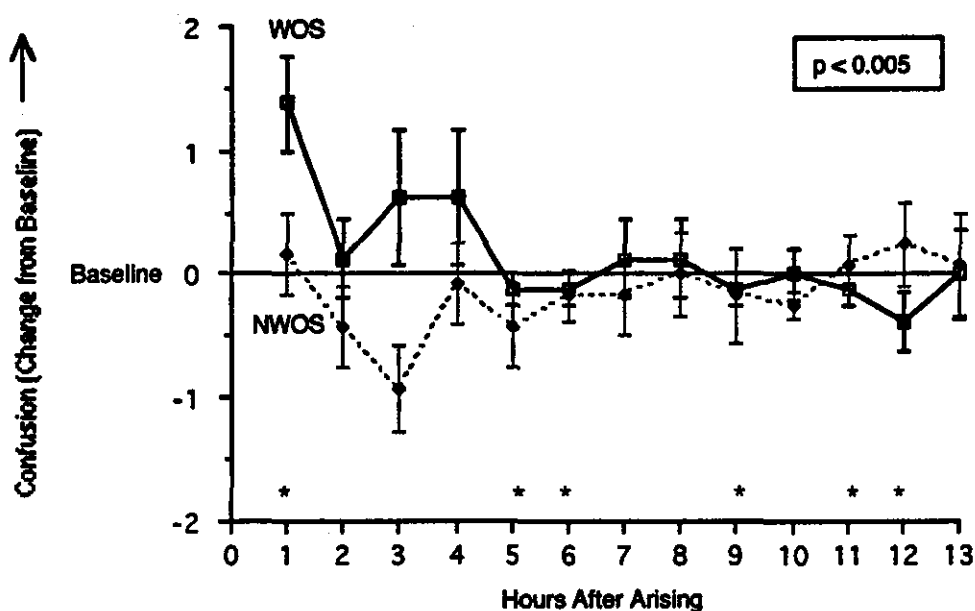
Fig. 4.12 Confusion after Sleep Extension.

The interaction between N/WOS x Time was significant, F = 2.76 [12, 72]
p = 0.004. Post-hoc tests revealed significant differences in both groups: 1 hour
after arising subjects in the WOS group were significantly more confused than at 5,
6, 9, 11 and 12 hours after arising (as asterisked in fig. 4.12). Again, confusion in
the WOS group 1 hour after arising was significantly different from the NWOS
group for all 13 hours after arising. In addition, 3 and 4 hours after arising, the
WOS group were more confused than the NWOS group 3 hours after arising. There
were clear differences between the groups for the first 4 hours after arising, the
WOS group were more confused. However after 4 hours, confusion in both groups
was comparable.

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To summarise, the WOS group were more fatigued after Sleep Extension compared
to the NWOS group. These differences were particularly evident for the first 4
hours after arising, but the adverse effects of oversleeping continued throughout the
day. In terms of vigor, the WOS group were significantly less energetic than the
NWOS group. The WOS group were also more confused than the NWOS group,
particularly during the first 4 hours after arising.

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C. Sleep Reduction

a) Fatigue

Main Effects
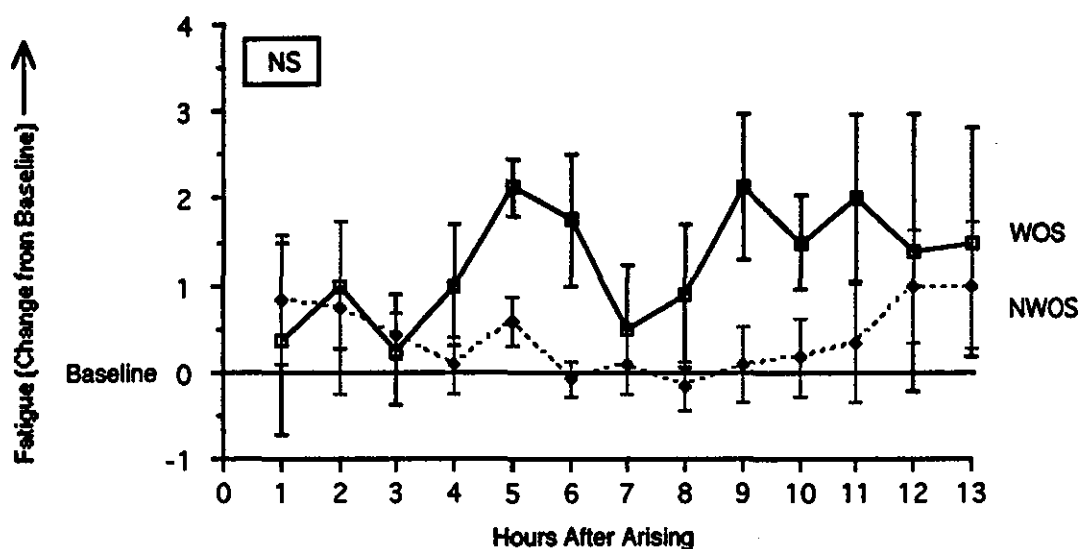
Fig.4.13 Fatigue in WOS and NWOS groups after Sleep Reduction.

Following Sleep Reduction, there were no significant differences in fatigue between the WOS and NWOS groups, $F = 3.45$ [1, 6] $p = 0.11$. Both groups reported increased fatigue relative to baseline. However, subjects in the WOS group described slightly greater fatigue. Similar levels of fatigue were observed in both groups three hours after arising. Fatigue scores then diverged: those in the WOS group remained more fatigued and indicated greater diurnal variation than those in the NWOS group, whose levels of fatigue were maintained close to baseline [see fig. 4.13]. These trends were not statistically significant.

b) Vigor

Following Sleep Reduction, both WOS and NWOS groups documented similar levels of diminished vigor relative to baseline. The main effects were not significant, $F = 0.01$ [1, 6] $p = 0.93$.

c) Confusion

Main Effects

There was a significant difference between the groups, $F = 7.71$ [1, 6] $p = 0.03$: the WOS group were in a greater confusional state following a night of Sleep Reduction than the NWOS group.
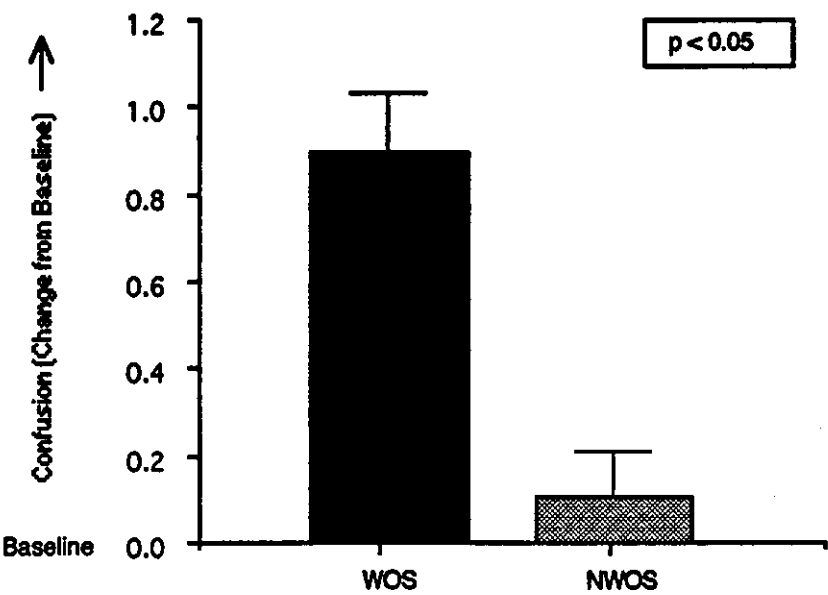
Fig. 4.14 Confusion in WOS and NWOS groups after Sleep Reduction.

There were no significant interactions between WOS / NWOS x Time, $F = 1.28$ [12, 72] $p = 0.25$.

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To summarise, both the WOS and NWOS groups complained of increased Fatigue and diminished Vigor after Sleep Reduction. However, subjects in the WOS group were in a greater confusional state than those in the NWOS group. Only the confusional state showed significance between the WOS / NWOS groups. Sleep reduction did not result in significant differences in either fatigue or vigor between the two groups or show any interaction with time.

3. Stanford Sleepiness Scale (SSS)

Subjects completed the SSS thirty minutes after arising. Symptomatic subjects were compared with subjects in the NWOS group after each of the 3 sleep conditions. Results were analysed using unpaired t-tests; comparing subjects with the WOS [n=4] following oversleep to the NWOS group [n=6].

Table 4.3a   Baseline SSS scores (mean & se)

Wornout Syndrome Group Mean	No Wornout Syndrome Group Mean
3.5 (.29)	2.8 (.31)
t = 1.49 [df. 8] p = .17	

SSS
2 = Functioning at a high level, not at peak, but able to concentrate
3 = Relaxed, awake, not at full alertness, responsive
4 = A little foggy, not at peak, let down
5 = Fogginess, starting to lose interest in remaining awake, slowed down

Both groups reported similar levels of sleepiness following the baseline sleep condition. These values represent their general state of sleepiness upon arising after a normal night's sleep, prior to any experimental manipulation. The symptomatic group cannot be differentiated from controls after obtaining normal sleep.

Table 4.3b   Sleep Extension SSS scores (mean & se)

Wornout Syndrome Group Mean	No Wornout Syndrome Group Mean
5.0 (.41)	3.3 (.49)
t = 2.39 [df.8] p = .04 *	

Following sleep extension, subjects in the WOS group were significantly more 'sleepy' upon awakening. Subjects in the NWOS group indicated sleepiness ratings similar to their baseline sleep scores [see fig. 4.15].

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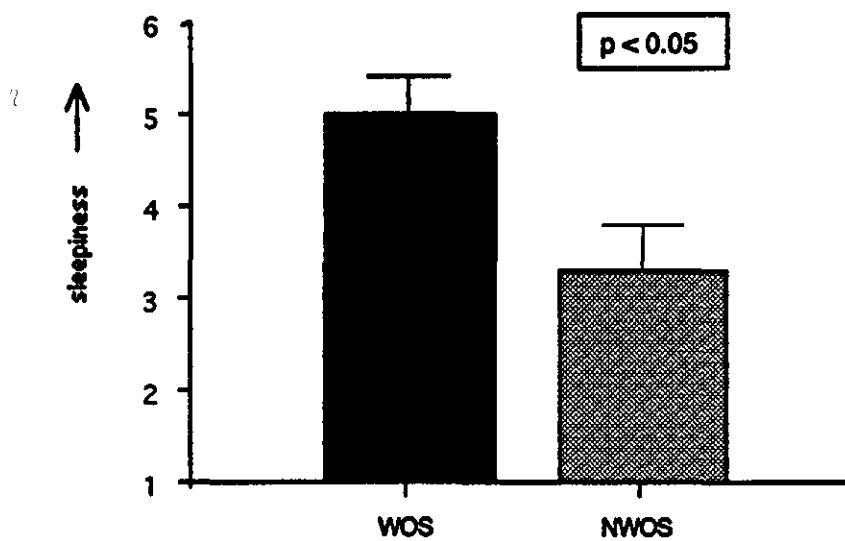
Fig. 4.15 Sleepiness after Sleep Extension.

Table 4.3c Sleep Reduction SSS scores (mean & se)

Wornout Syndrome Group Mean	No Wornout Syndrome Group Mean
4.5 (.5)	4.3 (.56)
t = .21 [df.8] p = .84	

After Sleep Reduction, subjects in both the WOS and NWOS groups reported a similar increase in sleepiness, relative to baseline. No statistical differences emerged.

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Sleepiness was measured by the SSS 30 minutes after arising following Baseline, Sleep Extension and Sleep Reduction regimens. Sleepiness remained comparable in both groups after baseline sleep. However, significant differences emerged after Sleep Extension [see fig. 4.15]. Subjects in the WOS group were more 'sleepy' than subjects in the NWOS group. Indeed, comparing their scores to baseline, sleepiness in the WOS group following Sleep Extension far exceeded that after baseline. Following Sleep Reduction, SSS scores were almost identical for both the WOS and NWOS groups. However, it was elevated compared to baseline. It was evident that subjects in the WOS group were marginally more sleepy after Sleep Extension than after Sleep Reduction. This was in contrast to subjects in the NWOS group, whose sleepiness after Sleep Reduction [mean = 4.3] surpassed that after Sleep Extension [mean = 3.3].

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Discussion

The Post-Sleep Questionnaire probed subjective restlessness during sleep, subjects undergoing Sleep Reduction were significantly more restless, compared to Baseline. One explanation for this increase in restlessness during a night of restricted sleep may be anxiety about arising earlier than normal; perhaps subjects expected to feel tired as a result of less sleep and this may have inadvertently caused restlessness throughout the sleep period.

A comparison of mood scores following Baseline, Sleep Extension and Sleep Reduction indicated that subjects were adversely affected by Sleep Reduction. This was reflected by accounts of enhanced fatigue and confusion, in addition to a decrement in vigor, for the first 2-3 hours after awakening [see Figs. 4.3 to 4.5]. Following Sleep Extension, subjective mood scores were comparable to Baseline.

Following this initial analysis, subjects were categorised according to their fatigue scores. Those subjects reporting enhanced fatigue [for the first 4 hours] relative to Baseline, following Sleep Extension were identified as the WOS group. Their mood scores were compared to the remaining subjects, the NWOS group. Following Sleep Extension certain trends and significant findings emerged. The adverse effects of oversleeping were particularly pronounced in the first four hours after arising. Subjects in the WOS group reported increased fatigue and confusion. There was a substantial decline in Vigor.

Graphs illustrating mood changes [see Figs. 4.6 to 4.14] indicated that both Sleep Extension and Restriction groups deviate from baseline. However both groups appeared to follow similar paths, to some extent. This suggests that it is not changes in sleep length per se which affected mood. Rather, mood variation is more attributable to the inherent sleep disruption (from normal routine). This conclusion has been made following investigations of subjective mood states after both sleep extension and restriction: both conditions resulted in altered mood states. One interesting finding was that following Sleep Reduction, both the WOS and NWOS groups responded similarly in terms of reporting elevated fatigue and diminished vigor. However, following Sleep Extension, only those subjects in the WOS group described enhanced fatigue and decreased vigor. Comparisons between the WOS and NWOS groups of confusional state revealed similar findings after Sleep Extension and Sleep Reduction: in both conditions, subjects in the WOS group were more confused.

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Results of this preliminary study suggested that some subjects experienced the 'wornout syndrome'; in these individuals oversleeping was found to be just as detrimental to mood as a comparable reduction in sleep length. This preliminary study provided evidence to support Globus' [1969] theory of a wornout syndrome following oversleep. Four subjects were identified as experiencing the adverse effects of oversleep upon mood.

The pilot study generated further questions and complexities; since four subjects were adversely affected following oversleep, the question arose as to the significance of individual differences, e.g. personality and its effect upon the relationship between sleep duration and mood. To resolve some of the issues raised by the pilot study, a more detailed investigation was designed; individual traits and biological rhythms were also considered. Those modulating factors to be incorporated into the following study are listed below:

[1] Personality variables
[2] Circadian typology into morning and evening types
[3] Effects of circadian rhythms
[4] Diurnal variation in mood
[5] Sleep habits: regular vs. irregular sleepers

A more comprehensive mood questionnaire was constructed [still based on the POMS], to incorporate additional dimensions of mood. In addition, it was decided to further investigate the area of subjective sleepiness and the multidimensionality of measures of sleepiness, particularly the SSS.

5

Further Investigations into the Effects of Sleep Length on Mood

5. Study 2

Further Investigations into the Effects of Sleep Length on Mood.

Tiredness vs. Sleepiness

Sleepiness is a transitional phenomenon, bridging the gap between wakefulness and sleep. It reflects a propensity or pressure to sleep. Sleepiness may also represent a physiological need state, represented by lowered cortical arousal, associated with alpha and theta activity, as measured by electroencephalography. Carskadon and Dement [1982] distinguished between physiological sleepiness, which reflects an underlying sleep propensity that surfaces if subjects are placed under quiet restful conditions, and manifest sleepiness, which is mediated by situational factors, e.g. boredom. They developed this classification to explain the multiple sleep latency test [MSLT], an objective test used to gauge levels of daytime sleepiness. Sleepiness must be differentiated from tiredness, which may be viewed as fatigue or disinclination for physical exertion.

The SSS is a widely used instrument for the assessment of subjective sleepiness. It is generally accepted that the descriptors defining each level of the scale are equivalent ways of characterising a particular level of sleepiness; and that sleepiness thus measured is an unidimensional construct. Existing research revealed ambiguity in measures of sleepiness, particularly in the structure of the SSS and its lack of relationship to objective measures [MacLean et al., 1989]. However, results from the present study into the 'wornout syndrome' suggested that this may not be so. Following sleep reduction one would expect subjects to report feeling more sleepy upon arising compared to baseline; paradoxically, it was subjects in the 'wornout syndrome' category that described enhanced sleepiness following sleep extension. Although the SSS purports to measure sleepiness, scores after oversleeping were higher than after sleep reduction. Therefore, it is argued, that the SSS does not adequately measure sleepiness.

It was hypothesised that subjects would feel wornout and fatigued after oversleeping; the SSS suggests that these subjects are sleepy. Globus [1969] observed that the adjective most often used by subjects following sleep extension was 'sleepy'. Like many other researchers, he too failed to clarify this term.

MacLean and Saskin [1992] highlighted possible weaknesses of the SSS. Analysis of the SSS results revealed several overlaps in subjective reports of levels of sleepiness at any one time. It was deemed necessary to investigate the structure of the SSS itself, particularly in an attempt to understand what we mean when we say we are sleepy. The ensuing study will attempt to delve deeper into the semantics of sleepiness. This is an important point that has a direct bearing on our understanding of the Wornout Syndrome. I will challenge the view that we merely feel 'sleepy' after oversleeping; instead I am proposing that we feel 'tired'. Moreover, I will demonstrate the ambiguities of the SSS and argue that the scale also measures tiredness under the guise of sleepiness. The importance of differentiating between the two is highlighted, specifically in order to understand the relationship between sleep and mood.

Method

1. Subjects

The subjects were 20 healthy volunteers recruited on campus. There were 4 male and 16 female subjects ranging in age from 18-35. All were free of any complaints of daytime sleepiness, with average nocturnal sleep [7-8 hours / night] and a willingness to conform to the strict experimental regimen.

2. Apparatus

i). Actigraphy

An actometer [Type: Z80-32K V1; available from: Gaehwiler Electronic, Eichtalstrasse 20, CH-8634 Hombrechtikon] was worn on the wrist. This is a small electronic device that responds to movement in one minute epochs. Actometers were worn initially for a period of one-week, to determine baseline sleep norms, and again on each experimental day and night, throughout the study.

3. Questionnaires

Subjects completed the following Questionnaires:
i).   General Sleep and Health Questionnaire
ii).   The 16PF
iii).   Morningness-Eveningness Questionnaire [MEQ]
iv).   Sleep Diary
v).   The Stanford Sleepiness Scale (SSS).
vi).   The Sleepiness Questionnaire
vii).   Mood Questionnaire

i). General Sleep and Health Questionnaire

This determined:-   a). General well-being of the individual
                      b). Usage of any medication
                      c). Subjects' usual quality and quantity of sleep.

ii). The 16PF

This scale [developed by Cattell et al., 1973] was utilised to assess personality. It was chosen on the grounds of its comprehensive assessment of the 16 primary and 4 secondary factors of personality. Subjects were presented with the questionnaire and were required to read the information on the cover. Once they understood the instructions they completed the form, which took approximately 40 minutes. Individual personality profiles were developed.

iii). Morningness-Eveningness Questionnaire [MEQ]

This questionnaire [developed by Horne and Ostberg, 1976] was presented in order to differentiate between morning and evening types of people. The questionnaire assesses subjects preferred times of retiring and awakening, their general well-being upon awakening and times when they are at their mental and physical peaks. Subjects are categorised as Owls (favourably predisposed towards the evening), Larks (predisposed towards the morning) or Neither.

iv). Sleep Diary

This scale was completed daily during the baseline week and subsequently on experimental days, where it also served to monitor compliance with the allocated sleep schedule.

This determined:- a). Sleep onset and wake-up times
  b). Quality of sleep
  c). A brief summary of the day's activities

v). The Stanford Sleepiness Scale (SSS).

The scale was completed on two occasions at the following times:-

  a). 1 hour after awakening
  b). 2 hours after awakening

vi). The Sleepiness Questionnaire

A 23-item Sleepiness Questionnaire (based on the individual items in the SSS) was developed [see Appendix] in order to distinguish between sleepiness and tiredness. Items in the SSS pertaining to fatigue were identified and distinguished from those more closely allied to sleepiness. These items were then randomly ordered and presented in the form of a questionnaire alongside the remaining descriptors used as controls (the principle of this categorisation was the factor loading utilised by MacLean et al., 1989 in their analysis of the SSS). The responses to the 23 descriptors were recorded as "yes", "no", or "can't say". This scale was administered hourly for the first 4 hours after awakening. The items were categorised as below:

Sleepiness	Tiredness
1. Sleepiness	1. Unable to concentrate
2. Losing interest in remaining awake	2. Let down
3. Sleep onset soon	3. Slowed down

vii). Mood Questionnaire

A mood scale (based on the Profile of Mood States, POMS) was completed hourly from awakening until retiring. The following factors were incorporated into the new mood scale (based on highest factor loading) from the six original dimensions in the POMS:-

Dimension	Factors
Fatigue	Wornout, Drowsy, Fatigued, Weary
Vigor	Alert, Energetic, Active
Confusion	Clear-headed, Confused
Tension	Tense, Calm
Depression	Happy, Sad, Hopeless, Lonely
Anger	Annoyed, Irritable, Bad-tempered

Mood ratings were based on a 5-point scale:
1. Not at all
2. A little
3. Moderately
4. Quite a bit
5. Extremely

— Samples of all questionnaires are presented in the Appendix. —

Procedure

Initially, all potential subjects were asked to complete the General Sleep and Health Questionnaire. Those volunteers able to meet a set criteria (e.g. lack of medical or sleep problems) were considered for the study.

In the first phase of the study subjects were required to wear an actometer and complete the Sleep Diary for a period of one-week. Subjects also visited the sleep laboratory on one occasion during the week for a short personality assessment (using the 16PF) and circadian profiling (via the Morningness-Eveningness Questionnaire: MEQ). From the actometer readings and daily log of the Sleep Diary, it was possible to determine baseline levels of sleep onset and times of awakening for each subject.

In the second phase of the experiment a personal sleep schedule was drawn up for each subject outlining the sleep regimen to be followed during each experimental night. The conditions were counterbalanced so that some subjects extended their

sleep first in week 1 and later reduced it in say, week 4, whilst others reduced their sleep in week 2 and extended it in week 3. Subjects were randomly assigned to these conditions.

Table 5.1 Experimental design showing the regimen followed by Subject 5.

	Condition *	Time of Retiring	Time of Awakening
Week 1 [Tues]	Baseline 1	2300 h	0700 h
Week 2 [Tues]	Sleep Extension	2300 h	0900 h
Week 3 [Tues]	Baseline 2	2300 h	0700 h
Week 4 [Tues]	Sleep Restriction	2300 h	0500 h

* Each experimental condition was preceded by 6 nights of normative sleep.

Upon arising, each subject was required to complete the following questionnaires throughout the experimental day:-

1. Mood Questionnaire - hourly
2. Activity Sheet - hourly
3. The Sleepiness Questionnaire - hourly for the first 4 hrs after arising
4. The SSS - hourly for the first 2 hrs after arising

Subjects were asked to follow their usual daily activity, the diary log enabled any deviations from the norm to be monitored.

After each experimental day subjects were required to return to the sleep laboratory in order to record compliance with the sleep schedule and to discuss any difficulties that arose during the study. At each meeting, completed questionnaires were collected and new ones issued for the subsequent experimental night [6 days later]. Once all the conditions had been fulfilled, the actometers were collected and the data downloaded to a personal computer. The actigraphs displayed sleep/wake patterns and levels of activity. Actigraph readings were compared to sleep diaries, and a satisfactorily high degree of correlation was attained in all cases.

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Results

Subject Profile
a). Sleep Habit
From the baseline data, subjects were classified as either regular or irregular sleepers (in the case of the latter if sleep/wake times varied by more than two hours from night to night). There were 11 regular and 9 irregular sleepers.

Table 5.2a Sleep Habit Regular vs. Irregular Sleepers

Regular Sleepers (46% = Morn. type, 9% = Eve. type)	N=11 [9F, 2M]
Irregular Sleepers (11% = Morn. type, 33% = Eve. type)	N=9 [7F, 2M]

b). Circadian Typology
According to the MEQ results, there were 5 morning types and 5 evening types, the remaining 10 subjects were categorised as neither morning nor evening type. Of the regular sleepers 46% were categorised as morning types, 9% were evening types and 45% were neither. Of the irregular sleepers, 11% were morning types, 33% were evening types and 56% were neither.

Table 5.2b Circadian Typology of Subjects.

Morning Type	N = 5
Evening Type	N = 5
Neither	N = 10

c). Personality

Personality was assessed via the 16PF and subjects were categorised as displayed in Table 5.3 [with one subject missing].

Table 5.3 The personality profiles of 19 subjects.

Primary Factors: Scores 1-3 — Scores 8-10

Factor	Descriptor	Male	Female	Total	Descriptor	Male	Female	Total
A	Reserved	1	2	3	Warm-hearted	0	3	3
B	Concrete-Thinking	0	2	2	Abstract-Thinking	2	2	4
C	Affected By Feelings	1	5	6	Emotionally Stable	0	1	1
E	Humble	1	4	5	Assertive	1	1	2
F	Sober	0	2	2	Happy-Go-Lucky	1	3	4
G	Expedient	2	8	10	Conscientious	0	0	0
H	Shy	0	4	4	Venturesome	2	3	5
I	Tough-Minded	1	0	1	Tender-Minded	0	7	7
L	Trusting	0	2	2	Suspicious	0	5	5
M	Practical	0	0	0	Imaginative	1	2	3
N	Forthright	1	3	4	Shrewd	1	2	3
O	Placid	1	1	2	Apprehensive	1	2	3
Q_1	Conservative	0	6	6	Experimenting	2	2	4
Q_2	Group-Dependent	0	2	2	Self-sufficient	1	3	4
Q_3	Undisciplined	0	3	3	Controlled	0	0	0
Q_4	Relaxed	1	2	3	Tense	0	6	6

Second-Order Factors: Below Mean of 5.5 — Above Mean of 5.5

Factor	Descriptor	Male	Female	Total	Descriptor	Male	Female	Total
Q_I	Introversion	1	8	9	Extroversion	2	8	10
Q_{II}	Low Anxiety	3	5	8	High Anxiety	0	11	11
Q_{III}	Tender minded Emotionality	0	10	10	Tough Poise	3	6	9
Q_{IV}	Subduedness	1	6	7	Independence	2	10	12

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The first sixteen factors (A through to Q4) represent the primary factors. Subjects with sten scores of 1 to 3 and 8 to 10 are classified by gender. For example in Factor H, 4 subjects (0M 4F) are described as shy with scores of 1 to 3, whilst 5 subjects (2M 3F) are venturesome with factor scores 8 to 10.

Factors QI to QIV represent the second-order factors. Here subjects are categorised according to whether they are below or above average (mean = 5.5). For example there were 9 introverts (1M 8F) and 10 extroverts (2M 8F).

The above table illustrates that more subjects were classified as moderate personality types, they were neither at one extreme nor the other. This is to be expected, since most individuals in the general population tend to fall into the middle ranges of personality type.

Post Sleep Questionnaire

This Questionnaire monitored subjects sleep onset and time of awakening after Baseline, Sleep Extension and Sleep Reduction. The findings are displayed in table 5.4a below:

Table 5.4a   Mean [with se] Sleep Onset and Awakening Times.

	Baseline (normative)	Extension (+2hrs)	Reduction (-2hrs)
Sleep Onset	0020h [0.16]	0010h [0.16]	0020h [0.17]
Time of Awakening	0740h [0.18]	0935h [0.23]	0540h [0.21]

All 20 subjects normally averaged 7hrs and 20 mins of sleep per night. On experimental days the average time of awakening for Sleep Extension was 0935h and for Sleep Reduction, 0540h. The variance [se] for both sleep onset and time of awakening were comparable across all 3 experimental conditions. Actometer readings were also obtained to indicate times of retiring and awakening. A satisfactorily high degree of correlation was obtained between actometer readings and subjective data.

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Sleep Quality

Sleep Quality was assessed 30 minutes after awakening, using a 5-point scale. A one-way ANOVA was computed, comparing the responses across all conditions.

Table 5.4b Mean [with se] ratings of subjective sleep quality.

Baseline	Extension	Reduction	1-way ANOVA
2.65 [0.13]	3.05 [0.2]	3.0 [0.15]	$p = 0.17$ NS $F = 1.83$ (2, 57)

Key
Quality of sleep obtained
2 = better than normal 3 = normal 4 = worse than normal

The mean values representing sleep quality are very similar following baseline, sleep extension and sleep reduction. The quality of sleep obtained after each experimental condition was perceived to be very close to normal. A one-way ANOVA on the data confirmed that the scores were not statistically different.

Analysis of Mood

a). Hourly mood scores were obtained for each of the six dimensions of affect: Fatigue, Vigor, Confusion, Depression, Anger and Tension. Two-way ANOVA's were performed separately for each mood dimension comparing the scores after Baseline, Sleep Extension and Sleep Reduction for all subjects together. The Between-Group factor was sleep length [3 levels] and the Within-Group factor was time [14 hours]. The results of this analysis comparing mood scores after Baseline, Sleep Extension and Sleep Reduction for all 20 subjects will be presented first.

b). Mean baseline values were calculated for each dimension of mood. Scores from each experimental condition [Extension and Reduction] were subtracted from mean baseline scores in order to examine changes from baseline. Subjects were categorised as having the 'wornout syndrome' if their fatigue scores for the first 4h following awakening after Sleep Extension were positive [> 0], relative to baseline. A two-way ANOVA was carried out between mood scores (e.g. Fatigue, Vigor)

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and Personality / Sleep Traits (e.g. Introversion, WOS, Regular Sleepers). The within factor (repeated measure) was Time. In terms of analysis, Sleep Extension was examined first, and then Sleep Restriction. Results are to be presented for significant findings and interesting trends thus:

      1. Main effects
      2. Within factor
      3. Interactions
      — as shown in Table 5.5:

Table 5.5 Mood Analysis

Sleep Extension	Sleep Reduction

a. Fatigue   x N/WOS
         x Regular /Irregular Sleepers
         x Introversion/Extroversion
b. Vigor   x Regular/Irregular Sleepers
         x N/WOS
         x Introversion/Extroversion
c. Confusion   x Regular/Irregular Sleepers
         x N/WOS
         x Morning/Evening type
         x Impulsivity
d. Depression x N/WOS
         x Impulsivity
e. Tension   x Impulsivity
         x N/WOS
         x Regular/Irregular Sleepers
f. Anger   x N/WOS
         x Morning/Evening types
	a. Fatigue x N/WOS
	         x Toughness Poise
	         x Introversion
	b. Vigor x Introversion/Extroversion
	         x N/WOS
	
	c. Confusion x N/WOS
	
	d. Depression x N/WOS
	
	e. Tension x N/WOS
	         x Introversion/Extroversion
	         x Impulsivity
	         x Morning/Evening type
	f. Anger x N/WOS

Results of Mood Analysis

Reviewing fatigue scores, eight subjects demonstrated the 'wornout syndrome' following oversleep. An interesting finding that emerged was 7 of the 8 subjects experiencing the 'wornout syndrome' were regular sleepers. Data was examined for

any order effects between conditions, and as there were no transfer effects, data was pooled for analysis.

A. Sleep Length and Mood

a) Fatigue

Main Effects

There was a significant difference in fatigue between the conditions, $F = 3.38$ [2, 57] $p = 0.04$. Post-hoc analysis revealed that subjects were significantly more fatigued after Sleep Reduction compared to Baseline, $p < 0.05$. Fig 5.1 illustrates the findings. Although the difference in fatigue after Sleep Extension was not significant, trends emerged indicating that subjects reported enhanced fatigue, compared to Baseline.
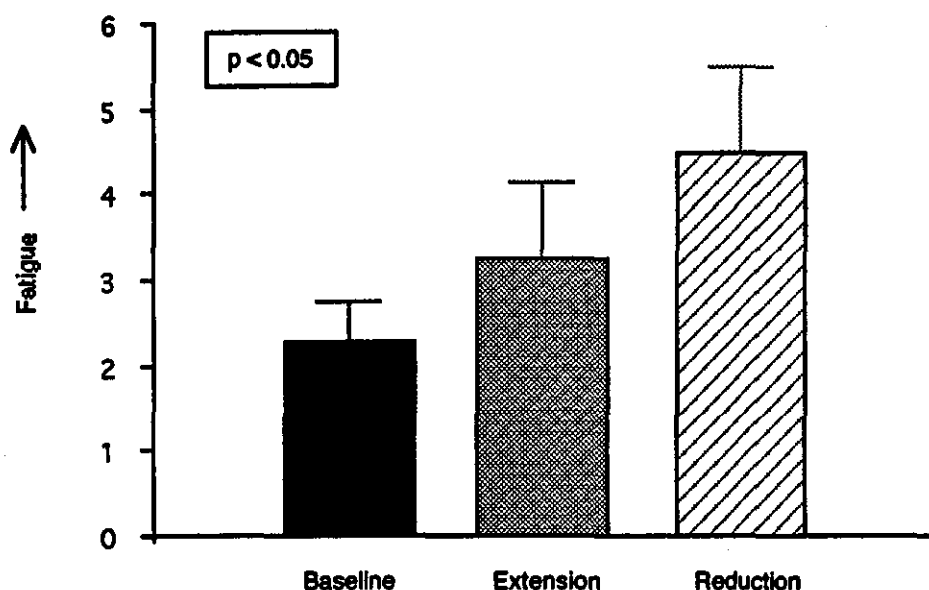
Fig. 5.1 Fatigue scores after Baseline, Sleep Extension and Sleep Reduction in all 20 subjects.

Interactions

Interactions between conditions x time were highly significant, $F = 2.8$ [26, 741] $p < 0.001$. Fig. 5.2 indicates that following Sleep Reduction, subjects were significantly more fatigued than Baseline or Sleep Extension for the first 3 hours after awakening. Post-hoc analysis revealed significance [$p < 0.05$] between Baseline and Sleep Reduction at hours 1, 2 and 3. Following Sleep Extension, subjects were significantly more fatigued than Baseline 5, 6, 7, 8 and 9 hours after awakening.

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Fig. 5.2 Interaction of Fatigue scores after Baseline, Sleep Extension and Sleep Reduction x Time in all 20 subjects.

b) Vigor

Main Effects

Vigor was not significantly different across conditions, $F = 1.66$ [2, 56] $p = 0.2$, although trends emerged indicating that subjects were least energetic following Sleep Reduction.

Interactions

Significant interactions were observed between the conditions x time, $F = 2.26$ [26, 728] $p < 0.001$. Fig. 5.3 illustrates the findings. At hour 1, subjects in Sleep Extension were more energetic than either Baseline or Sleep Reduction. Following Sleep Reduction, subjects were significantly less energetic than Baseline and Sleep Extension for the first 5 hours after awakening. Thereafter, Vigor was comparable across conditions.
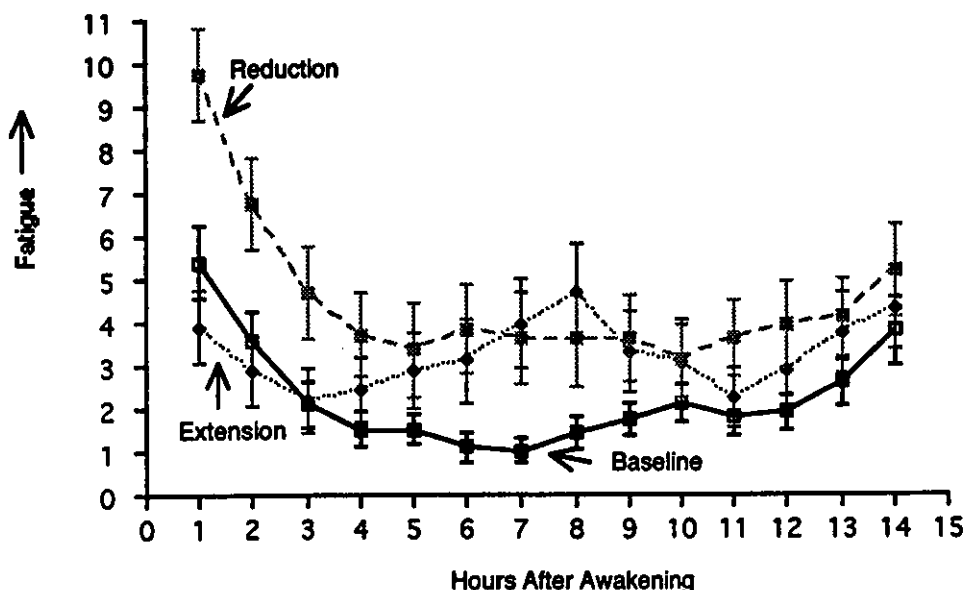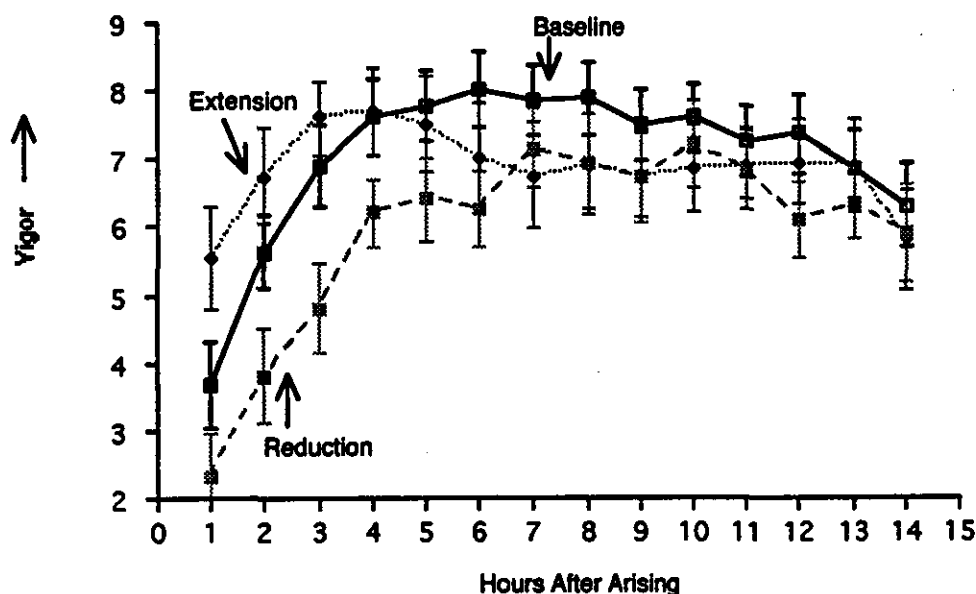
Fig. 5.3 Interaction of Vigor scores after Baseline, Sleep Extension and Sleep Reduction x Time in all 20 subjects.

c) Confusion

The differences in confusional state between conditions failed to approach significance, $F = 1.05$ [2, 57] $p = 0.36$. Similarly, the interactions between conditions x time were not significant, $F = 0.75$ [26, 741] $p = 0.8$.

d) Depression

There were no significant differences in Depression between the conditions, $F = 0.52$ [2, 57] $p = 0.6$ or in the interactions between conditions x time, $F = 0.98$ [26, 741] $p = 0.49$.

e) Tension

Similarly, for Tension, there were no significant differences between the conditions, $F = 1.36$ [2, 57] or in the interactions between conditions x time, $F = 0.93$ [26, 741] $p = 0.57$.

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f) Anger

Although the differences in anger between the conditions did not reach significance, $F = 1.43$ [2, 57] $p = 0.24$, the interactions between the conditions x time approached significance, $F = 1.48$ [26, 741] $p = 0.06$. Fig. 5.4 illustrates the findings. Post-hoc analyses revealed that after Sleep Reduction, subjects were more angry for the first 2 hours after awakening, compared to Baseline or Sleep Extension. Thereafter, scores were comparable across conditions.
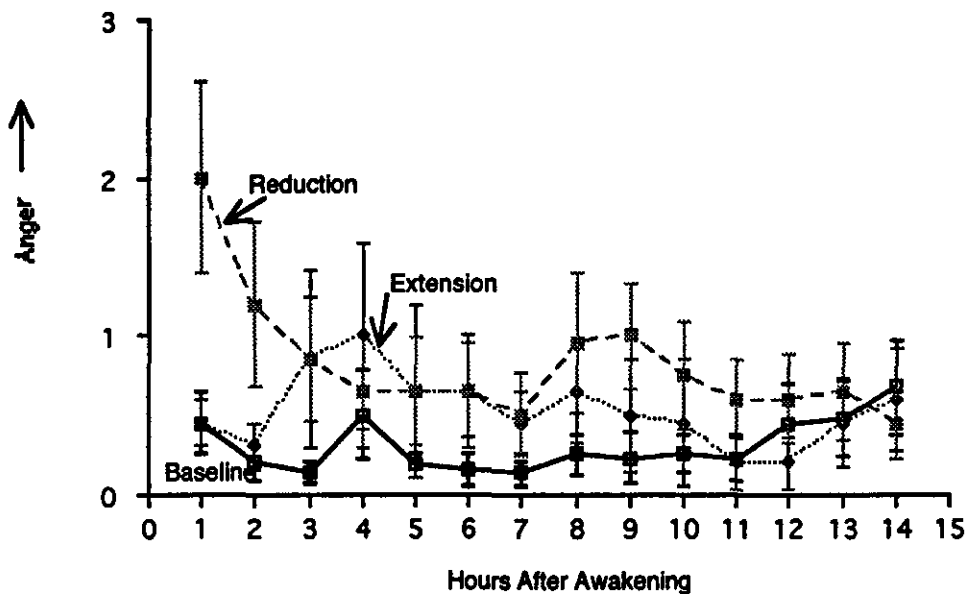
Fig. 5.4 Interaction of Anger scores after Baseline, Sleep Extension and Sleep Reduction x Time in all 20 subjects.

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The above analysis compared actual mood scores after Baseline, Sleep Extension and Sleep Reduction for all 20 subjects together. It was evident that subjects were adversely affected following Sleep Reduction, this was reflected by enhanced levels of Fatigue. Although the differences were not statistically significant following Sleep Extension, subjects indicated trends towards increased fatigue compared to Baseline [see fig. 5.1]. The following analyses will differentiate between regular and irregular sleepers and will compare subjects with elevated fatigue [the WOS group] to the remaining subjects [the NWOS group]. Differences in personality in relation to mood following Sleep Extension and Sleep Reduction will also be examined.

B. Sleep Extension

a) Fatigue

Main Effects

There was a significant difference in fatigue scores between subjects exhibiting the WOS and the NWOS, $F = 49.7$ [1, 18] $p < 0.001$. Figure 5.5 illustrates the divergent trends; the WOS group were significantly more fatigued than the NWOS group. Fatigue in the NWOS group was very similar to baseline.
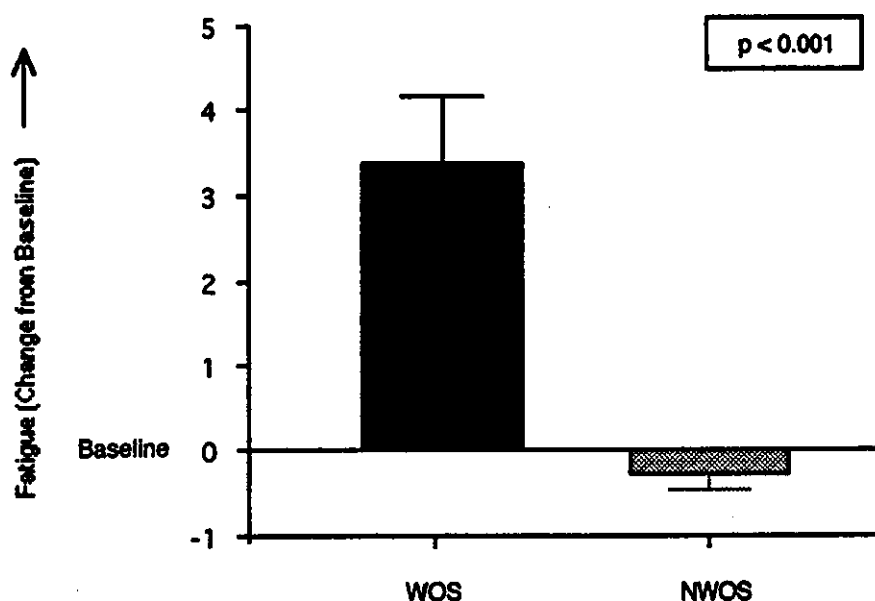
Fig. 5.5 Fatigue in WOS and NWOS groups after Sleep Extension.

A significant difference was observed in fatigue between regular and irregular sleepers, $F = 6.49$ [1, 18] $p = 0.02$. Regular Sleepers described elevated fatigue [see fig. 5.6]. The mean fatigue score in irregular sleepers was equivalent to that of baseline. This is undoubtedly an important finding, it is evident that regular and irregular sleepers are differentially affected by Sleep Extension.
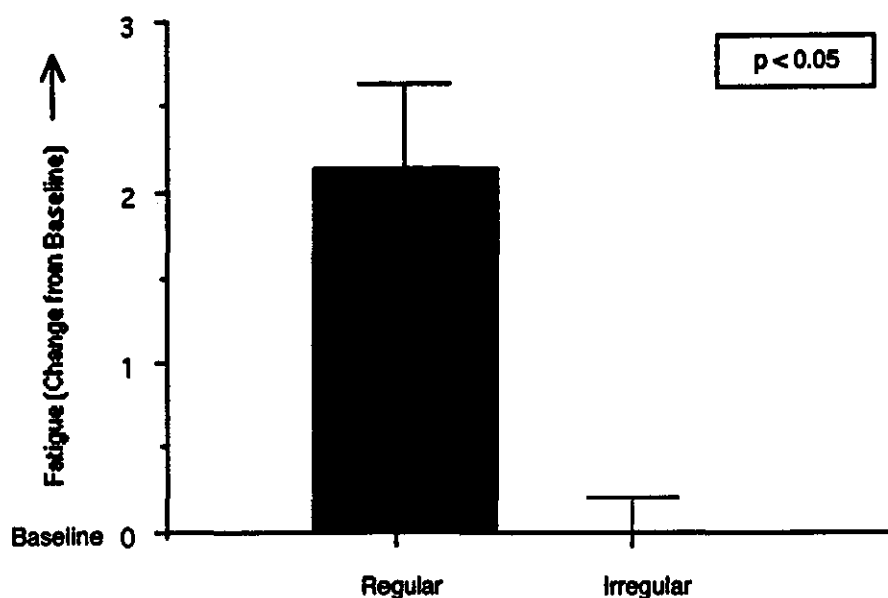
Fig.5.6 Fatigue in Regular and Irregular Sleepers after Sleep Extension.

There was a significant difference in fatigue scores between Introverts and Extroverts, $F = 6.44$ [2, 17] $p = 0.008$. Post hoc tests revealed that Introverts were more fatigued following Sleep Extension, $p < 0.01$ [see fig. 5.7]. Subjects classified as neither Introvert nor Extrovert also described increased fatigue relative to baseline, but to a lesser degree than Introverts. Interestingly Extroverts found the extra sleep beneficial to mood; this was reflected in their diminished fatigue scores, relative to baseline.
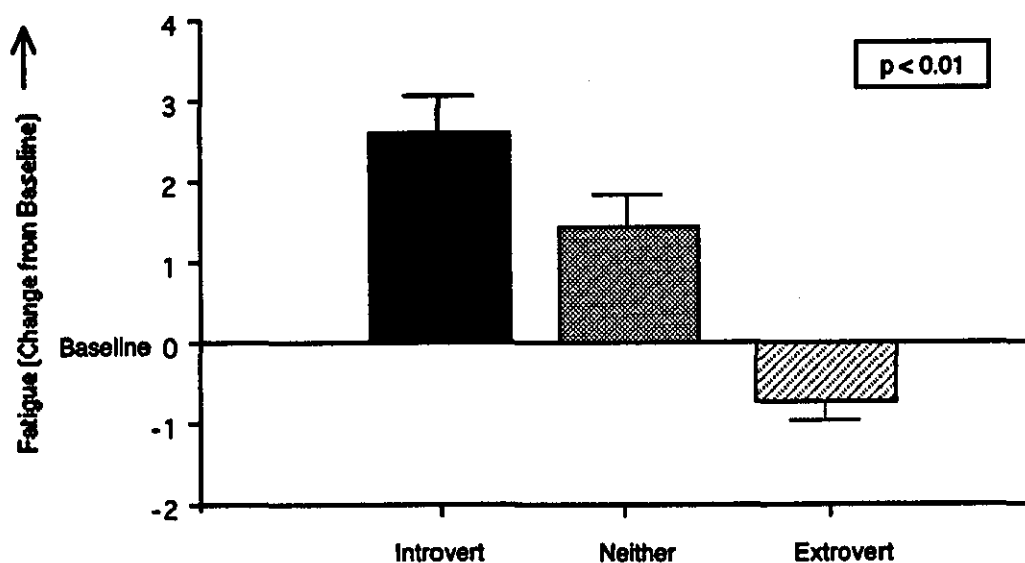
Fig. 5.7 Fatigue in Introverts and Extroverts after Sleep Extension.

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Figure 5.8 shows fatigue changes throughout the day in all 20 subjects. After Sleep Extension, there were trends indicating that subjects were more fatigued than baseline, throughout the day. After 1700 hours the increasing fatigue levelled off gradually and descended to below baseline after 2000 hours.
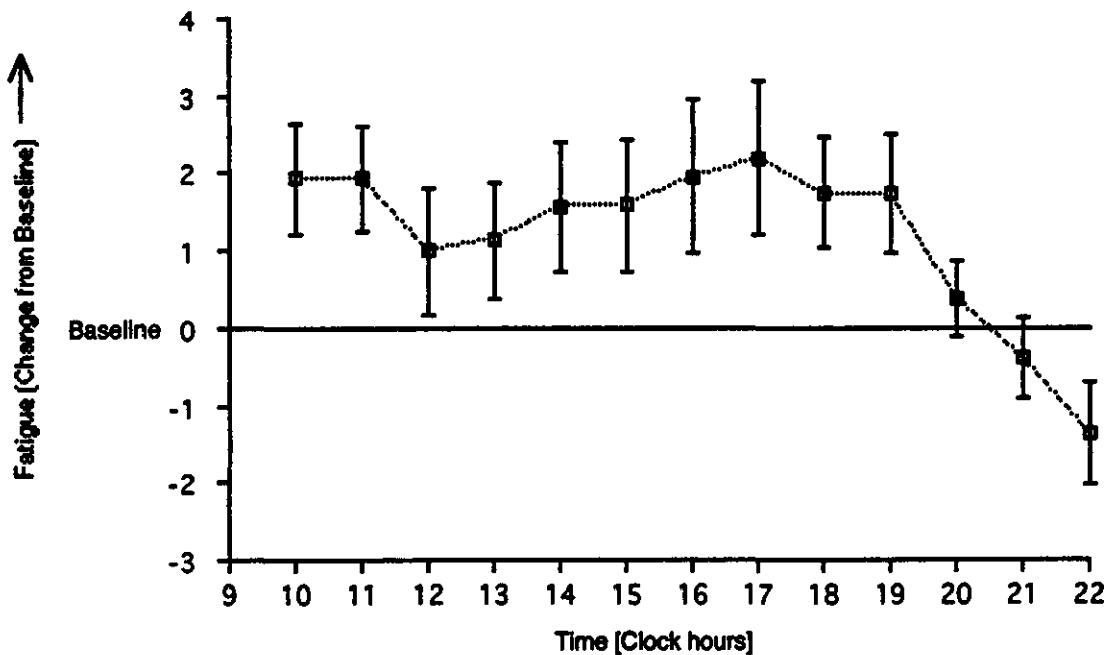
Fig. 5.8 Diurnal variation in Fatigue after Sleep Extension.

Interactions

Figure 5.9 indicates significant differences between regular and irregular sleepers over time, F = 2.35 [12, 216] p = 0.007. Regular Sleepers showed elevated fatigue throughout until 1900 hours when levels dropped to those of Irregular Sleepers. Fatigue in Irregular Sleepers was comparable to baseline.
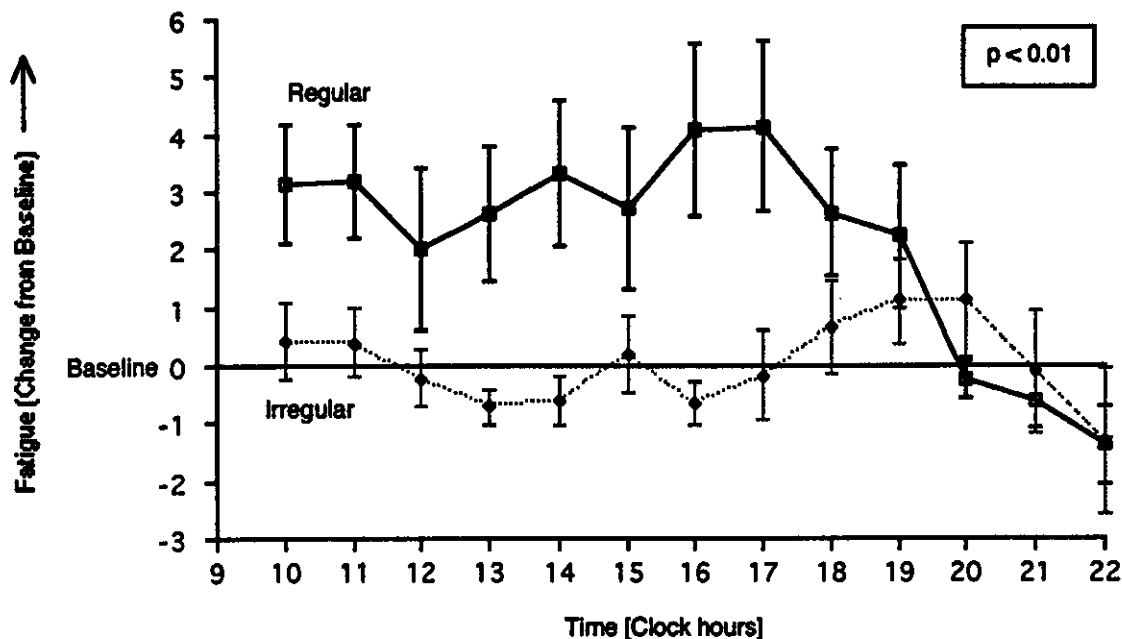
Fig. 5.9 Fatigue In Regular and Irregular Sleepers after Sleep Extension.

Post-hoc analyses indicated significant differences in fatigue between Regular and Irregular sleepers at the following times: Regular sleepers at 1600 and 1700 hours exhibited significantly divergent scores from Irregular sleepers at 1300, 1400, 1600 and 2200 hours. In addition, fatigue in Irregular sleepers at 2200 hours was significantly different from Regular sleepers at 1100 and 1400 hours.

Examining fatigue scores between the WOS and NWOS groups over time revealed significant differences, $F = 9.32$ [12, 216] $p < 0.001$ [see fig. 5.10]. The WOS group were adversely affected by Sleep Extension. They exhibited increased fatigue throughout the day approaching baseline after 1900 hours. In contrast the NWOS group exhibited fatigue levels similar to baseline. Post-hoc analysis indicated significant differences in the WOS and NWOS groups between 1000 to 1800 hours.
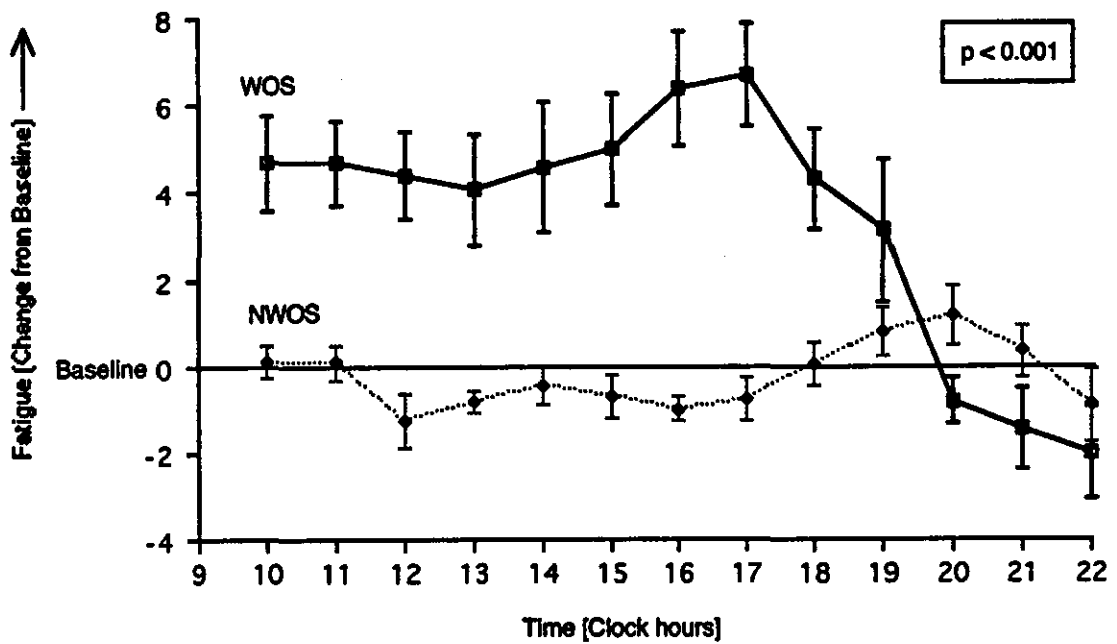
Fig. 5.10 Fatigue in WOS and NWOS groups after Sleep Extension.

b) Vigor

Main Effects.

There was a significant difference in Vigor between Regular and Irregular Sleepers, $F = 5.52$ [1, 18] $p = 0.03$. Regular Sleepers were adversely affected by Sleep Extension. By contrast, Irregular Sleepers described a higher level of energy, and perceived the extra sleep as beneficial to mood [see fig. 5.11].
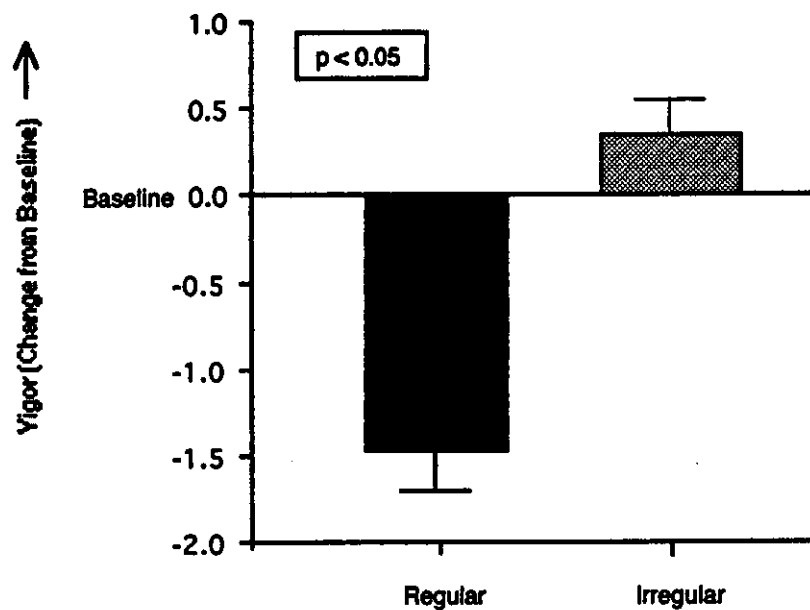
Fig. 5.11 Vigor in Regular and Irregular Sleepers after Sleep Extension.

Significant differences were observed in Vigor between subjects in the WOS and NWOS groups, $F = 26.7$ [1, 18] $p < 0.001$. Figure 5.12 indicates that subjects in the WOS group described diminished energy levels after Sleep Extension, relative to baseline. Subjects in the NWOS group were more vigorous: they found the extra sleep beneficial to mood.
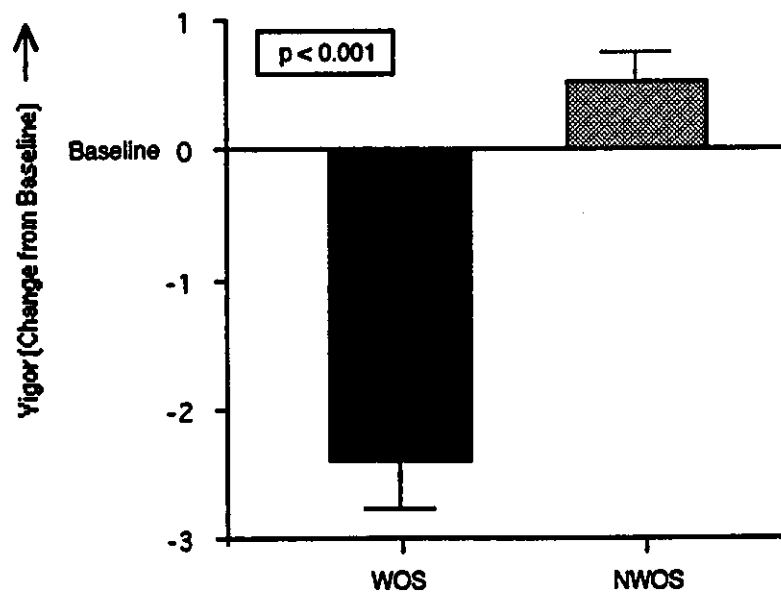
Fig. 5.12 Vigor in the WOS and NWOS groups after Sleep Extension.

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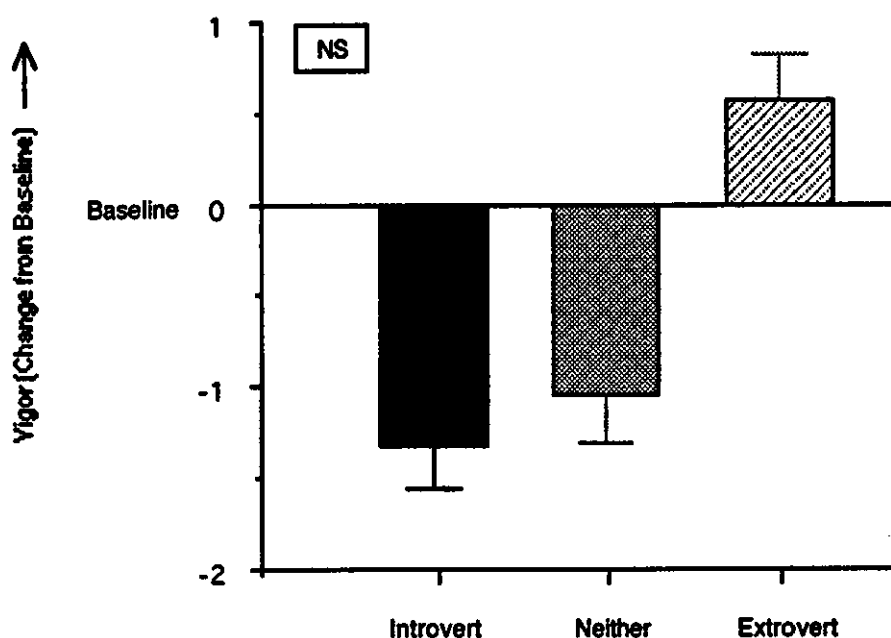
Fig. 5.13 Vigor in Introverts and Extroverts after Sleep Extension.

There was an interesting trend between Introverts and Extroverts. Although not significant, $F = 2$ [2, 17] $p = 0.17$, Figure 5.13 illustrates divergent trends, both Introverts and Neither groups reported diminished Vigor, whilst Extroverts described increased Vigor following Sleep Extension.

Within-factor: Time

Figure 5.14 indicates changes in vigor throughout the day. Vigor was reported to be below baseline upon awakening, it approached baseline levels at 1300 hours. Levels of vigor gradually decreased to a trough at 1700 hours, followed by a sharp incline towards baseline levels at 2000 hours.
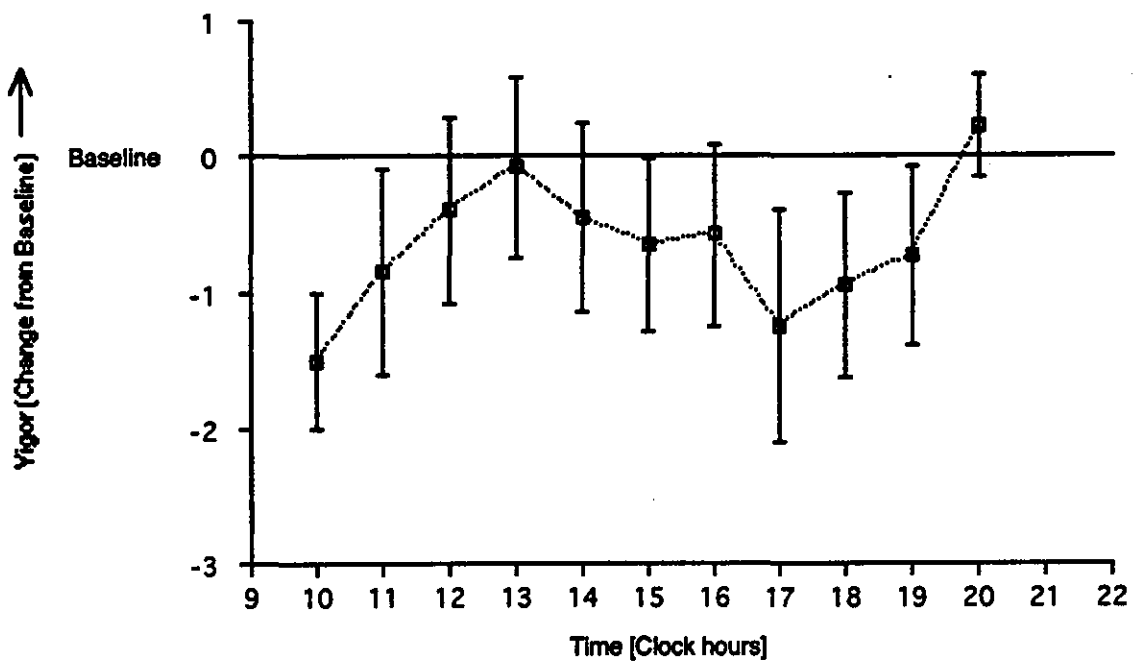
Fig. 5.14 Diurnal variation in Vigor after Sleep Extension.

Interactions
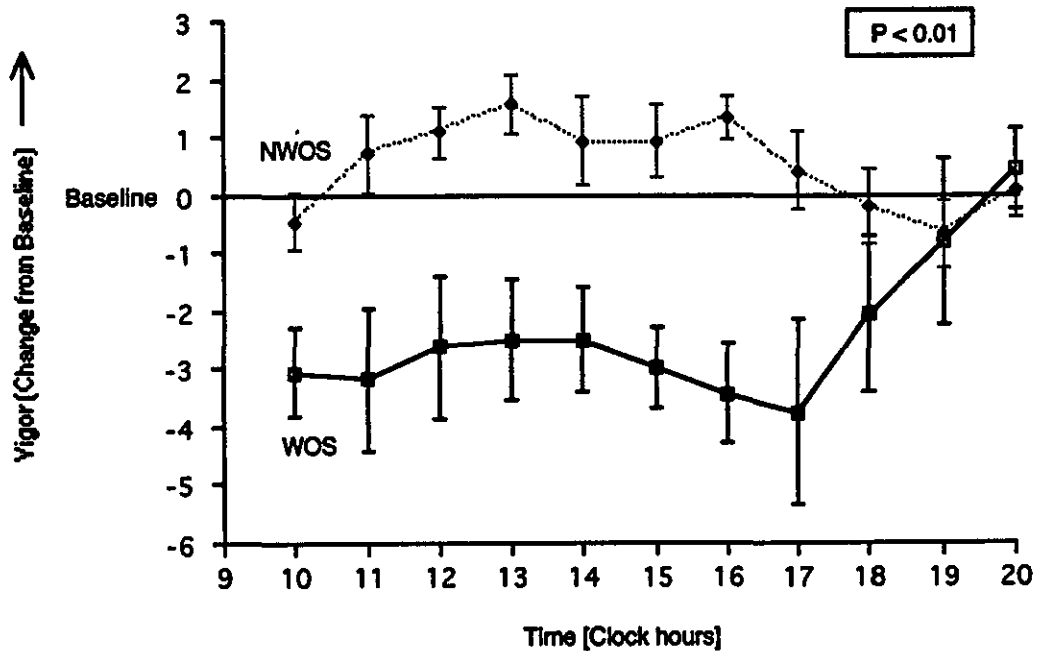
Fig. 5.15 Vigor in WOS and NWOS groups after Sleep Extension.

Figure 5.15 illustrates changes in Vigor over time in the WOS and NWOS groups.
Significant differences emerged, F = 2.65 [10, 180] p < 0.01. Subjects in the WOS

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group described diminished vigor throughout the day, Vigor improved after 1700 hours and only approached baseline levels after 1900 hours. Subjects in the NWOS group displayed little deviation from baseline; if anything, scores improved slightly, towards increased Vigor. Post-hoc analyses indicated significant differences [p < 0.01] between the groups at the following times: WOS at 1000h and 1100h vs. NWOS from 1100 to 1600h; WOS at 1200, 1300 and 1400h vs. NWOS at 1300 and 1600h; WOS at 1500h vs. NWOS from 1200 to 1600h; WOS at 1600 and 1700h vs. NWOS from 1100 to 1700h.

c) Confusion

Main Effects
Although there were no significant differences in confusional state between subjects, the divergence between regular and irregular sleepers approached significance, $F = 3.72$ [1, 18] $p = 0.07$. Certain trends emerged: Regular Sleepers were in a greater confusional state than Irregular Sleepers. In addition, contrasting trends were evident in Confusional state between subjects in the WOS and NWOS groups. Although not significant, $F = 2.89$ [2, 17] $p = 0.11$, it was apparent that the WOS group were in a greater confusional state; the NWOS group deviated very little from baseline.

Although the results were not significant, morning type subjects were considerably confused following Sleep Extension, subjects classified as neither or Evening type exhibited little variation from baseline. Non-significant divergent trends emerged in Confusion between low and high impulsives. Subjects classified as not very impulsive revealed trends towards a greater confusional state than either the highly impulsive or neither groups.

d) Depression

Main Effects
There were no significant differences between Depression after Sleep Extension. However, certain trends emerged: the difference between the WOS and NWOS groups approached significance, $F = 3.13$ [1, 18] $p = 0.09$. The WOS group were considerably more depressed than the NWOS group, who by contrast, exhibited little deviation from baseline.

In terms of Impulsivity, subjects classified as low impulsives described elevated depression after Sleep Extension, $F = 2.09$ [2, 17] $p = 0.15$. Subjects in the neither and high impulsivity groups were unaffected by the oversleep.

Interactions

A further trend became evident: diurnal variation in Depression between the WOS and NWOS groups approached significance, $F = 3.13$ [1, 18] $p = 0.09$. The WOS group reported an enhanced depressive state relative to baseline.

e) Tension

Main Effects
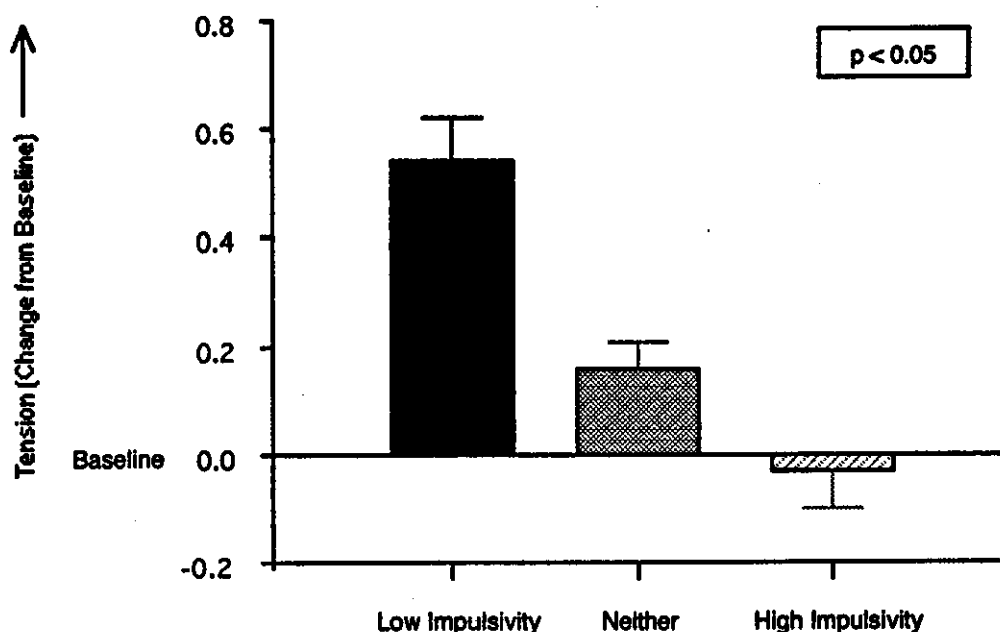
Fig. 5.16 Tension after Sleep Extension.

There was a significant difference in Tension between subjects in the 3 Impulsivity groups, $F = 3.42$ [2, 17] $p = 0.05$. Post-hoc analysis revealed differences between subjects in the low and high impulsivity groups, $p < 0.05$ [see fig. 5.16]. Low impulsives were in a state of greater tension following Sleep Extension.

The difference in Tension between the WOS and NWOS groups approached significance, $F = 3.43$ [1, 18] $p = 0.08$. Subjects in the WOS group were slightly more tense than those in the NWOS groups.

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Although the findings were not significant, $F = 2.83$ [1, 18] $p = 0.11$, differences in tension between regular and irregular sleepers indicated certain trends; regular sleepers described increased tension relative to irregular sleepers.

f) Anger

Main Effects

Figure 5.17 displays significant differences in Anger between the WOS and NWOS groups, $F = 4.94$ [1, 18] $p = 0.04$, the WOS group described greater anger following Sleep Extension, relative to the NWOS group.
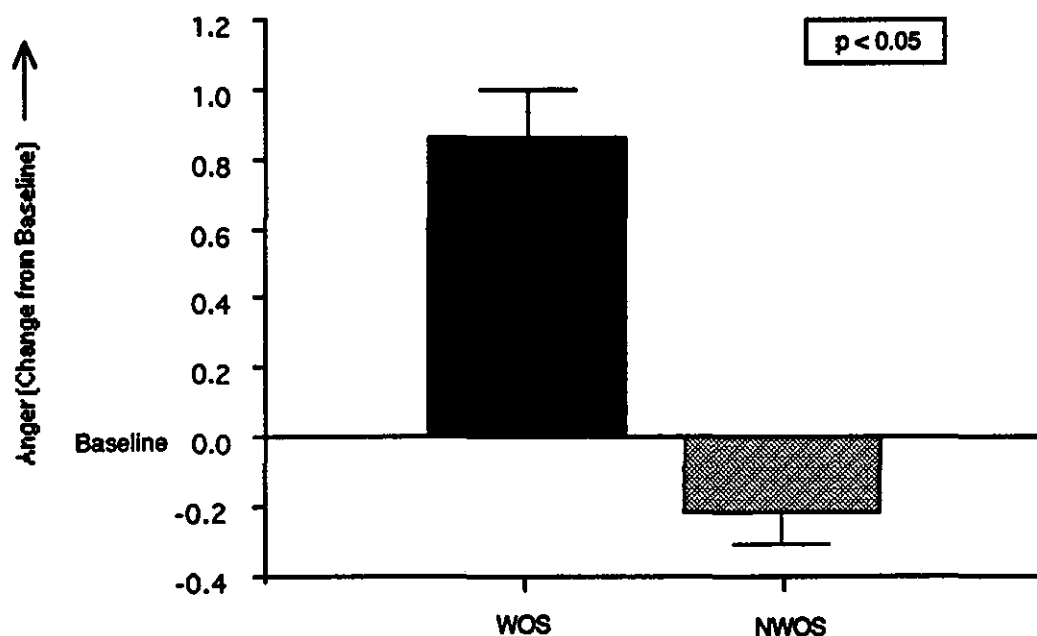
Fig. 5.17 Anger after Sleep Extension.

Interactions

A two-way ANOVA examining Anger in Morningness [3 groups] over time revealed a significant difference in Anger, $F = 1.64$ [22, 187] $p = 0.04$. Figure 5.18 illustrates the divergent trends. All 3 groups reported similar levels of anger upon awakening. Anger diverged markedly at 1200 hours, subjects in the Neither group reported a rapid increase in anger, peaking at 1300 hours. Morning type subjects reached a peak in anger at 1800 hours while evening types showed considerable diurnal variation. Post-hoc analysis indicated significant differences between

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subjects in the neither Morning nor Evening types at 1300h compared to 2000 and
2100h.
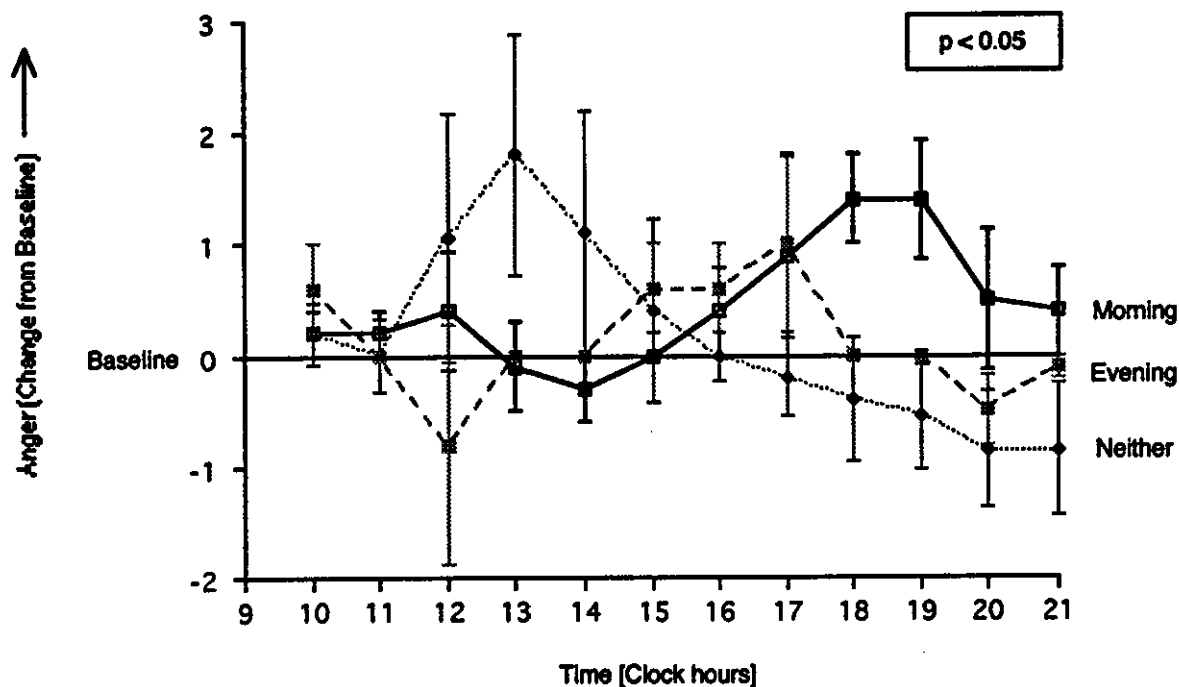
Fig.5.18 Anger in Morning and Evening Types after Sleep Extension.

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C. Sleep Reduction

a) Fatigue

Within-factor: Time

Figure 5.19 illustrates variation in fatigue throughout the day in all 20 subjects.
Upon awakening, levels of fatigue approached baseline. However, subjects
described a consistent pattern of elevated fatigue throughout the day.
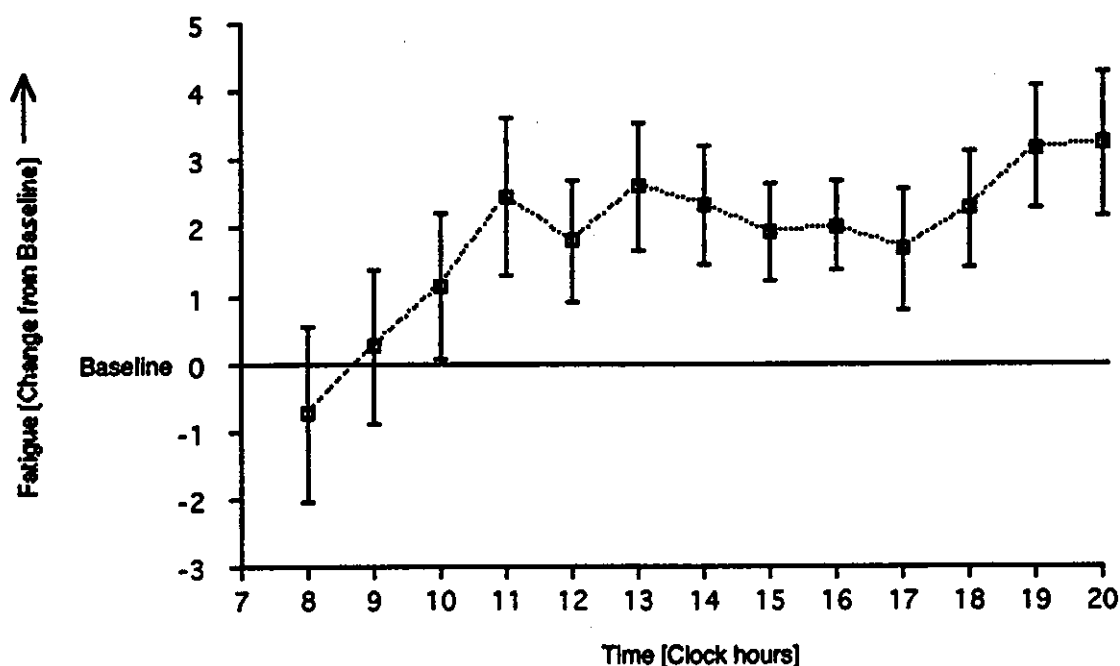
Fig. 5.19 Diurnal variation in Fatigue after Sleep Reduction [n=20].

Interactions

There was a significant difference in fatigue scores between subjects in the WOS [n=8] and NWOS [n=12] groups over Time, F = 2.28 [14, 252] p < 0.01. Figure 5.20 illustrates that subjects in the NWOS group were adversely affected by Sleep Reduction: this was reflected in their elevated fatigue scores. This difference between the groups was particularly evident in the first 4 hours after awakening. Subjects in the WOS group described less fatigue than usual [baseline] upon awakening. Fatigue approached baseline 4 hours after arising and was maintained at a similar level until 1600 hours. It was then followed by an increase in fatigue, surpassing the NWOS group. Post-hoc analyses revealed significant differences [p < 0.01] between the groups at the following times: WOS at 0800h vs. NWOS from 0900 to 1600h and at 1900 and 2000h; WOS at 0900h vs. NWOS at 1100 and 1300h.
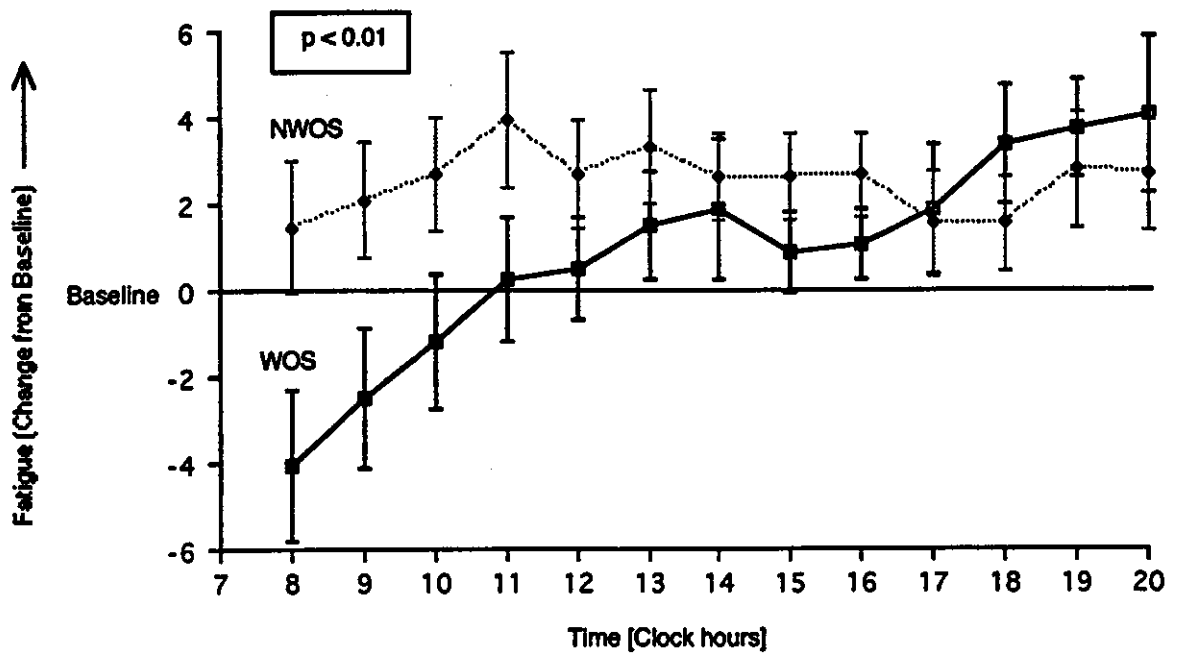
Fig. 5.20 Diurnal variation in Fatigue after Sleep Reduction.

Fatigue over time in Toughness Poise approached significance, $F = 1.48$ [28, 238] $p = 0.06$. Fatigue in Tough-minded subjects consistently exceeded baseline throughout the day. Tender minded subjects described diminished fatigue upon awakening, approaching baseline at 1000 hours, and rising steadily until 1300 hours where it levelled off for the rest of the day [see fig. 5.21]. Post-hoc analyses indicated significant differences [$p < 0.05$] between Tender minded subjects at 0800 and 0900h and Tough-minded subjects at 2000h.

93
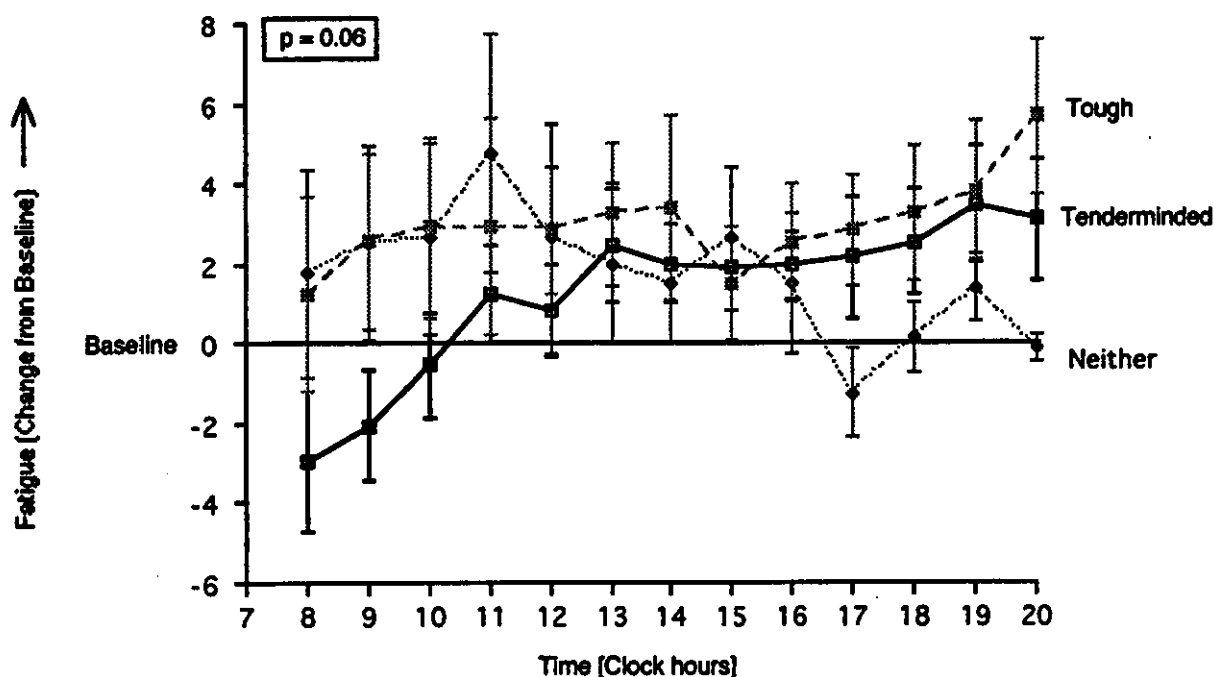
Fig. 5.21 Fatigue in Tough and Tender minded subjects.

Regarding the interaction between Fatigue and Introversion x Time, trends indicated that Extroverts were more fatigued than both the Neither group and Introverts for the first three hours, F = 1.43 [28, 238] p = 0.08. Interestingly, Introverts were least affected by Sleep Reduction.

b) Vigor

Main Effects

Significant differences were obtained for Introversion / Extroversion, F = 3.62 [2, 17] p < 0.05. Vigor in both Introverts and Extroverts remained close to baseline levels after Sleep Reduction, whilst Neither types showed a significant drop in Vigor.
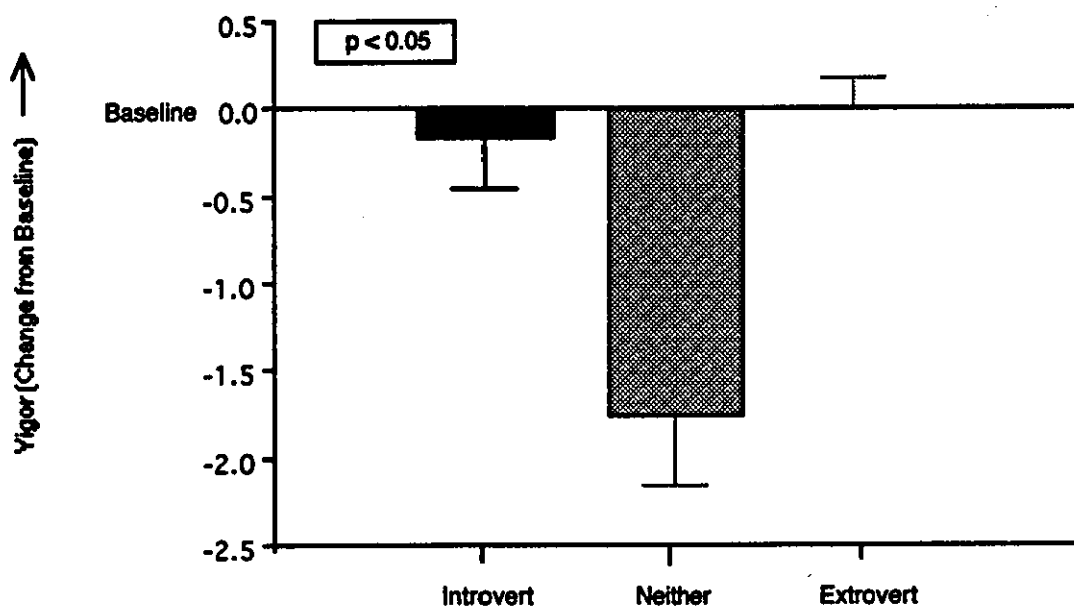
Fig. 5.22 Vigor after Sleep Reduction.

Within-factor: Time
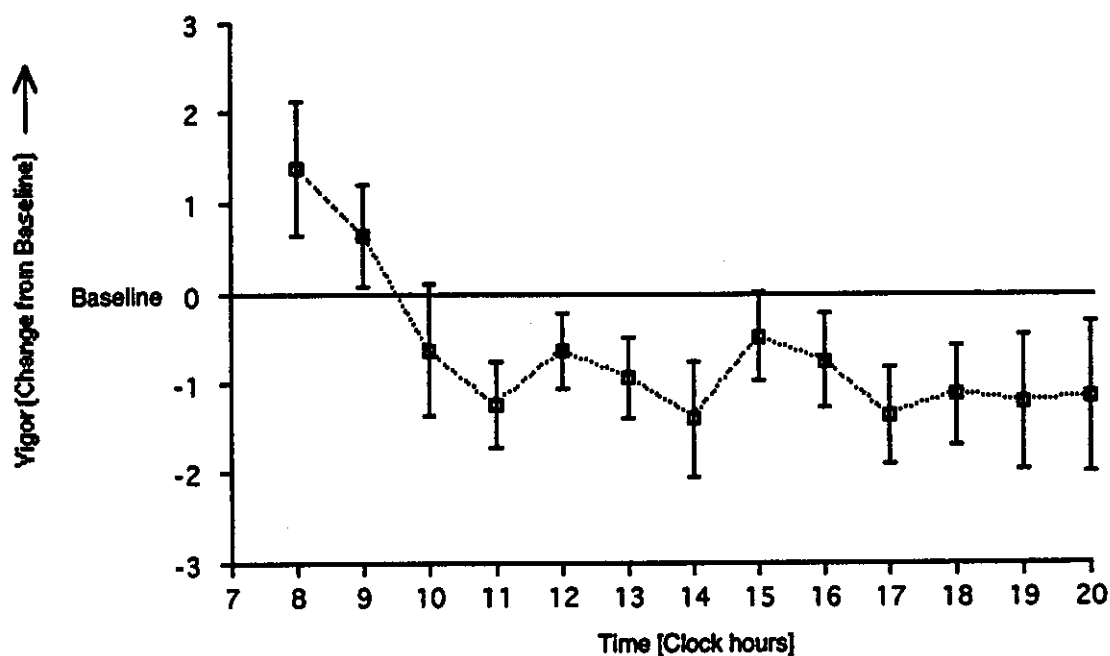
Fig. 5.23 Diurnal variation in Vigor after Sleep Reduction [n=20].

Figure 5.23 shows subjective accounts of vigor levels throughout the day. Energy levels declined sharply after awakening, falling to below baseline at 1000 hours, and remained at that level all day.

Interactions

Figure 5.24 indicates that the difference in Vigor between the WOS and NWOS groups approached significance, F = 1.69 [12, 216] p = 0.07. Following Sleep Reduction subjects in the NWOS group were adversely affected, as reflected in their reports of diminished vigor relative to both baseline and the WOS group. Subjects in the WOS group responded positively to the effects of Sleep Reduction, by exhibiting an increase in energy at least for the first 3 hours after awakening. Vigor gradually decreased thereafter. Subjects in the NWOS group reported less vigor than baseline levels for most of the day. Post-hoc analyses revealed significant differences [p < 0.05] between subjects in the WOS and NWOS groups at the following times: WOS at 0800h vs. NWOS from 1000 to 1500h and from 1600 to 1900h; WOS at 0900h vs. NWOS at 1100h.
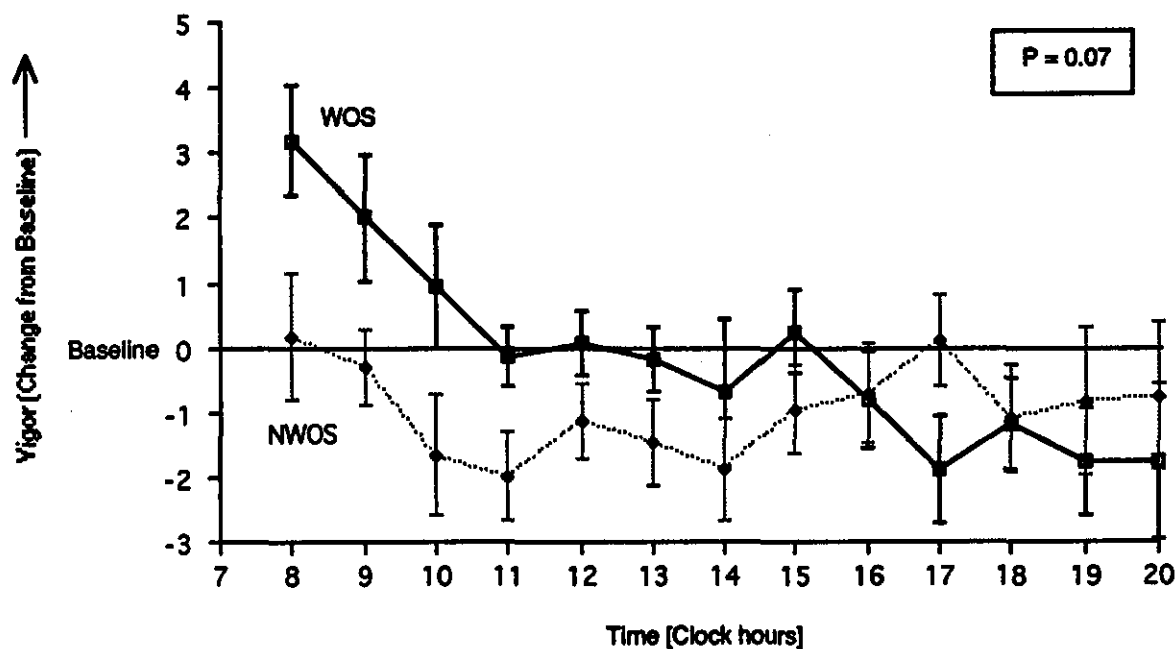
Fig. 5.24 Diurnal fluctuation in Vigor after Sleep Reduction.

c) Confusion

Main Effects

Interesting trends emerged in Confusion between the WOS and NWOS groups. Although not significant, $F = 2.25$ [1, 18] $p = 0.15$, subjects in the NWOS group described elevated confusion after Sleep Reduction whilst subjects in the WOS group reported diminished confusion relative to baseline.

Interactions

For Confusion, the interaction between WOS x Time was significant, $F = 2.42$ [12, 216] $p < 0.01$. Figure 5.25 shows that the differences between the WOS and NWOS groups were most pronounced in the first 4 hours. For the first 5 hours after arising, subjects in the WOS group were less confused after Sleep Reduction than after Baseline.
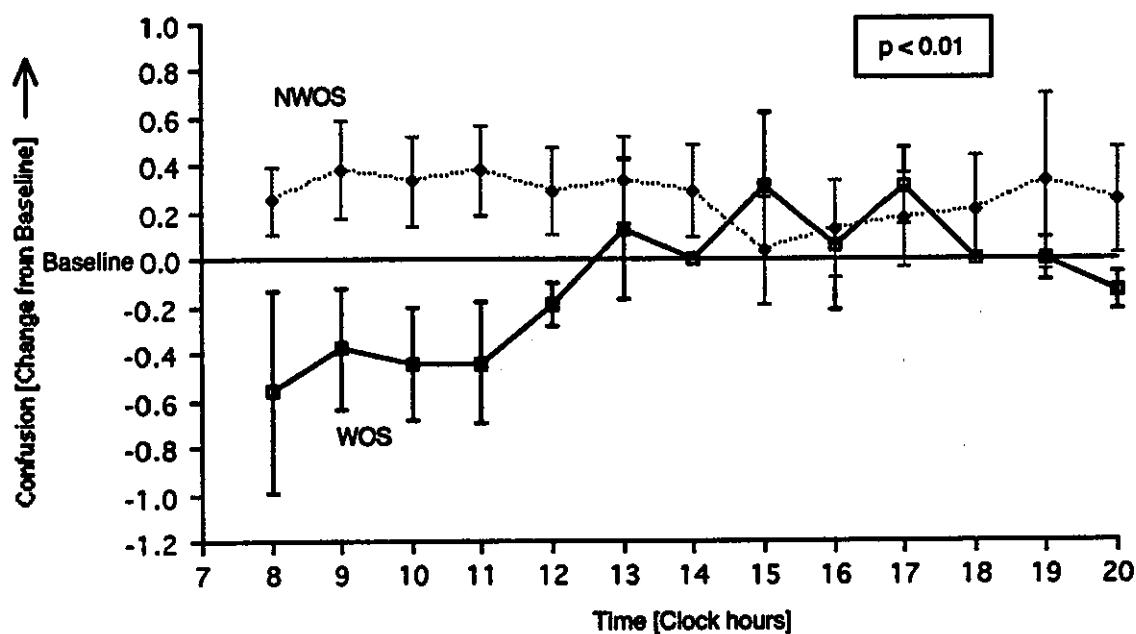
Fig. 5.25 Confusion in WOS and NWOS groups over time.

d) Depression

Although the differences were not significant, $F = 26$ [1, 18] $p = 0.12$, trends emerged between groups: Subjects in the NWOS group were in a greater depressive state than subjects in the WOS group, who in contrast, were less depressed following Sleep Reduction.

e) Tension

Main Effects

The difference in Tension between subjects in the WOS and NWOS groups approached significance, F = 3.38 [1, 18] p = 0.08. The NWOS group described increased Tension after Sleep Reduction, whilst scores improved slightly in the WOS group. Non-significant trends were evident in Tension between Introverts and Extroverts, F = 2.78 [2, 17] p = 0.09. Extroverts reported elevated tension following Sleep Reduction, whilst Introverts and Neither groups described little deviation from baseline.

Interactions

A significant interaction was observed in Tension between Impulsivity x Time, F = 1.87 [22, 187] p = 0.01. Figure 5.26 describes the fluctuating levels of tension. Subjects categorised as low impulsives reported the least tension for the first 6 hours of the day, elevating rapidly thereafter and peaking at 1700 hours. Tension in both the high impulsives and neither categories was maintained close to baseline, exhibiting slight fluctuations throughout the day.
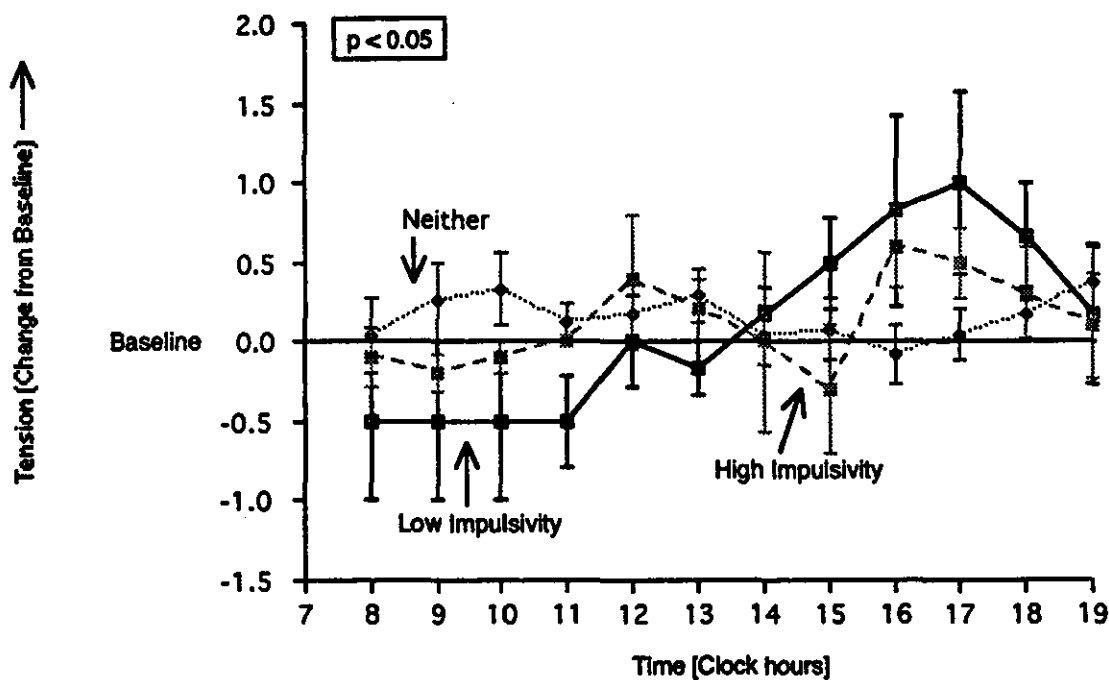
Fig. 5.26 Tension in Low and High Impulsivity after Sleep Reduction.

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Figure 5.27 displays significant interactions between Tension x Morning and Evening Types x Time, $F = 1.78$ [22, 187] $p = 0.02$. Morning types reported less tension upon awakening, levels of tension increased, peaking at 1400 hours. Evening types were slightly more tense for the first 5 hours, culminating at 1200 hours, diminishing sharply after 1200 hours to reach a nadir at 1500 hours, with a secondary peak at 1600 hours. Subjects in the Neither category exhibited little deviation from baseline until 1500 hours, before rising steadily to peak at 1900 hours.
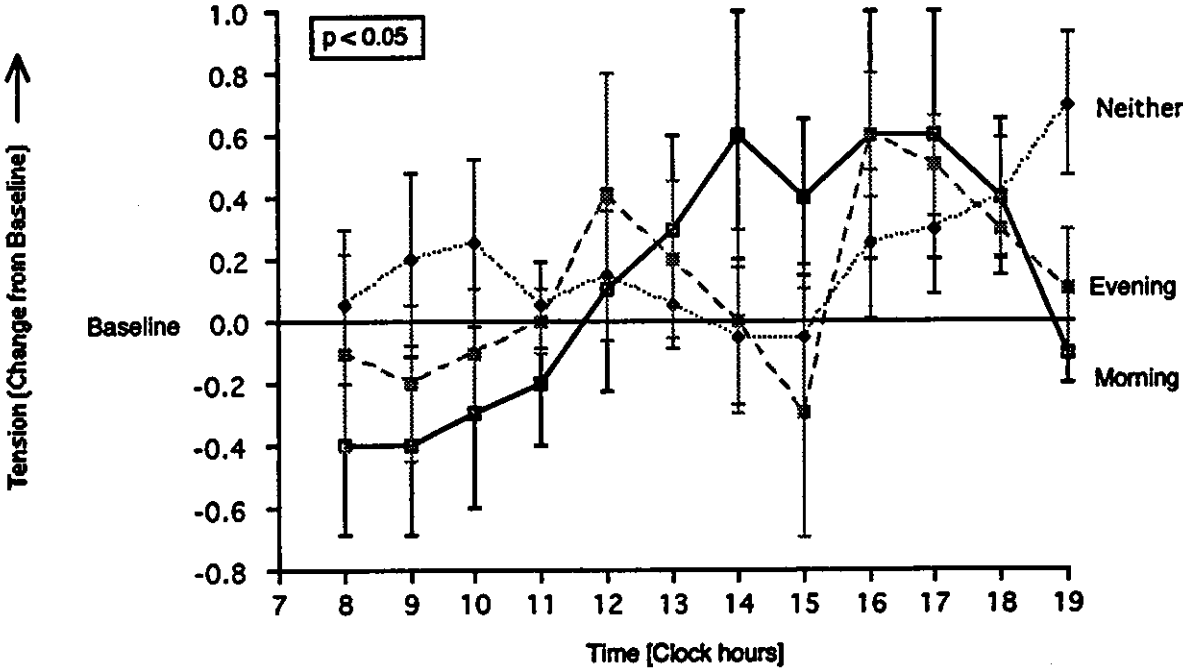
Fig. 5.27 Tension in Morning and Evening types after Sleep Reduction.

f) Anger

Main Effects

There was a significant difference in Anger between the WOS and NWOS groups, $F = 4.54$ [1, 18] $p < 0.05$. Subjects in the WOS group exhibited very little deviation from baseline. However, subjects in the NWOS group reported elevated levels of Anger following a night of Sleep Reduction [see fig. 5.28].
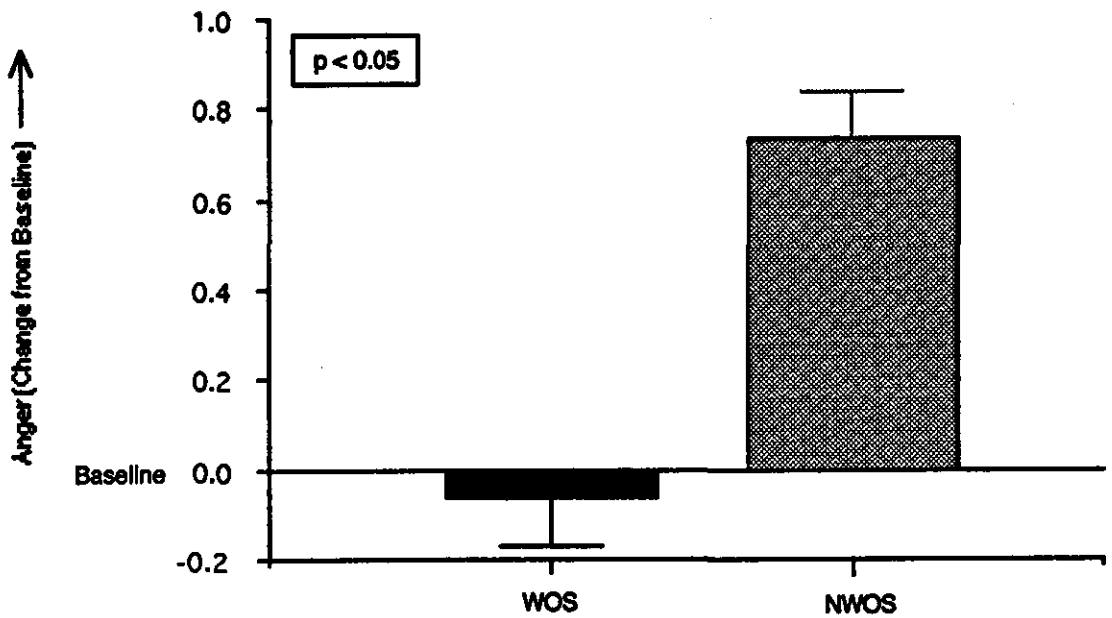
Fig. 5.28 Anger in WOS and NWOS groups after Sleep Reduction.

Summary of Results

Sleep Extension

1. Subjects adversely affected by Sleep Extension, as gauged by subjective fatigue ratings, notably for the first 4 hours after awakening, were categorised as:

  (i) Regular Sleepers

  (ii) Introverts and

  (iii) Subjects in the WOS group.

Subjects in these groups reported most fatigue after Sleep Extension. It is suggested that these characteristics act as contributory factors to exacerbate the 'wornout syndrome' following oversleeping.

2. The negative effects of Sleep Extension upon Vigor indicated the following groups to show a marked drop in energy following oversleep:

  (i) Regular Sleepers

  (ii) The WOS group

  (iii) Introverts

The first two groups were significant and the introversion group almost significant.

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3. Non-significant trends emerged in Confusion: Regular Sleepers, Morning types, the WOS group and Low Impulsives all exhibited an increase in Confusional state following Sleep Extension.

4. Similarly, for the mood variable Depression, non-significant trends pointed to subjects in the WOS and Low Impulsivity groups showing an elevated depressive state after Sleep Extension.

5. Significant differences were observed in Tension between Low and High Impulsivity groups. Subjects classified as Low Impulsive were significantly more tense following Sleep Extension. Non-significant trends indicated that the WOS group, Regular Sleepers and Introverts were in a state of greater tension.

6. In terms of Anger, a significant difference surfaced between WOS and NWOS groups, with subjects classified as WOS reporting an enhanced level of Anger following Sleep Extension.

Sleep Reduction.

1. Fatigue ratings following Sleep Reduction pointed to subjects in the NWOS group reporting enhanced fatigue, whilst the WOS group found the Sleep Reduction beneficial. These effects are the opposite to those following Sleep Extension.

2. Subjects in the WOS group exhibited increased Vigor for the first 3 hours, in contrast to the NWOS group, who exhibited adverse effects upon Vigor following Sleep Reduction.

3. Following Sleep Reduction, the NWOS group were in a significantly greater confusional state, whilst the WOS group described diminished confusion for the first 5 hours. Morning type subjects were less confused than evening types for the first 3 hours after awakening. This was expected, since morning types are known to function at their best in the early hours after awakening.

4. Subjects in the WOS group described declining depressive feelings after Sleep Reduction, whilst subjects in the NWOS group described an elevation in depression. Morning types perceived Sleep Reduction most beneficial to mood.

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5. For tension ratings, the NWOS group exhibited trends towards increased tension. Although non-significant, Morning types, Introverts and subjects classified as low Impulsives reported less tension than Evening types, Extroverts and highly Impulsive subjects.

6. Following Sleep Reduction, Anger was significantly elevated in subjects in the NWOS group relative to the WOS group.

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The above findings illustrate the diversity and complex nature of the relationship between sleep and mood. Examining subjects responses by categorising them into personality type and whether they were regular or irregular sleepers was in my view, extremely enlightening. It is evident that a number of subjects are adversely affected by oversleep: these subjects are individuals with regular sleep/wake habits, Introverts, and cautious, rather than impulsive. Interesting observations were made comparing subjects descriptions of mood states after Sleep Extension and Sleep Restriction. Whereas after Sleep Extension, subjects in the WOS group described enhanced levels of Fatigue, Anger and diminished levels of Vigor, these subjects found Sleep Restriction beneficial to mood. In fact, subjects in the WOS group reported diminished fatigue, confusion and depression, in addition to increased vigor after Sleep Reduction. Subjects in the NWOS group were mostly unaffected by Sleep Extension, but were adversely affected by Sleep Reduction. The NWOS group described increased fatigue, confusion, depression, anger and tension, alongside diminished vigor, after curtailing their sleep by 2 hours for one night. Moreover, Morning type subjects described beneficial effects upon the mood states of confusion and depression after Sleep Reduction, relative to baseline.

Assessing individual personality profiles, it was evident that subjects in the WOS group were significantly more introverted than subjects in the NWOS group. Another significant difference was observed between the groups: the WOS group were emotionally tender minded, whilst subjects in the NWOS group ranked considerably higher on the attribute toughness poise. Subjects in both groups indicated comparable levels of both anxiety and independence. Overall, subjects in the WOS group were more relaxed, timid, insecure and self-sufficient. In addition they were less shrewd and less impulsive than subjects in the NWOS group.

Sleepiness [SSS Questionnaire]

The sleepiness ratings of the 8 subjects with the 'wornout syndrome' were compared to the 12 without, following baseline sleep, extension and reduction. Subjects completed the SSS twice for each condition (1 and 2 hours after arising). Unpaired t-tests were conducted between the groups for each hour after arising.

Table 5.17 Baseline Sleep and SSS scores (mean & se)

1 hr after arising		2 hrs after arising	
WOS Group	NWOS Group	WOS Group	NWOS Group
3 (.5)	3.36 (.51)	2.5 (.19)	2.3 (.38)
t = -.5 [df. 18] p = .63		t = .47 [df. 18] p = .64	

Both the WOS and NWOS groups made virtually identical ratings of subjective alertness at each of the 2 hourly tests following baseline sleep. One hour after arising both groups rated their sleepiness around 3 [representing a relaxed, awake, responsive state]. Two hours after awakening both groups improved, 'functioning at a high level' and were 'able to concentrate'. So, under normal sleep, the SSS did not distinguish any differences between the symptomatic and control group.

Table 5.18 Sleep Extension SSS scores (mean & se)

1 hr after arising		2 hrs after arising	
WOS Group	NWOS Group	WOS Group	NWOS Group
3.1 (.3)	1.91 (.29)	2.63 (.32)	1.27 (.2)
t =2.91 [df. 18] p < .01*		t = 3.78 [df. 18] p < .01*	

Following sleep extension, significant differences emerged between the two groups at each test [see fig. 5.25]. The NWOS group reported significantly decreased levels of sleepiness (p < .01) compared to the WOS group. Interestingly, the level of sleepiness described by subjects in the WOS group remained comparable to their baseline reports, suggesting that there was no change from baseline and as such these subjects were not worn out at all. Instead, it appeared that subjects in the NWOS group found the extra sleep beneficial, as reflected by their reduction in

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sleepiness at each of the 2 hours after awakening. The NWOS group were improved by sleep extension. A similar improvement in alertness was clearly not evident in the WOS group, so on the basis of these findings, it appears that these subjects were not 'wornout' at all after sleep extension.
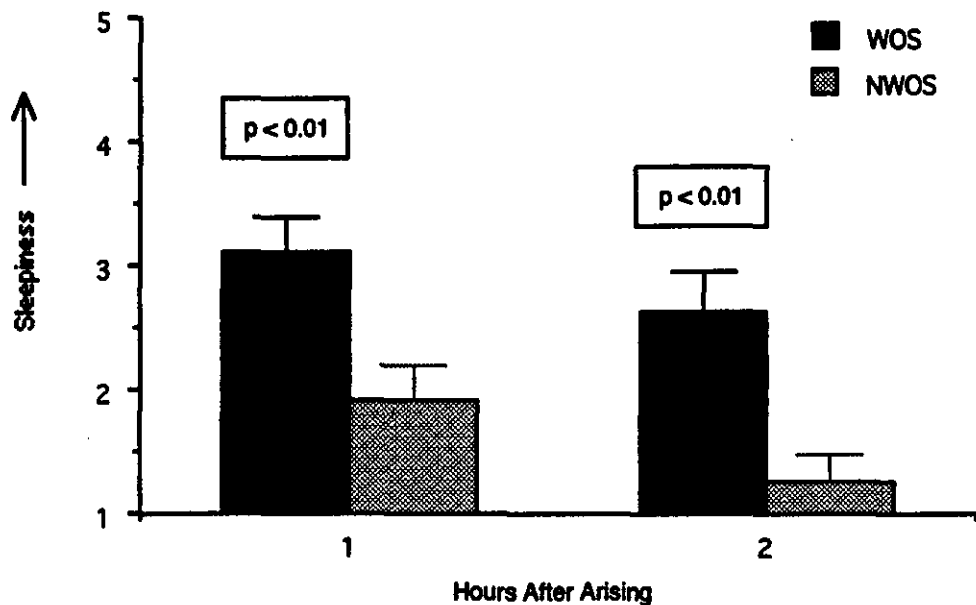
Fig. 5.25 Sleepiness [SSS ratings] after Sleep Extension.

Table 5.19 Sleep Reduction SSS scores (mean & se)

1 hr after arising		2 hrs after arising	
WOS Group	NWOS Group	WOS Group	NWOS Group
4.13 (.52)	4.36 (.56)	2.89 (.23)	3.55 (.53)
t = -.30 [df. 18] p = .77		t = -1.03 [df. 18] p = .32	

Following sleep reduction there was very little difference between the sleepiness ratings of subjects in the WOS and NWOS groups. Both groups reported greatest sleepiness [a little foggy, not at peak, let down] in the first hour after awakening. This fell to baseline levels in the WOS group in the second hour, but sleepiness in the NWOS group remained elevated.

Tiredness vs. Sleepiness

Comparing the data obtained from the SSS and the 23-item Sleepiness Questionnaire [SQ] revealed some interesting trends. If the items that comprised the 7 levels of the SSS were internally consistent, then subjects who rated themselves at Level 1 say, should endorse the corresponding items from the SQ, 100% of the time and all other items 0% of the time. A brief examination of the data indicated that this was certainly not the case. Subjects rating themselves at Level 1 on the SSS on the whole did show high endorsement levels for the corresponding items on the SQ. However they also endorsed other items associated with other levels of the SSS; thereby creating a great deal of overlap. Subjects who rated themselves at Level 3 often chose items from the SQ that were most consistent with items from scale 4 rather than 3. Further investigation revealed increased discrepancy.

It became evident that the SSS measures at least two dimensions, which I shall refer to as Sleepiness and Tiredness. Variables purporting to measure each of these dimensions were identified. Those items with highest factor loadings for each dimension were selected as follows [after MacLean, 1991]:

Sleepiness	Tiredness
Losing Interest in Remaining Awake	Let Down
Sleepiness	Slowed Down
Sleep Onset Soon	Unable to Concentrate

The main hypothesis regarding the SSS was that subjects experiencing the 'wornout syndrome' following oversleep would select more items from the 'tiredness' category than the 'sleepiness' category. Subjects were already identified as those experiencing the 'wornout syndrome' following sleep extension [N=8] and those who did not [N=12].

The responses on the 23-item Sleepiness Questionnaire were examined for the first 4 hours upon awakening following sleep extension and restriction. The number of subjects checking any of the items in the 2 categories [sleepiness and tiredness] was noted and Chi-square analysis was conducted on the actual scores, comparing subjects' scores on the Tiredness and Sleepiness scales for each hour after awakening. The findings will be presented for baseline, sleep extension and sleep reduction conditions.

Baseline Sleep.

Chi-Squared analysis was conducted to investigate significant differences in the selection of 'Tiredness' and 'Sleepiness' items between WOS and NWOS groups. Following Baseline sleep, subjects in the WOS group selected more 'Tiredness' than 'Sleepiness' items 2, 3 and 4 hours after arising, although the differences were not significant.

Similarly, there were no significant differences in the selection of 'Sleepiness' and 'Tiredness' items by subjects in the NWOS group, although trends indicated that for the first 3 hours after arising, more items were selected from the Tiredness category.

Figures 5.26 and 5.27 plots the selection of descriptors ['tiredness' and 'sleepiness'] made by subjects in the WOS and NWOS groups. For the first hour after arising from Baseline Sleep, subjects in the NWOS group selected twice as many items from both the Tiredness and Sleepiness categories than the WOS group. At subsequent tests (hour 2, hour 3) the number of items selected by members of both the WOS and NWOS groups was very similar.
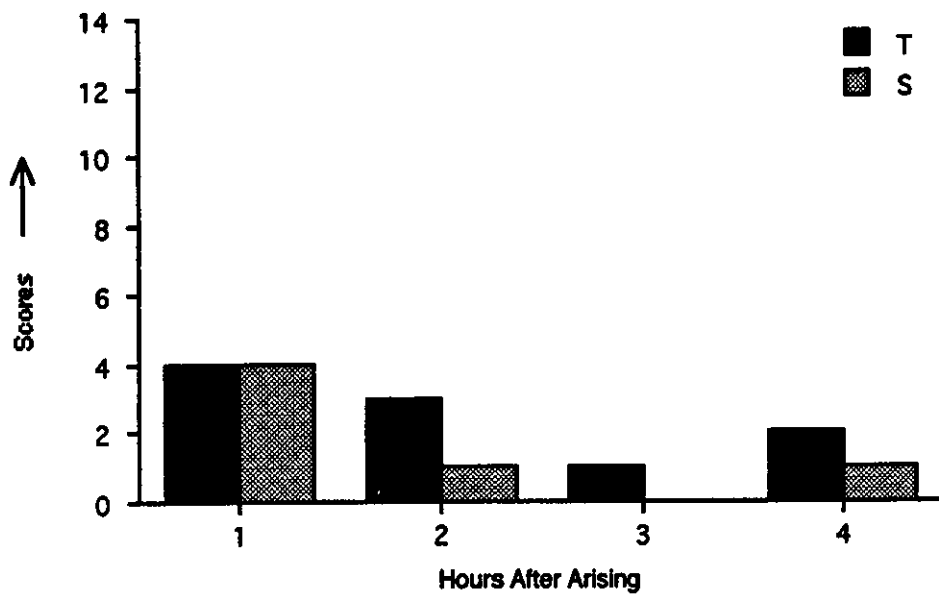
Fig. 5.26 Number of 'Tiredness' and 'Sleepiness' items selected by subjects in the WOS group after Baseline Sleep.
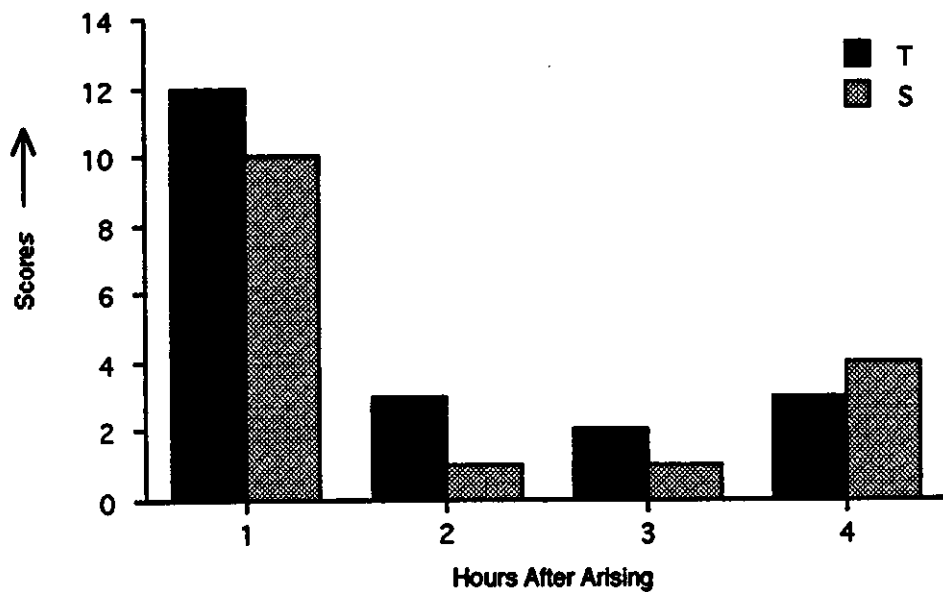
Fig. 5.27 Number of 'Tiredness' and 'Sleepiness' items selected by subjects in the NWOS group after Baseline Sleep.

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Sleep Extension

Following Sleep Extension, the WOS group selected more 'Tiredness' items than 'Sleepiness' items at all 4 hours tested [fig. 5.28]. Results of the Chi-Squared analysis revealed that these differences approached significance at hour 2, Chi-square = 3.6 [df. 1]. In addition, at hour 1, the NWOS group selected more items from the 'Tiredness' category, although the difference was not significant.

Overall, the WOS group were reportedly more tired than the NWOS group after Sleep Extension. More 'sleepiness' items were also selected by subjects in the WOS group. However, comparing Figures 5.28 and 5.29, it was evident that the WOS group had selected more items from the 'Tiredness' category.

Compared to Baseline, the WOS group selected more items from both the Tiredness and Sleepiness categories. However, subjects in the NWOS group selected fewer items from both categories for the first 2 hours after arising; thereafter the number of items selected was comparable across conditions. These findings indicate that subjects in the NWOS category found the additional sleep beneficial to mood, this was reflected in their accounts of diminished levels of both Tiredness and Sleepiness after sleep extension.
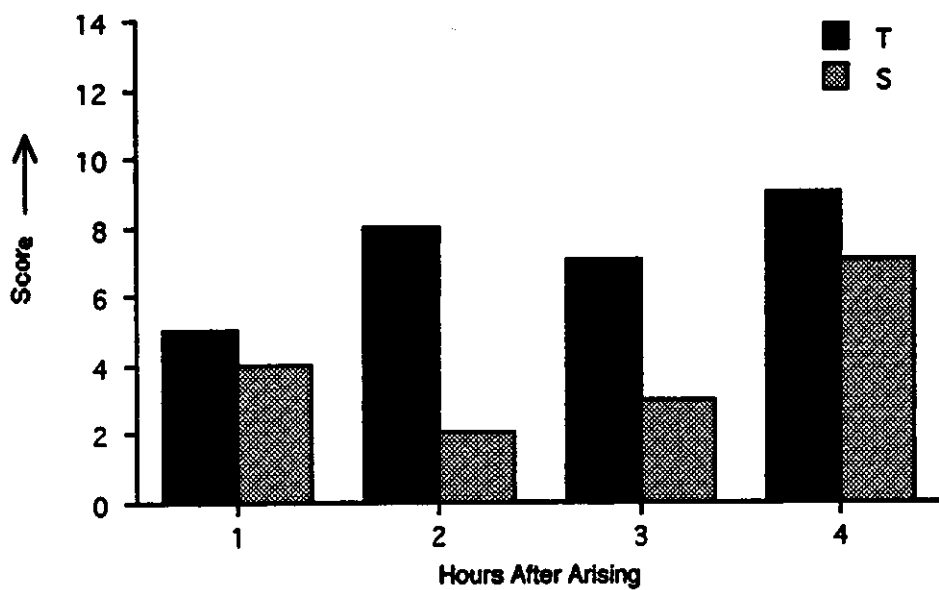
Fig. 5.28 Number of 'Tiredness' and 'Sleepiness' items selected by subjects in the WOS group after Sleep Extension.
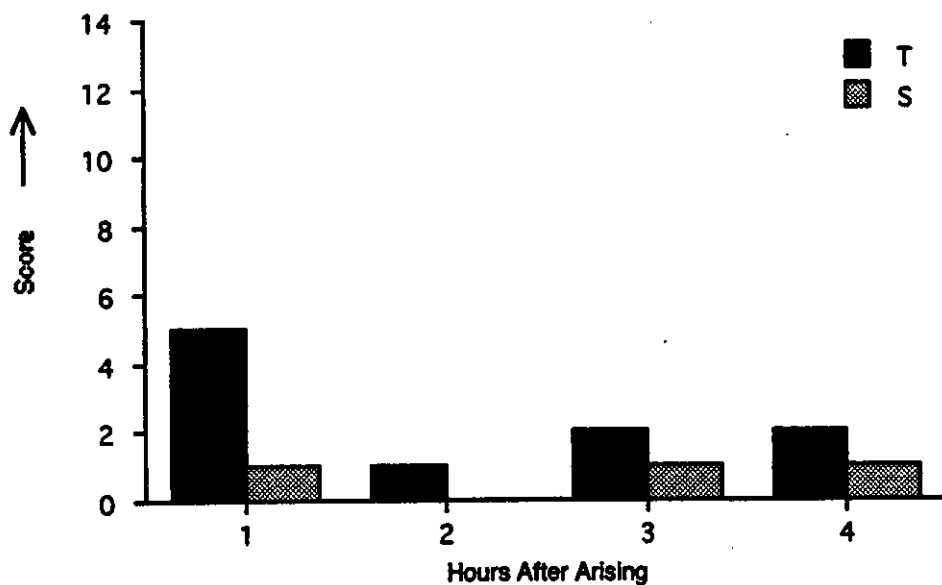
Fig. 5.29 Number of 'Tiredness' and 'Sleepiness' items selected by subjects in the NWOS group after Sleep Extension.
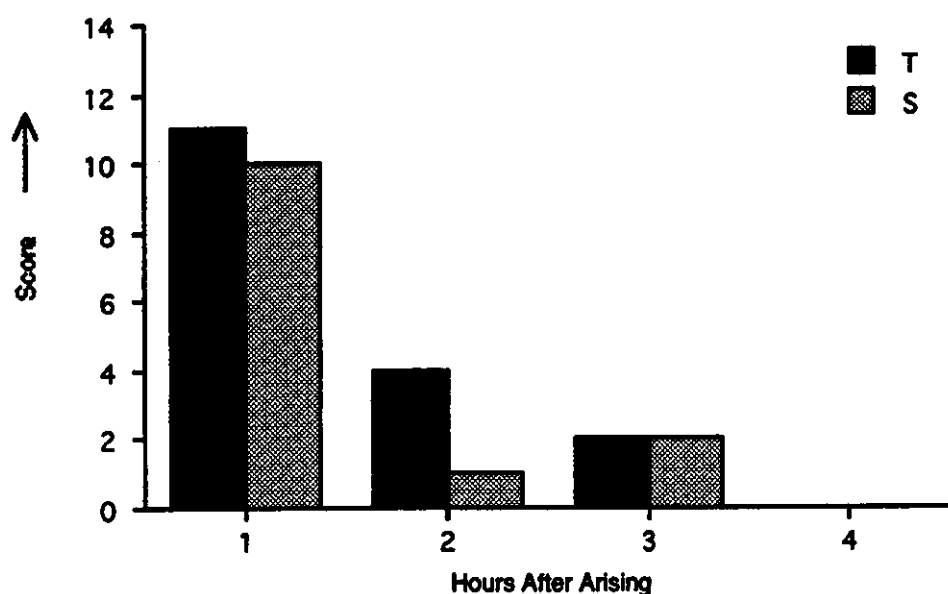
Fig. 5.30   Number of 'Tiredness' and 'Sleepiness' items selected by subjects in the WOS group after Sleep Reduction.
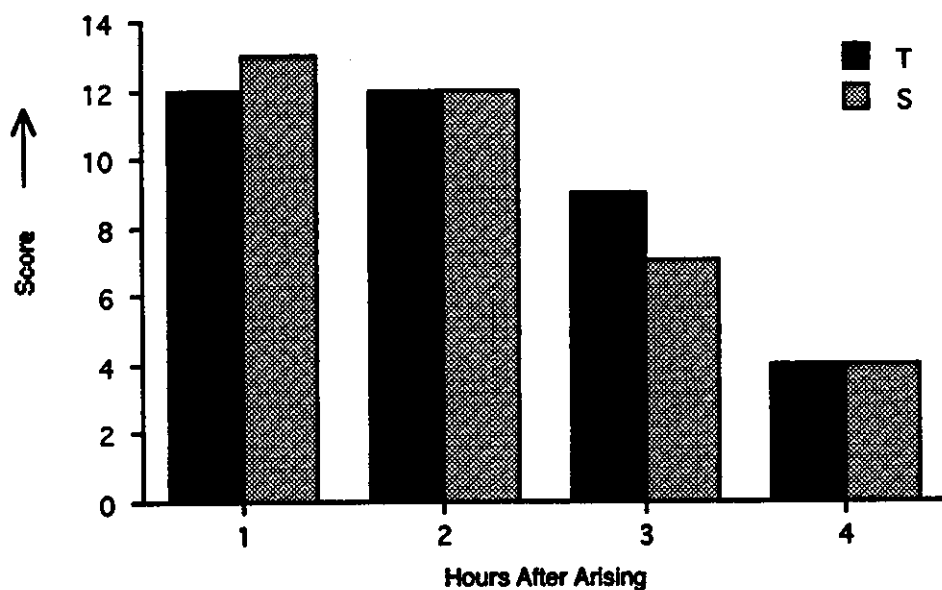
Fig. 5.31   Number of 'Tiredness' and 'Sleepiness' items selected by subjects in the NWOS group after Sleep Reduction.

Following Sleep Reduction, the NWOS group described similar levels of tiredness and sleepiness for all 4 hours tested. The WOS group described similar levels to the NWOS group for hour 1, but both Tiredness and Sleepiness diminished rapidly thereafter for the WOS group. The decline was more gradual for subjects in the NWOS group [see figs. 5.30 and 5.31]. Although certain trends were evident, Chi-square failed to yield significant findings.

Discussion

It is argued that subjects identified as experiencing the 'wornout syndrome' following Sleep Extension were selecting more items pertinent to the 'Tiredness' scale than to the 'Sleepiness' scale. If subjects were genuinely sleepy following Extension, one would expect ratings to be biased towards the 'Sleepiness' dimension. Instead, items pertaining to 'Tiredness' were predominantly endorsed. These results support the hypothesis that the SSS is not an unidimensional measure and that following Sleep Extension subjects felt more *fatigued* than sleepy.

After Baseline sleep, identical numbers of Sleepiness and Tiredness items were selected at hour 1. However, by hours 2 and 3, more Tiredness items were selected by subjects in the WOS group. Comparing these results to those after Sleep Extension reveals interesting findings: Although similar numbers of Tiredness and Sleepiness items were selected by the WOS group at hour 1, by hours 2 and 3, these differences were greatly enhanced.

Following Sleep Reduction subjects in the NWOS group selected more items from both the Tiredness and Sleepiness categories than subjects in the WOS group [see figs. 5.30 and 5.31]. Subjects in the NWOS group selected similar number of Tiredness and Sleepiness items throughout the 4 hours tested. Overall, the clinical group [WOS] were less affected by Sleep Reduction.

Personality

Individual personality profiles were constructed using Cattell's 16PF. Subjects were allocated scores between 1 and 10 for the categories of Extroversion, Anxiety, Toughness Poise and Independence. Those with scores between 1-3 and 8-10 were considered to be extreme, the remainder with scores between 4-7 were moderate. Independent t-tests were performed for each personality variable in turn, comparing the WOS and NWOS group means.

i. Extroversion

There were significant differences in personality between the WOS and NWOS groups, $t = -2.5$ [df.17] $p = 0.02$. Subjects with the WOS were more inclined towards introversion whilst the NWOS group tended towards Extroversion.

Fig. 5.32 Extroversion in the WOS and NWOS Groups.

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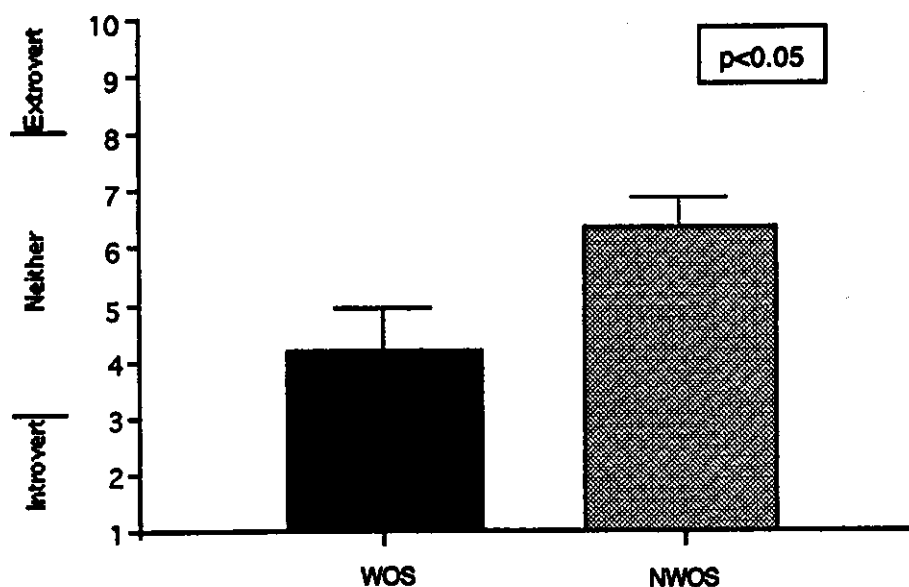
II. **Toughness Poise**

There was a significant difference between the groups, $t = -2.79$ [df.17] $p = 0.01$. The WOS group were emotionally tender minded, compared to the NWOS group who were more tougher.

Fig. 5.33 Toughness Poise in WOS and NWOS Groups.
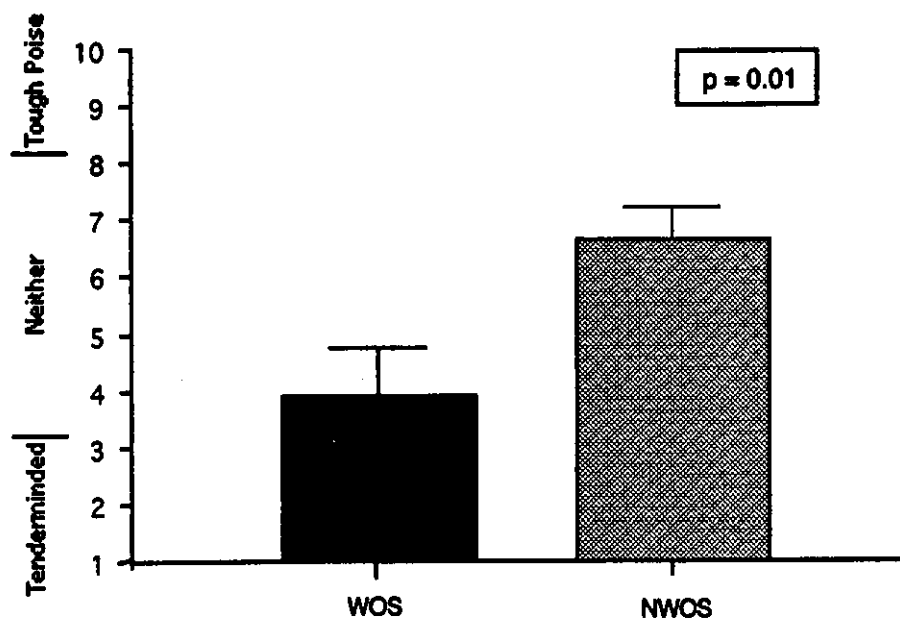
III. **Anxiety**

Subjects in both the WOS and NWOS groups were moderately anxious. There was very little difference in anxiety levels between the groups.

Table 5.20 Anxiety scores in WOS and NWOS groups.

WOS Group Mean [se]	NWOS Group Mean [se]
6.24 [.65]	6.16 [.62]
$t = 0.08$ [df. 17] $p = 0.94$	

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iv. Independence

Subjects in both the WOS and NWOS groups were comparable in levels of independence, and not significantly different.

Table 5.21 Independence scores in WOS and NWOS groups.

WOS Group Mean [se]	NWOS Group Mean [se]
6.1 [.69]	6.58 [.67]
t = -0.49 [df. 17] p = 0.63	

Discussion

My primary concern in this study was to investigate whether subjects experiencing the 'wornout syndrome' differed in personality from those unaffected by oversleep. The personality profiles of the WOS and NWOS groups were compared. Overall, subjects experiencing the 'wornout syndrome' were more relaxed, timid and insecure. They were also more self-sufficient, less shrewd and less impulsive than the NWOS group. There was little difference between the groups in intelligence, emotional stability, imagination and radicalism. However, due to the small sample size, it must be acknowledged that factors can become confounded. In the present study, the number of females [N = 16] far exceeded the number of males [N = 4]. Therefore, gender differences were not examined in the present analysis. However, results must be interpreted with caution, due to the confounding nature of such factors. For instance, present findings have indicated that WOS subjects tended towards introversion. But, due to the small sample size in this study, it could be argued that WOS subjects tend to be female.

In the present study, subjects were also categorised as regular or irregular sleepers. Irregular sleepers were defined as those whose times of awakening and retiring varied by more than 2 hours in the baseline week. Findings revealed that regular sleepers experienced the ill-effects of oversleeping, whilst irregular sleepers were generally unaffected. Taub [1978] studied mood in irregular sleepers and controls and observed that negative affect (deactivation-sleep, depression, general deactivation, inert-fatigued) was significantly greater and positive mood states (cheerful, energetic, general activation) significantly less in the irregular sleepers.

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A substantial body of evidence [e.g. Eysenck, 1978] suggests that discrete variations in the personality trait of extroversion are associated with individual differences in the way basic cognitive processes are carried out. Extroversion consists of several closely associated traits. These include impulsivity, sociability, adventurousness, enthusiasm, high activity level, and boredom proneness. More recently, researchers [e.g. Dickman, 1990] have attempted to identify the specific characteristics of extroverted individuals that account for the overall relationship between extroversion and cognitive functioning. This research suggests that impulsivity is the component of extroversion that is most consistently associated with individual differences in the way basic perceptual and memorial processes are executed [Dickman and Meyer,1988]. Dickman defined impulsivity as the "tendency to deliberate less than most people of equal ability before taking action". Dickman distinguished between functional impulsivity which was associated with positive consequences, where the individual benefits from his impulsive behaviour, from dysfunctional impulsivity, which in most cases had negative consequences. One finding of particular interest was that subjects experiencing the 'wornout syndrome' were less impulsive than the other group. Impulsivity may be viewed as one factor of Tough Poise [a second-order factor in the 16PF], in general, the WOS group presented lower scores. According to Cattell, subjects scoring high on Tough Poise [>5.5] are likely to be enterprising, decisive and resilient, but are likely to miss the subtle relationships of life and tend to orient their behaviour towards the obvious. When faced with difficulties they are likely to involve rapid action with insufficient consideration and thought. This trait corresponds with Dickman's 'dysfunctional impulsivity'.

The mean score on Tough Poise for subjects experiencing the 'wornout syndrome' was 7.4, subjects in the NWOS group achieved a maximum score of 10. Dickman suggested that Impulsivity was one dimension of extroversion. This relationship was observed in this study; subjects experiencing the 'wornout syndrome' tended towards Introversion [score = 4.2] whilst subjects in the NWOS group exhibited traits characteristic of extroversion [score = 6.4]. Fatigue scores of morning types were compared to evening types. These results revealed expected trends — owls reported elevated fatigue in the morning and larks, in the evening.

6

Sleep and Mood: The Influence of Personality and Individual Differences

6. Study 3

Sleep and Mood: The Influence of Personality and Individual Differences.

Results of the previous study highlighted several areas which required further investigation. It became apparent that personality and individual differences were of vital importance in understanding the adverse effects of oversleep. I decided to pursue this area, and to focus upon specific individual traits which predispose subjects to the 'wornout syndrome'.

Results from the previous study suggested that the following personality characteristics would predispose subjects to the 'wornout syndrome' following oversleep:

    (i)   Introversion
    (ii)   Low Impulsiveness
    (iii)   Emotional Tender-Mindedness
and (iv)   Morningness

It was also hypothesised that individuals with the following traits would not experience any adverse effects of oversleeping:

    (i)   Extroversion
    (ii)   High impulsiveness
    (iii)   Toughness poise
and (iv)   Eveningness

A new study was thus designed to compare the effects of oversleep and sleep restriction in individuals falling into the above personality categories. The aim of this study was to establish the importance of personality in relation to mood states, following manipulations in sleep length. Therefore, only subjects categorised as 'extreme' in the above personality traits were selected for the study. All subjects recruited were healthy sleepers, maintaining regular sleep/wake schedules. The results of the previous study contributed to framing this experiment.

Subjects

i) Initial Screening

Subjects were asked to complete a General Sleep and Health Questionnaire. Healthy subjects, sleeping regularly at nightime and refraining from daytime nap-taking were recruited. All volunteers were asked to complete the following questionnaires:-

1. Cattell's Sixteen Personality Factor [16PF].
2. Horne and Ostberg's Morningness-Eveningness Questionnaire [MEQ].
3. Eysenck's Personality Inventory [EPI].

ii). Final Selection

Subjects were selected if they were regular sleepers and fell into one or more of the following groups:-

1.   Introvert [< 5.5 on the 16PF]
2.   Extrovert [> 7.2 on the 16PF]
3.   Low Impulsive [< 5.0 on the EPI]
4.   High Impulsive [> 7.0 on the EPI]
5.   Low Tough Poise [< 5.5 on the 16PF]
6.   High Tough Poise [> 7.0 on the 16PF]
7.   Morning type [> 56 on the MEQ]
8.   Evening type [< 35 on the MEQ]

Fifty subjects were screened and thirty four volunteers passed the selection criteria and were recruited. There were 17 males and 17 females with an average age of 19.7 years. Subjects were placed in the appropriate categories [e.g. extroversion] if they exhibited extreme scores [as specified above]. Subjects were eligible to be categorised into as many personality groupings as were appropriate [e.g. subject 3 was categorised as introvert, morning type and low tough poise]. Table 6.1 only tabulates the extreme groupings and not subjects classified in the 'neither' groups. Although subjects were only selected if they were 'extreme' types, they could still fall into the 'neither' category for other personality groupings.

Personality

Table 6.1 displays subjects' personality profiles.

Category	Number of Subjects	Males Number [%]	Females Number [%]
Low Impulsivity	15	4 [27]	11 [73]
High Impulsivity	11	8 [73]	3 [27]
Low Tough Poise	12	7 [58]	5 [42]
High Tough Poise	11	7 [64]	4 [36]
Introvert	15	6 [40]	9 [60]
Extrovert	11	9 [82]	2 [18]
Morning Type	4	1 [25]	3 [75]
Evening Type	4	3 [75]	1 [25]

Table 6.2 below shows the degree of overlap amongst different conditions [e.g. two subjects were classified as both introverts and as morning types, and eight introverts were also entered in the category of low tough poise].

Table 6.2 Subjects Personality Characteristics [N=34].

	Introvert	Extrovert	Morning Type	Evening Type	Low Tough Poise	High Tough Poise	Low Impulsive	Highly Impulsive
Introvert			2	1	8	4	10	1
Extrovert			1	1	2	6	2	6
Morning Type	2	1			1	1	3	1
Evening Type	1	1			1	2	1	3
Low Tough Poise	8	2	1	1			5	3
High Tough Poise	4	6	1	2			6	3
Low Impulsive	10	2	3	1	5	6		
Highly Impulsive	1	6	1	3	3	3		

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Procedure

The procedure employed was analogous to that used in the previous study.

Phase One

During Phase One subjects were required to wear a wrist actometer and to complete a Sleep Diary for a one-week period to determine their habitual sleep/wake patterns.

Phase Two

During Phase Two subjects were required to undergo two experimental nights: one sleep extension [+2h from baseline] and one reduction [-2h from baseline] in addition to two baseline nights.

A counterbalanced design was employed so that while some subjects underwent the Sleep Extension condition first, others partook in Sleep Reduction. Subjects were randomly assigned to either condition. An interval of one-week elapsed between experimental conditions [the experiment was always conducted on the same night each week].

Following each experimental night these questionnaires were administered throughout the day :-

1. Mood Questionnaire - hourly until bedtime
2. Sleepiness Questionnaire - hourly until 1700 hours
3. SSS - hourly until 1700 hours
4. Post-Sleep Questionnaire - 30 minutes after arising in the morning

Results

Post-Sleep Questionnaire

The Post-Sleep Questionnaire was administered to monitor subjects sleep onset and awakening times as well as to ensure that there were no reported sleep problems. Table 6.3 displays the mean sleep onset and awakening times across all conditions for the 34 subjects.

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Table 6.3 Mean [with se] Sleep Onset and Awakening Times.

	Baseline (normative)	Extension (+2hrs)	Reduction (-2hrs)
Sleep Onset	0030h [0.11]	0030h [0.14]	0030h [0.11]
Time of Awakening	0800h [0.06]	0935h [0.12]	0620h [0.14]

All 34 subjects normally averaged 7.5 hours of sleep on baseline nights. On experimental days the average time of awakening for sleep extension was 0935h and for sleep reduction, 0620h. The variance [se] for both sleep onset and time of awakening was comparable across all 3 conditions.

The Post-Sleep Questionnaire probed subjective quality of sleep. One-way Analyses of Variance were computed for each of the 4 items [see Table 6.4]. Significant findings were further subjected to Post-hoc analyses using Tukeys HSD test.

Table 6.4 Mean [with se] ratings of subjective Sleep Quality.

Five-point Scale	Baseline	Extension	Reduction	1-way ANOVA
Difficulty in getting to sleep 2.0=Harder than usual 3.0=Same as usual 4.0=Easier than usual	2.9 [0.15]	2.93 [0.19]	3.0 [0.14]	p = 0.38 F = 0.98 (2, 87)
Restlessness during sleep 2.0=More restless than usual 3.0=As usual 4.0=Less than usual	2.95 [0.11]	2.9 [0.18]	2.93 [0.2]	P = 0.98 F = 0.02 (2, 87)
Quality of sleep obtained 2.0=Better than normal 3.0=Normal 4.0=Worse than normal	2.9 [0.13]	2.9 [0.14]	2.97 [0.16]	p = 0.93 F = 0.07 (2, 87)
Difficulty in getting up 2.0=Difficult 3.0=Moderate 4.0=Easy	2.77 [0.1]	3.6 [0.18]	1.83 [0.17]	p < 0.0001 * F = 31.7 (2, 87)

There were no significant differences between the groups in either difficulty getting to sleep, restlessness during sleep or quality of sleep obtained. All subjects reported quality of sleep analogous to normal sleep. This was presumed, since subjects were not expected to differ here.
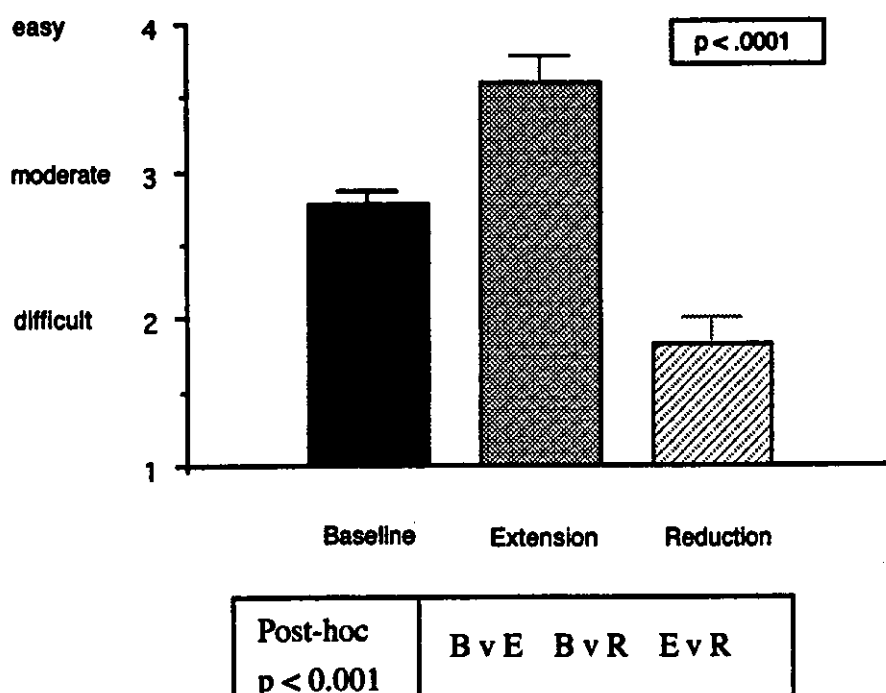
Fig. 6.1 Difficulty In Getting Up

A significant difference emerged between the sleep conditions in terms of 'difficulty getting up in the morning', F = 31.7 [df.2, 87] p < 0.0001. Following Sleep Extension, subjects found it significantly easier to get up than either Baseline and Sleep Reduction. By contrast, Sleep Reduction increased difficulty in getting up compared to both Baseline and Sleep Extension [see fig. 6.1]. Post-hoc tests revealed significant differences between the conditions, p < 0.001.

The 'Wornout Syndrome'.

Those subjects with fatigue values > 0 (baseline) for the first four hours following awakening after sleep extension were classified as 'wornout syndrome'. There were 17 subjects in each of the WOS and NWOS groups.

Personality

Table 6.5 Personality profiles of subjects in the 'wornout syndrome' and 'non wornout syndrome' groups.

Category	WOS No. of Subjects	%	NWOS No. of Subjects	%
Morning type	2	11.8	2	11.8
Neither Morning nor Evening type	15	88.2	11	64.6
Evening type	0	0	4	23.6
Low Impulsivity type	11	64.7	4	23.6
Neither Low nor high Impulsivity type	5	29.4	3	17.6
High Impulsivity type	1	5.9	10	58.8
Introvert	11	64.7	4	23.6
Neither Introvert nor Extrovert	5	29.4	3	17.6
Extrovert	1	5.9	10	58.8
Tender minded Emotionality	10	58.8	2	11.8
Neither Tender minded nor Tough	4	23.5	7	41.2
Toughness Poise	3	17.6	8	47.0
Male	5	29.4	12	70.6
Female	12	70.6	5	29.4

Independent t-tests were conducted for subjects in the WOS and NWOS groups for each personality factor in turn.

i. Extroversion

There was a significant difference between the WOS and NWOS groups in terms of Extroversion, $t = -5.09$ [df. 32] $p < 0.0001$. Subjects in the WOS group were verging on Introversion, whilst subjects in the NWOS group were bordering on Extroversion.

Fig. 6.2 Extroversion scores in WOS and NWOS Groups.
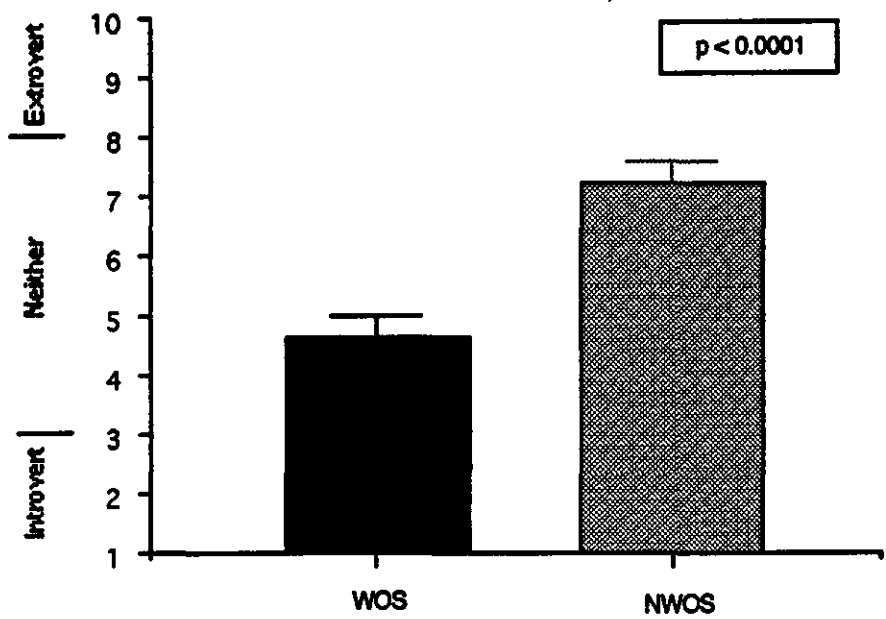
ii. Anxiety

Although not significant, the WOS group indicated trends towards increased anxiety compared to subjects in the NWOS group.

Table 6.6 Anxiety scores in WOS and NWOS groups.

WOS Group Mean [se]	NWOS Group Mean [se]
6.33 [.38]	5.36 [.4]
t = 1.74 [df. 32] p = 0.09	

iii. Toughness Poise

Although the difference in Toughness poise between subjects in the WOS and NWOS groups was not significant, the NWOS group mean was greater, indicating that these subjects were tougher than those in the WOS group.

Table 6.7 Toughness Poise scores in WOS and NWOS groups.

WOS Group Mean [se]	NWOS Group Mean [se]
5.32 [.54]	6.72 [.62]
t = -1.71 [df. 32] p = 0.1	

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iv. Independence

There was a significant difference between the WOS and NWOS groups, t = -2.16 [df. 32] p < 0.05. Although both groups were encompassed in the 'neither' category, subjects in the NWOS group were more independent than those in the WOS group. The WOS group were intermediate, not dependent or independent, whilst the NWOS group verged towards independence.

Fig. 6.3 Independence scores In WOS and NWOS groups.
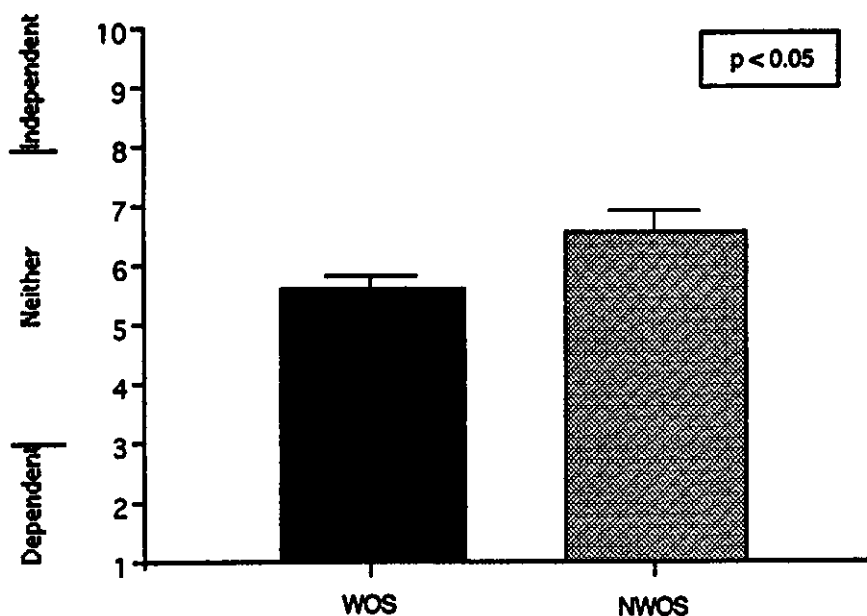
To summarise the findings pertaining to personality, subjects in the 'wornout syndrome' group were: a) neither morning nor evening type, b) not impulsive, c) emotionally tender minded, d) introverts and e) predominantly female.

Analysis of Mood

As the conditions were counterbalanced, data was examined for any order effects between conditions. There were no transfer effects and data was thus pooled for analysis.

A. Hourly mood scores were obtained for each of the six dimensions of affect: Fatigue, Vigor, Confusion, Depression, Anger and Tension. Two-way ANOVA's were performed separately for each mood dimension comparing the scores after

Baseline, Sleep Extension and Sleep Reduction for all subjects together. The Between-Group factor was sleep length [3 levels] and the Within-Group factor was time [14 hours]. The results of this analysis comparing mood scores after Baseline, Sleep Extension and Sleep Reduction for all 20 subjects will be presented first.

B. Hourly scores were obtained for each of the six dimensions of mood: Fatigue, Vigor, Confusion, Tension, Anger and Depression. For each subject, mean baseline scores were calculated. The scores following sleep extension and restriction were subtracted from baseline scores. To see whether personality traits had an effect on mood following sleep manipulations [extension, reduction], separate ANOVAS were conducted. For example, fatigue ratings were compared for introversion and extroversion after both sleep extension and sleep reduction. Two-way ANOVA's were performed for each personality variable in turn following sleep extension and restriction. Posthoc comparisons using the Tukey HSD test were conducted for each factor.

The above comparisons were made using clock time (e.g. 0700, 0800 etc. to 2200h) and real time (e.g. 0, 1, 2 –14 hours after arising) data. This was done in order to account for any circadian variations arising and also because following reduction, no comparable baseline data was available for the first 2h (since subjects in the baseline condition awoke 2h after those in sleep reduction).

Analyses of Variance were performed for the first 4 hours after arising, since this time period was most important in terms of the 'wornout syndrome'. Analyses were also conducted for 'all time' data, but since significant findings were only evident in the first 4 hours, only this data will be presented for Sleep Extension and Sleep Reduction data [B].

A. Sleep Length and Mood

a) Fatigue

Main Effects
The difference in fatigue between the conditions approached significance, $F = 3.0$ [2, 99] $p = 0.05$. Post-hoc analysis revealed that subjects were significantly more fatigued after Sleep Reduction compared to Baseline, $p < 0.05$. Fig 6.4 illustrates the findings. Although the difference in fatigue after Sleep Extension was not significant, trends emerged indicating that subjects reported enhanced fatigue, compared to Baseline.

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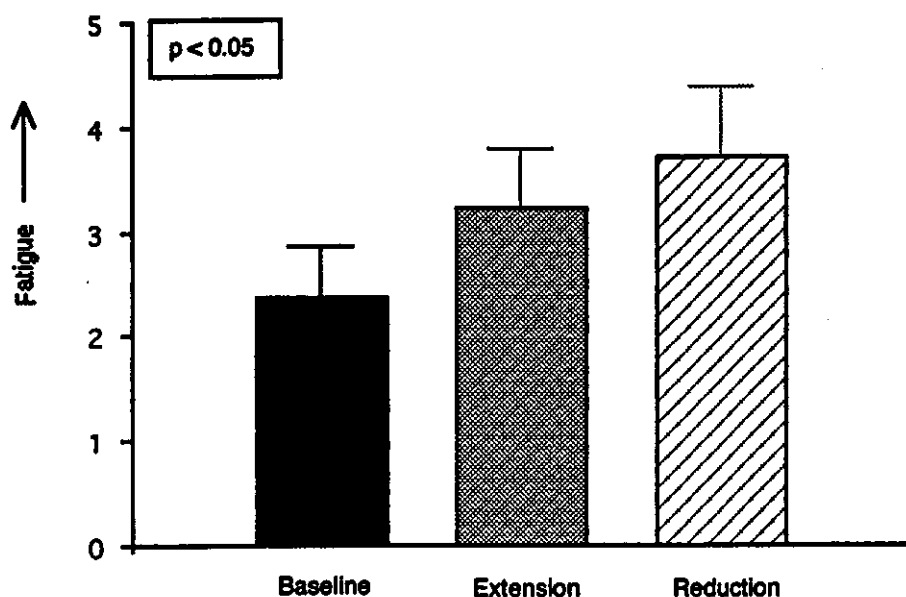
Fig. 6.4 Fatigue scores after Baseline, Sleep Extension and Sleep Reduction in all 20 subjects.

Interactions

Interactions between conditions x time were highly significant, $F = 6.84$ [26, 1287] $p < 0.001$. Fig. 6.5 indicates that following Sleep Reduction, subjects were significantly more fatigued than Baseline or Sleep Extension for the first 5 hours after awakening. Although subjects were more fatigued after Sleep Reduction than after Sleep Extension, it was evident that following Sleep Extension subjects were significantly more fatigued compared to Baseline. Post-hoc analysis revealed significance [$p < 0.05$] between Baseline and Sleep Reduction at hours 1, 2, 3, 4 and 5. Following Sleep Extension, subjects were significantly more fatigued than Baseline 1, 2, 3, 4, 5 and 14 hours after awakening.

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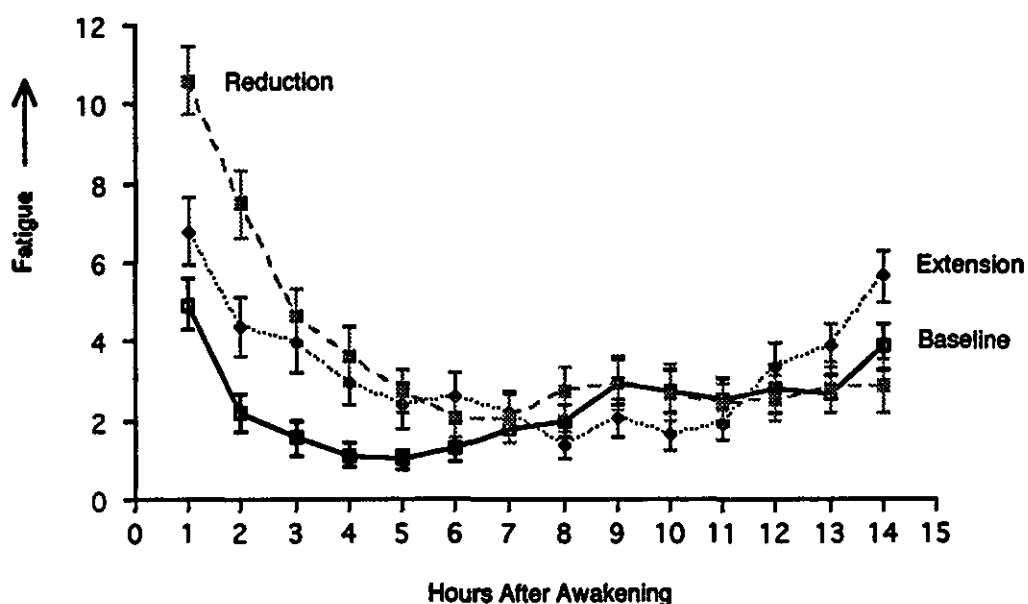
Fig. 6.5 Interaction of Fatigue scores after Baseline, Sleep Extension and Sleep Reduction x Time in all 20 subjects.

b) Vigor

Vigor was not significantly different across conditions, $F = 2.18$ [2, 99] $p = 0.12$. Significant interactions were observed between the conditions x time, $F = 4.32$ [26, 1287] $p < 0.001$. Fig. 6.6 illustrates the findings. Following Sleep Extension subjects were less energetic than Baseline at hours 2, 3, 12, 13 and 14. After Sleep Reduction, subjects were significantly less energetic than Baseline for the first 5 hours after awakening and at hour 8. Overall, subjects reported least energy after Sleep Reduction, post hoc analyses reported significant differences in Vigor between Sleep Extension and Sleep Reduction at hours 1, 2 and 14.

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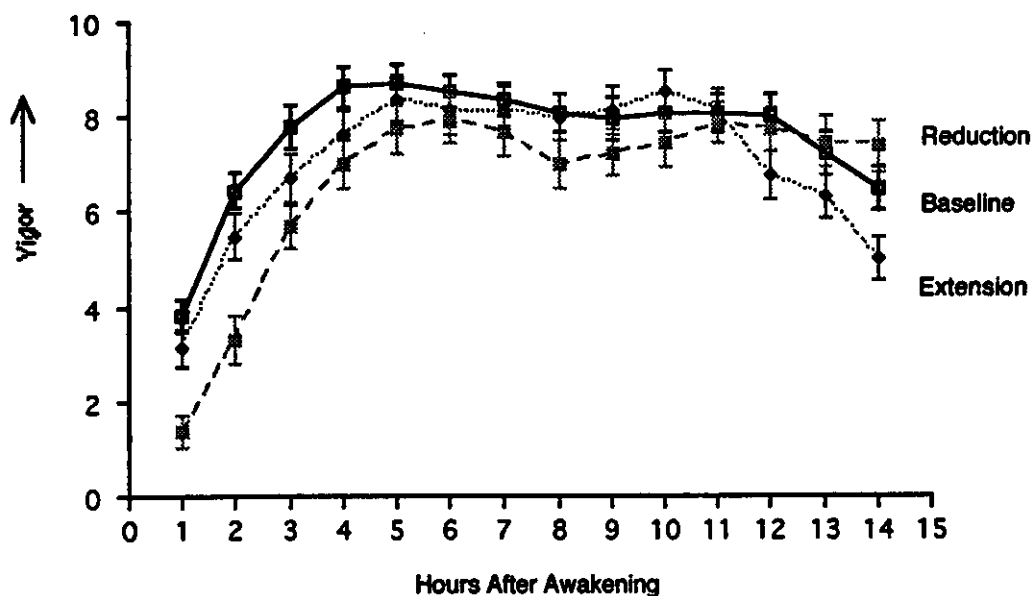
Fig. 6.6 Interaction of Vigor scores after Baseline, Sleep Extension and Sleep Reduction x Time in all 20 subjects.

c) Confusion

The differences in confusional state between conditions failed to approach significance, $F = 0.8$ [2, 99] $p = 0.45$. However, the interactions between conditions x time were significant, $F = 2.95$ [26, 1287] $p < 0.001$.
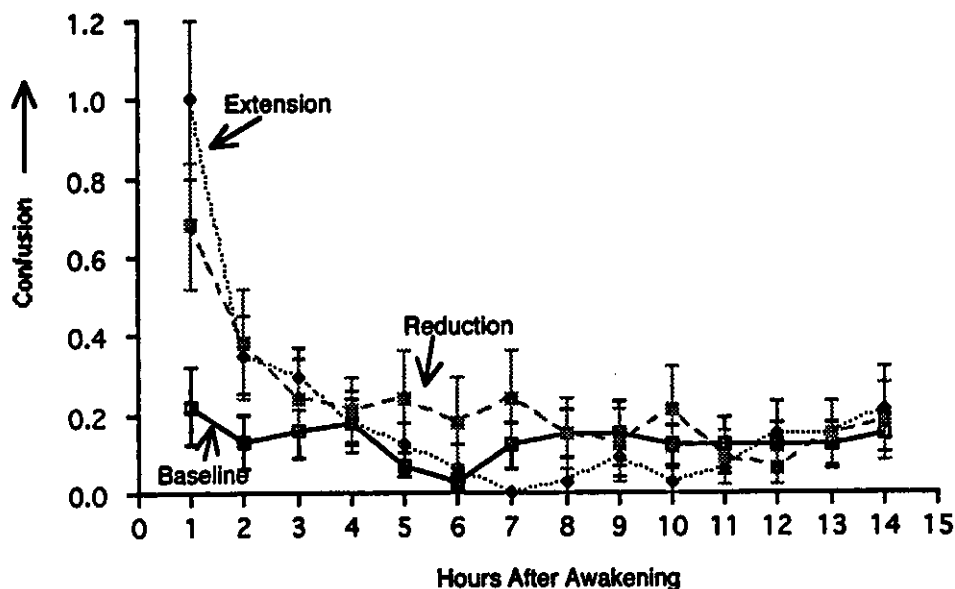
Fig. 6.7 Interaction of Confusion scores after Baseline, Sleep Extension and Sleep Reduction x Time in all 20 subjects.

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Fig. 6.7 illustrates that both Sleep Extension and Sleep Reduction resulted in an enhanced confusional state for the first 2 hours after awakening. Thereafter, levels of confusion were comparable across conditions.

d) Depression

There were no significant differences in Depression between the conditions, $F = 1.4$ [2, 99] $p = 0.25$. Significant interactions were observed between conditions x time, $F = 2.25$ [26, 1287] $p < 0.001$.
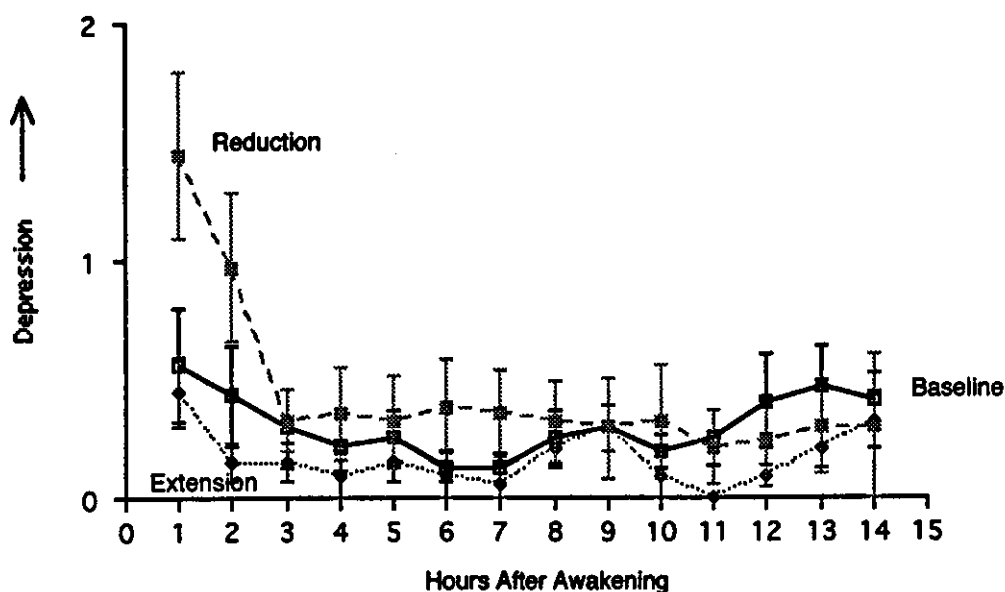
Fig. 6.8 Interaction of Depression scores after Baseline, Sleep Extension and Sleep Reduction x Time in all 20 subjects.

e) Tension

There were no significant differences in Tension between the conditions, $F = 0.2$ [2, 99] or in the interactions between conditions x time, $F = 1.03$ [26, 1287] $p = 0.42$.

f) Anger

Although the differences in anger between the conditions did not reach significance, $F = 1.92$ [2, 99] $p = 0.15$, the interactions between the conditions x time was significant, $F = 3.2$ [26, 1287] $p < 0.001$. Fig. 6.9 illustrates the findings. Post-hoc analyses revealed that after Sleep Reduction, subjects were more angry for the

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first 4 hours after awakening, compared to Baseline or Sleep Extension. Thereafter, scores were comparable across conditions.
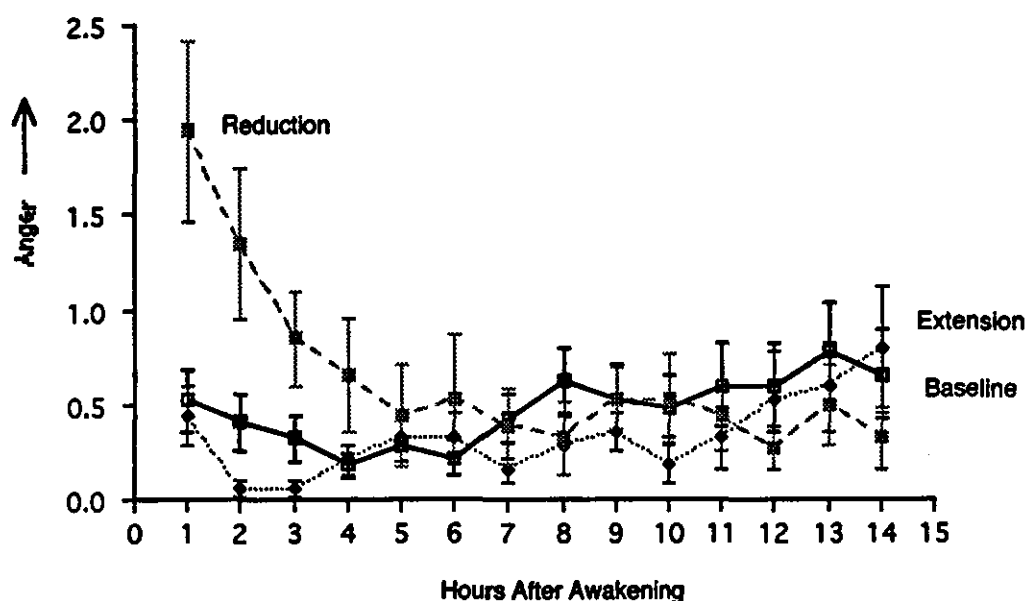
Fig. 6.9 Interaction of Anger scores after Baseline, Sleep Extension and Sleep Reduction x Time in all 20 subjects.

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The above analysis compared actual mood scores after Baseline, Sleep Extension and Sleep Reduction for all 34 subjects together. It was evident that subjects were adversely affected following both Sleep Extension and Sleep Reduction, this was reflected by enhanced levels of Fatigue compared to Baseline [see fig 6.5]. Additionally, Vigor was significantly decreased [see fig. 6.6] and confusion significantly elevated [see fig 6.7]. The following analyses will differentiate subjects with elevated fatigue [the WOS group] to the remaining subjects [the NWOS group]. Differences in personality in relation to mood following Sleep Extension and Sleep Reduction will also be examined in considerable depth.

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Sleep Extension

a) Fatigue [clock time].

Main Effects
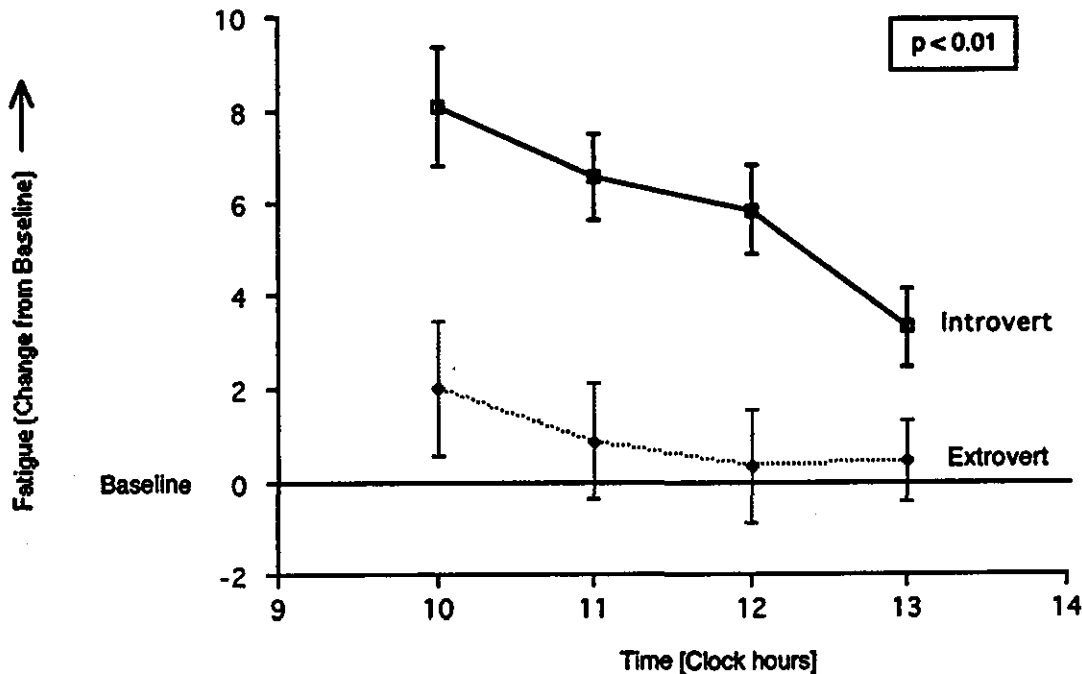
Fig. 6.10 Fatigue in Introverts and Extroverts following Sleep Extension.

There was a significant difference in Fatigue between Introversion / Extroversion, $F = 6.18$ [2, 31] $p = 0.006$. Post-hoc comparisons revealed significant differences between Introverts and Extroverts at all hours, $p < 0.005$, but not with the Neither group. Introverts were significantly more fatigued than Extroverts [see fig. 6.10]. Extroverts reported very little change from baseline. Findings suggested that introverts were adversely affected following oversleep, and that extroverts were unaffected by the oversleep.

Significant differences were observed in Toughness Poise, $F = 3.47$ [2, 31] $p < 0.05$. Post-hoc analysis indicated significant discrepancies between Tender minded and Tough individuals, $p < 0.05$, but not with the Neither group. Tender minded subjects described elevated fatigue [see fig. 6.11].

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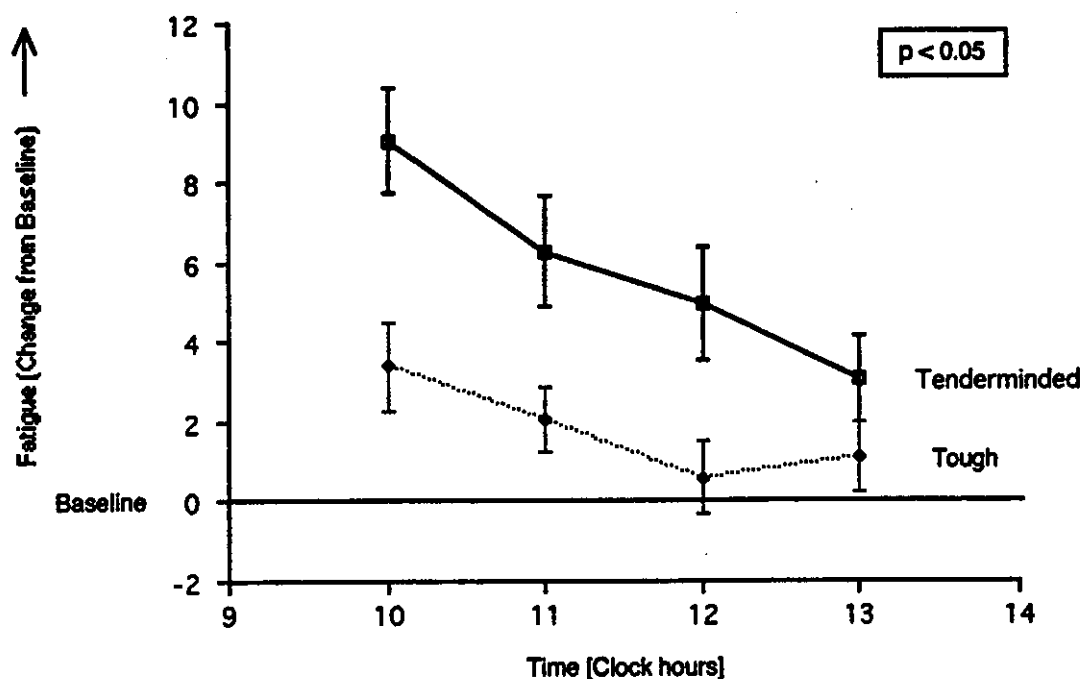
Fig. 6.11 Fatigue in Tender minded and Tough Individuals.

Although not significant, differences between morning and evening types highlighted trends, $F = 2.56$ [2, 31] $p = 0.09$, towards morning types describing increased fatigue compared to evening types. Figure 6.12 illustrates distinct differences in fatigue for the first three hours after arising: Following Sleep Extension, oversleep was detrimental to the well-being of morning types. Evening types reported very little deviation from baseline.
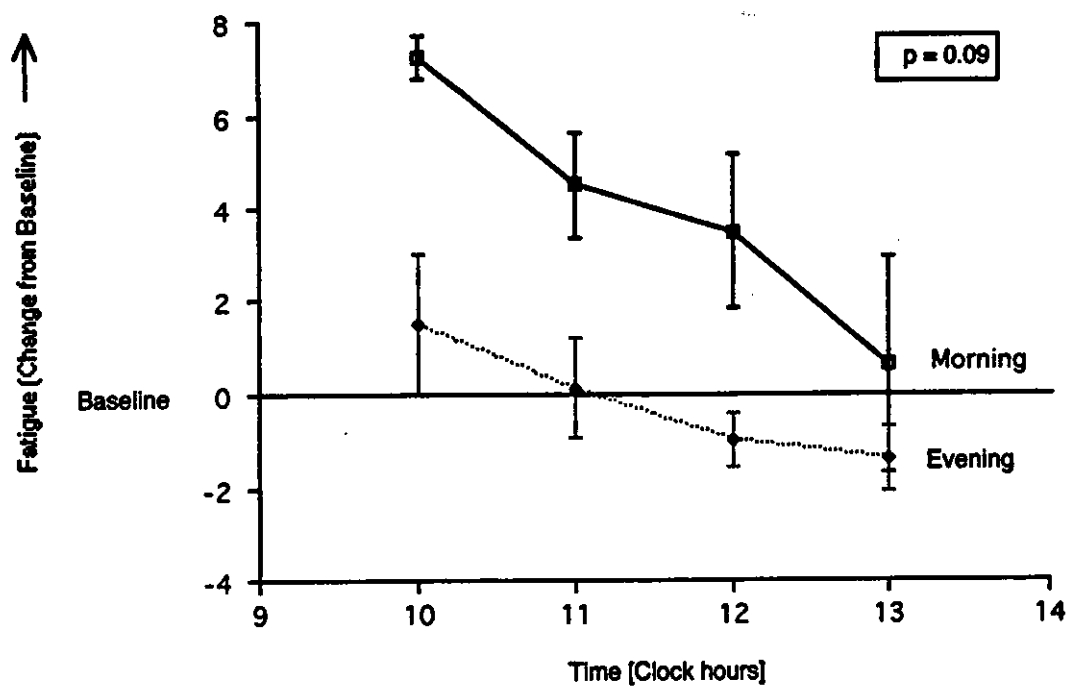
Fig. 6.12 Fatigue in Morning and Evening Types after Sleep Extension.
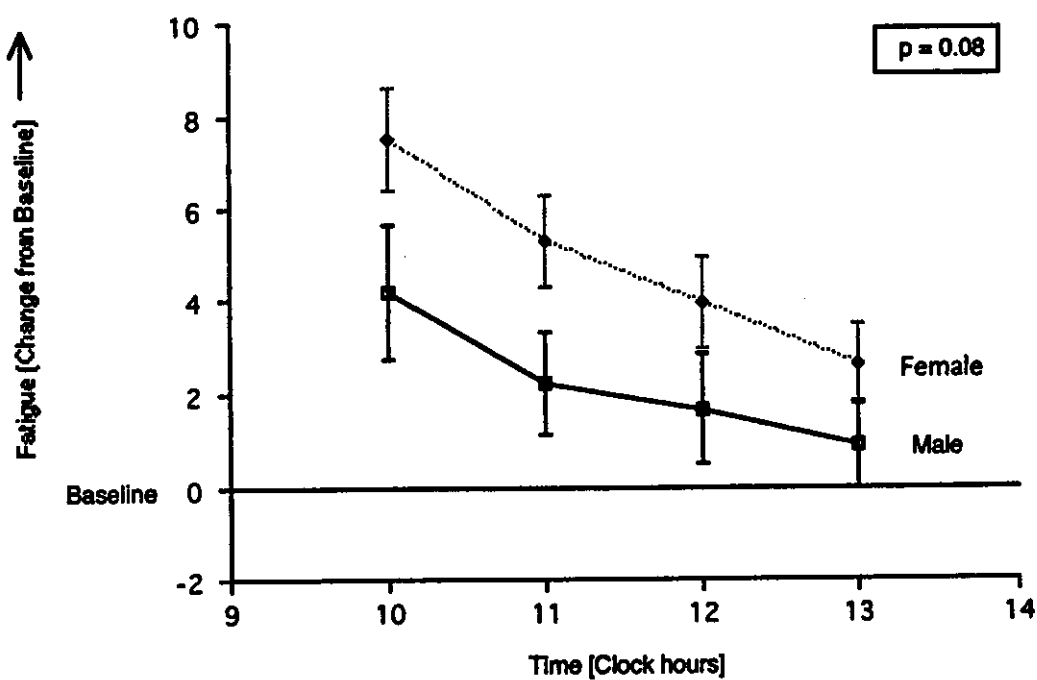
Fig. 6.13 Fatigue in Males and Females after Sleep Extension.

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Similarly, the difference between males and females approached significance, $F = 3.16$ [1, 32] $p = 0.08$. Following Sleep Extension, females described elevated levels of fatigue compared to males [see fig. 6.13]. One point of interest is that in this study, there were 17 WOS subjects and 17 NWOS subjects, of these there was a 5:12 and a 12:5 split by gender, Chi square analysis = 5.7 [df.1] $p < 0.02$. Clearly, the WOS group contained a greater number of females, and the NWOS group, more males.

Fatigue [Hours after Arising]

Main Effects

There was a significant difference in Introversion, $F = 7.23$ [2, 31] $p = 0.003$. Introverts reported increased fatigue with Sleep Extension compared to Extroverts. By contrast, Extroverts described a slight decrease in fatigue compared to baseline. After Sleep Extension, post-hoc analysis revealed significant differences between Introverts and Neither, $p < 0.05$, and between Introverts and Extroverts, $p < 0.01$.

Although not significant, differences between males and females approached significance, $F = 3.35$ [1, 32] $p = 0.08$, females described trends towards elevated fatigue following Sleep Extension. Trends emerged in Toughness Poise; tender minded subjects were more fatigued, $F = 2.8$ [2, 31] $p = 0.08$. There were no differences in fatigue between morning and evening types, $F = 2.13$ [2, 31] $p = 0.14$ or between low and high impulsives, $F = 0.52$ [2, 31] $p = 0.6$.

As the results were very similar for both 'hours after awakening' and 'clock time' the remaining data for vigor, confusion, depression, tension and anger will be discussed for 'clock time'.

b) Vigor

Main Effects

Significant differences emerged in Vigor in the Introversion groups, $F = 4.68$ [2, 31] $p = 0.02$. Post-hoc analysis revealed the difference was between Introverts and Extroverts, $p = 0.01$ [see fig. 6.14].

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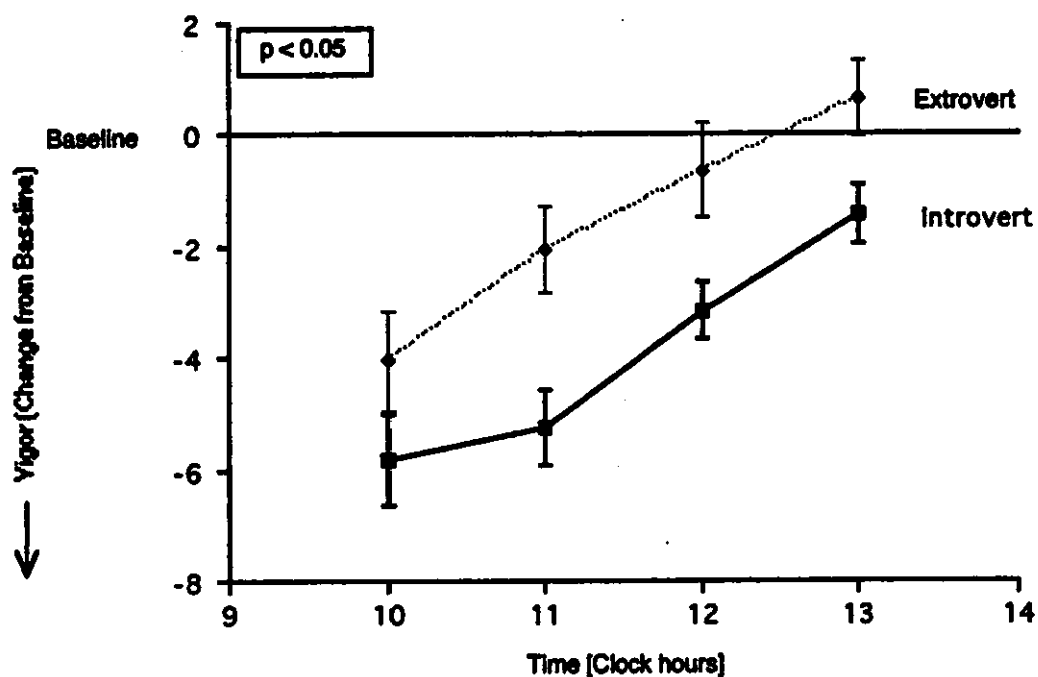
Fig. 6.14 Vigor in Introverts and Extroverts after Sleep Extension.
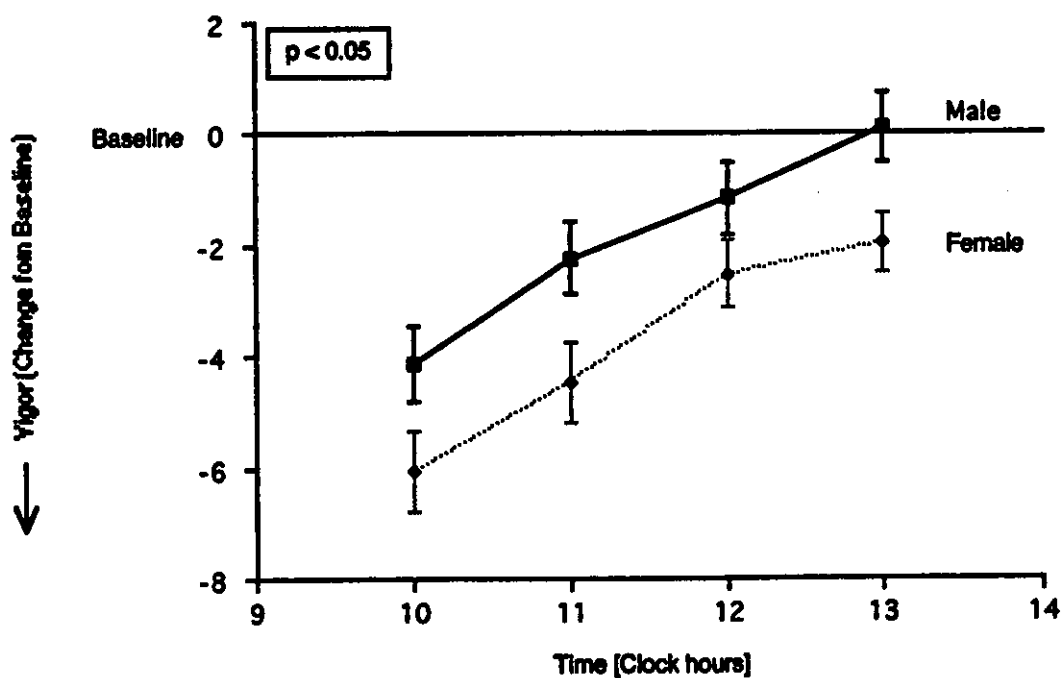
Fig. 6.15 Vigor in Males and Females after Sleep Extension.

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There was a significant difference in Vigor between males and females, F = 5.82 [1, 32] p = 0.02. Although males and females described similar trends of diminished vigor, females reported a greater decrement in state of energy following Sleep Extension [see fig. 6.15].

c) Confusion

Main Effects
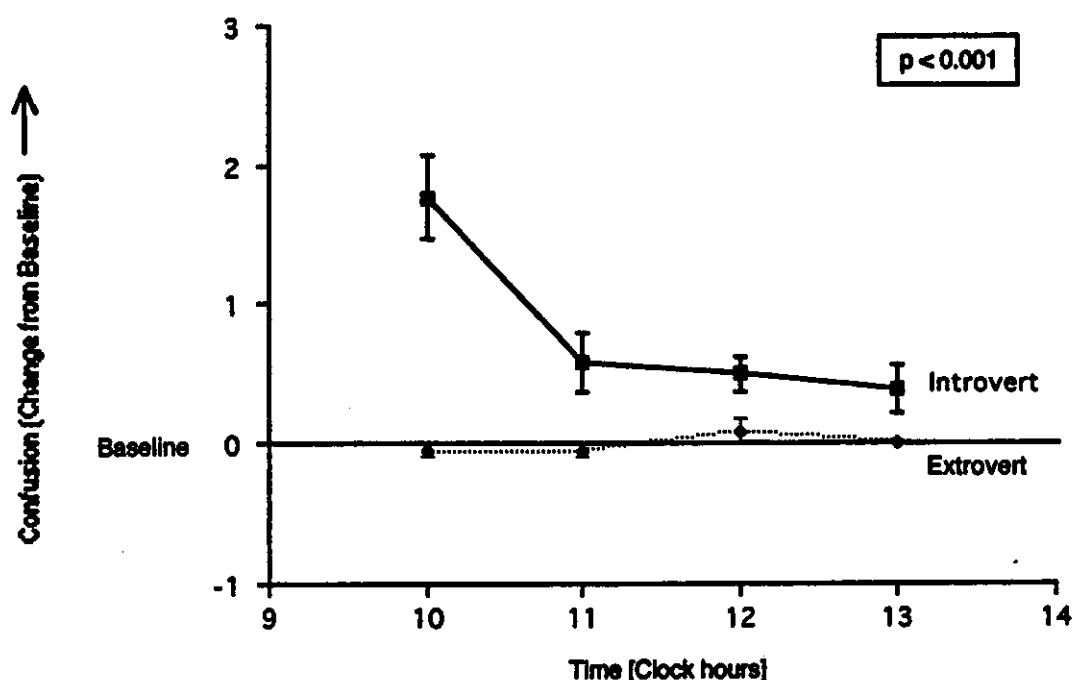
Fig. 6.16 Confusion in Introverts and Extroverts after Sleep Extension.

Significant differences emerged in Introversion, F = 11.97 [2, 31] p < 0.001. Post-hoc analysis revealed the difference was between Introverts and both Neither type and Extroverts, p < 0.01. Introverts reported a greater confusional state than Extroverts, who described very little change from baseline [see fig. 6.16].

Non-significant trends emerged in impulsivity, F = 2.94 [2, 31] p = 0.07, post-hoc tests indicated the trends were between low impulsives and neither groups. There were no significant differences in confusional state between males and females, between tough and tender-minded subjects, or between morning and evening types.

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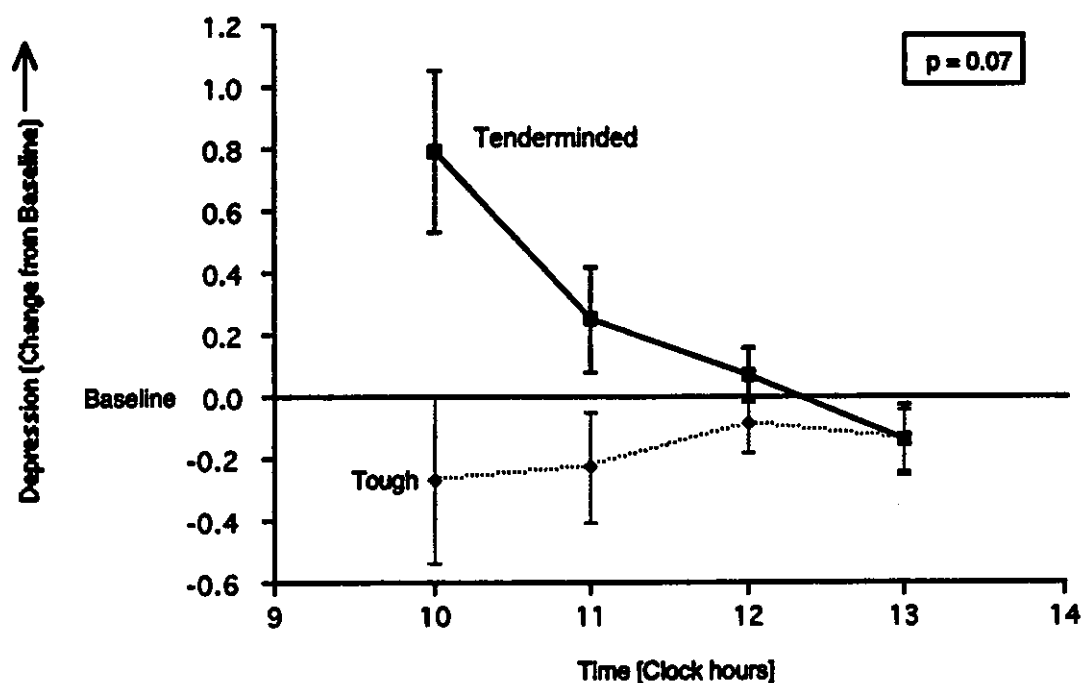
d) Depression

Fig. 6.17 Depression in Toughness Poise following Sleep Extension.

Following Sleep Extension, there was a significant difference in Depression between tender minded and neither groups, $F = 4.64$ [2, 31] $p = 0.02$. Post-hoc tests revealed the difference between tender minded and tough individuals approached significance, $p = 0.07$. Figure 6.17 illustrates that tender minded subjects described an elevated depressive state relative to tough-minded subjects, particularly for the first 3 hours. Four hours after arising, both groups reported similar levels of depression after Sleep Extension. Subjects in all other groups described non-significant changes in depression following Sleep Extension.

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Sleep Reduction

a) Fatigue

Following Sleep Reduction, there were no significant differences in fatigue between morning and evening types, introverts and extroverts, tough and tender minded individuals, low and high impulsives, or between males and females. Subjects in all groups responded similarly.

b) Vigor

Main Effects

The difference in Vigor between introverts and extroverts approached significance, $F = 3.05$ [2, 31], $p = 0.06$. Figure 6.18 illustrates trends, Extroverts described elevated energy levels after Sleep Reduction.
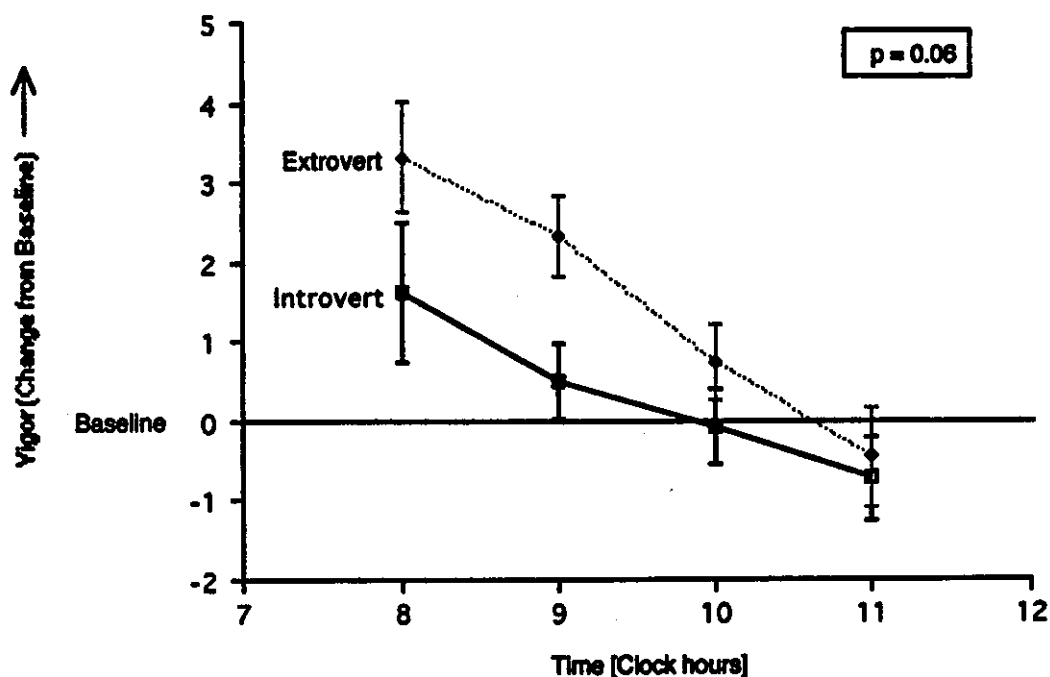
Fig. 6.18 Vigor in introverts and Extroverts after Sleep Reduction.

Following Sleep Reduction there were no significant differences between groups for either Confusion, Depression or Tension. There was a significant difference in anger between extroverts and neither groups, but not between introverts and extroverts, $F = 3.72$ [2, 31], $p = 0.04$ [Post-hoc test $p < 0.05$].

Summary of Results

The WOS group consisted primarily of subjects who were:
      (i)   Not very impulsive
      (ii) Introverted
      (iii) Emotionally Tender minded and
      (iv) Predominantly female
      — [see Table 6.5].
By contrast, subjects in the NWOS group were:
      (i)   Extroverted
      (ii) High in Toughness Poise
      (iii) Impulsive and
      (iv) Predominantly male.

Analysis [Two-way ANOVA's] revealed that the WOS group were significantly more introverted and dependent than the NWOS group, who were categorised as extrovert and independent. Non-significant trends indicated that subjects in the WOS group were emotionally tender minded and more anxious than individuals in the NWOS group.

Analyses of variance compared all six mood variables [e.g. fatigue, vigor, confusion, depression, anger and tension] across specific personality factors [e.g. introversion, toughness poise, impulsivity and morningness]. The findings will be discussed on data analysed for the first four hours after arising, since this period is most critical in reference to the wornout syndrome [after Globus, 1969].

Following Sleep Extension, introverts described significantly greater levels of fatigue and confusion, in addition to significantly diminished Vigor. By contrast, extroverts detailed little change from baseline in all mood variables. Subjects categorised as emotionally tender minded indicated significantly elevated levels of fatigue and depression after Sleep Extension. Subjects high in toughness poise were unaffected by the oversleep. Non significant trends were evident; morning types

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were more fatigued than evening types following Sleep Extension. Similarly, trends indicated that females described enhanced fatigue and diminished vigor, relative to males. Examining subjects mood states after Sleep Reduction, it was evident that extroverts, who were unaffected by Sleep Extension, described elevated levels of vigor. There were no significant differences between the personality categories for the remaining mood variables.

Subjects with certain personality characteristics were particularly affected by Sleep Extension. Introversion was a significant category. Findings were observed specifically between introverts and extroverts. Introverts were adversely affected by Sleep Extension, but not after Sleep Reduction. Emotionally tender minded subjects were also affected by oversleeping: this was reflected by accounts of enhanced fatigue and depressive feelings. Gender differences were observed after Sleep Extension but not after Sleep Reduction: females were adversely affected by oversleep but not by sleep restriction.

SSS Questionnaire

The sleepiness ratings of the 17 subjects with the 'wornout syndrome' were compared to the 17 without, after baseline sleep, extension and reduction. Subjects completed the SSS hourly for the first 5 hours after arising.

Baseline

Table 6.21 Sleepiness [SSS] scores after Baseline Sleep.

Hrs after arising	Wornout Syndrome [N=17] mean (se)	No Wornout Syndrome [N=17] mean (se)
1	3.32 (.27)	4.06 (.39)
2	2.15 (.2)	2.85 (.35)
3	1.88 (.25)	1.94 (.23)
4	1.32 (.11)	1.68 (.21)
5	1.47 (.13)	1.68 (.2)

SSS
1 = Active, vital, alert and wide awake
2 = Functioning at a high level, not at peak, but able to concentrate
3 = Relaxed, awake, not at full alertness, responsive
4 = A little foggy, not at peak, let down
5 = Fogginess, starting to lose interest in remaining awake, slowed down
6 = Sleepiness, preferred to be resting, fighting sleep, feeling woozy
7 = Almost unable to stay awake, struggling to remain awake

Following a night of baseline sleep, both the WOS and NWOS groups described similar levels of sleepiness at the hourly testing in the morning. An SSS score between 3 and 4 for the first hour after arising diminished to a score between 1 and 2, four hours later.

Sleep Extension

Table 6.22 Sleepiness [SSS] scores after Sleep Extension.

Hrs after arising	Wornout Syndrome [N=17] mean (se)	No Wornout Syndrome [N=17] mean (se)
1	4.18 (.26)	3.0 (.23)
2	3.29 (.22)	2.06 (.22)
3	2.71 (.27)	1.71 (.19)
4	1.88 (.26)	1.24 (.11)
5	1.88 (.24)	1.35 (.15)

Results were analysed using repeated measures ANOVA. The between group variable was the condition [WOS and NWOS]. The within group factor was time [5 levels]. A significant finding emerged between WOS and NWOS groups: $F= 17.8$ [1, 32] $p < 0.001$. Those subjects in the WOS group selected higher SSS scores (representing a greater level of sleepiness) compared to subjects in the NWOS group.
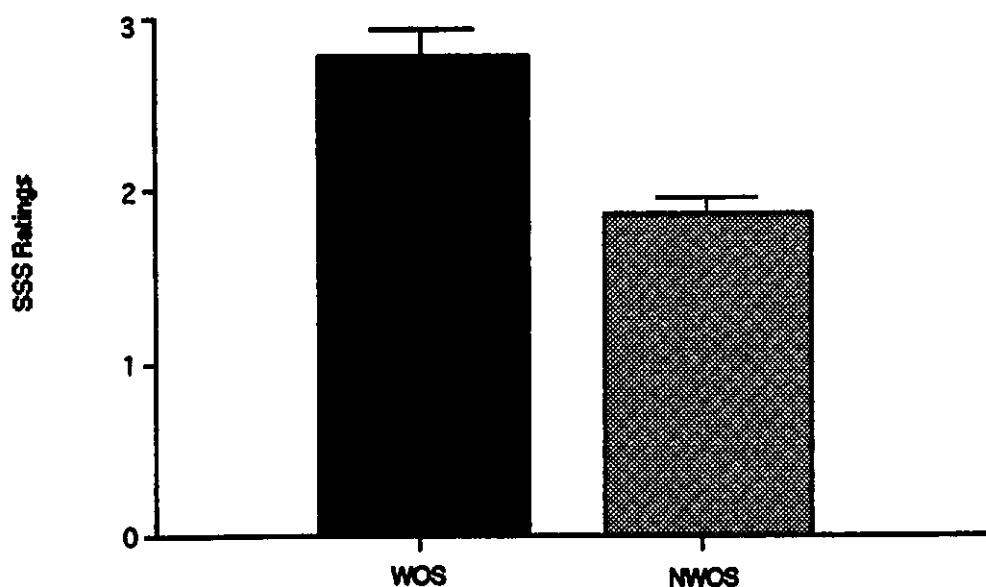
Fig. 6.19 Sleepiness Ratings [SSS] in the WOS and NWOS groups.

Interactions

Interactions between N/WOS x time after Sleep Extension did not yield significant differences, $F = 1.78$ [4, 128] $p = 0.14$. However, Post-hoc analyses revealed significant differences [$p < 0.05$] between the WOS and NWOS groups at the following times:

    i. WOS at hr 1 vs. NWOS from hrs 1 to 5

    ii. WOS at hr 2 vs. NWOS from hrs 2 to 5

    iii. WOS at hr 3 vs. NWOS at hrs 3 to 5

    iv. WOS at hr 4 vs. NWOS at hr 1

    v. WOS at hr 5 vs. NWOS at hr 1
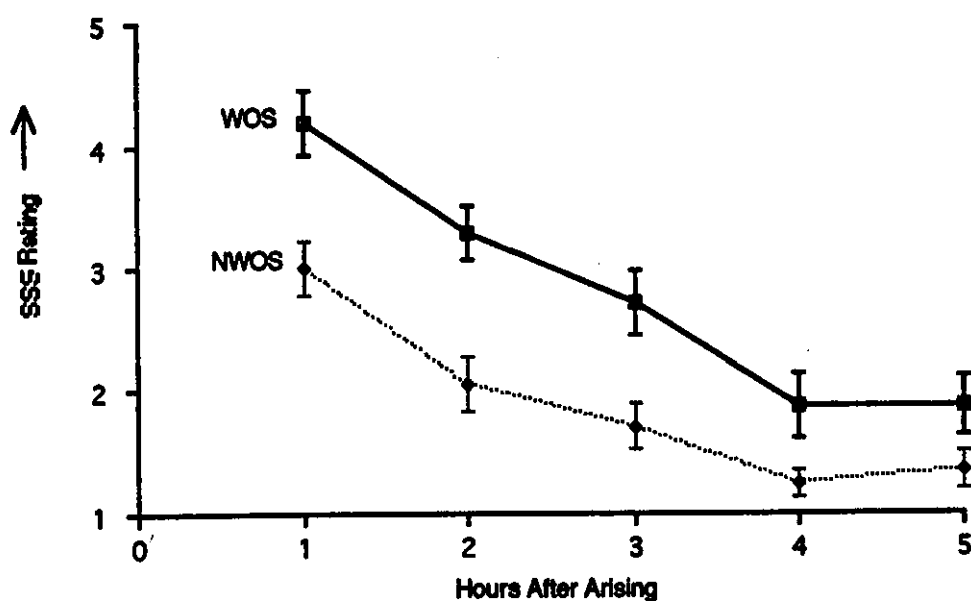
Fig. 6.19 SSS Ratings In the WOS and NWOS groups over Time.

Reduction

It is clear [see table 6.23] that following Sleep Reduction, the mean values in both the WOS and NWOS groups were similar across time.

Table 6.23 Sleepiness [SSS] scores after Sleep Reduction.

Hrs after arising	Wornout Syndrome [N=17] mean (se)	No Wornout Syndrome [N=17] mean (se)
1	5.29 (.37)	5.82 (.37)
2	4.12 (.34)	3.82 (.54)
3	3.18 (.34)	2.82 (.39)
4	2.18 (.25)	2.0 (.4)
5	2.18 (.27)	1.82 (.37)

Tiredness vs. Sleepiness

The SSS was administered as a measure of sleepiness. However, due to earlier concerns in its usage, a revised Sleepiness Questionnaire was devised. It was hypothesised that the SSS measures two distinct dimensions, namely Tiredness and Sleepiness. The following variables were selected from the SSS as measures of these dimensions:

Tiredness	Sleepiness
Let Down	Losing Interest in Remaining Awake
Slowed Down	Sleepiness
Unable to Concentrate	Sleep Onset Soon

It was hypothesised that subjects experiencing the 'wornout syndrome' following oversleep would be more 'tired' than 'sleepy'; that they would select more items from the Tiredness category rather than the Sleepiness one, following Sleep Extension. Subjects responses were examined following Baseline, Extension and Reduction conditions and the actual numbers in each group selecting items from these categories was noted for the first 4 hours following awakening. Chi-Squared analysis was conducted to investigate any significant differences in the selection of Tiredness and Sleepiness items by subjects in the WOS and NWOS groups.

Results

Baseline Sleep

Both WOS and NWOS groups described similar trends relating to the number of items selected in both Tiredness and Sleepiness categories. Chi-Squared analysis failed to reveal significant differences between WOS and NWOS groups after Baseline sleep. This was encouraging, since results were not expected to differ here. Overall, subjects in the NWOS group selected more items from the Tiredness category than the WOS group [see figs. 6.15 and 6.16]. Similarly for hours 1, 2 and 4, subjects in the NWOS group selected more Sleepiness items than the WOS group.
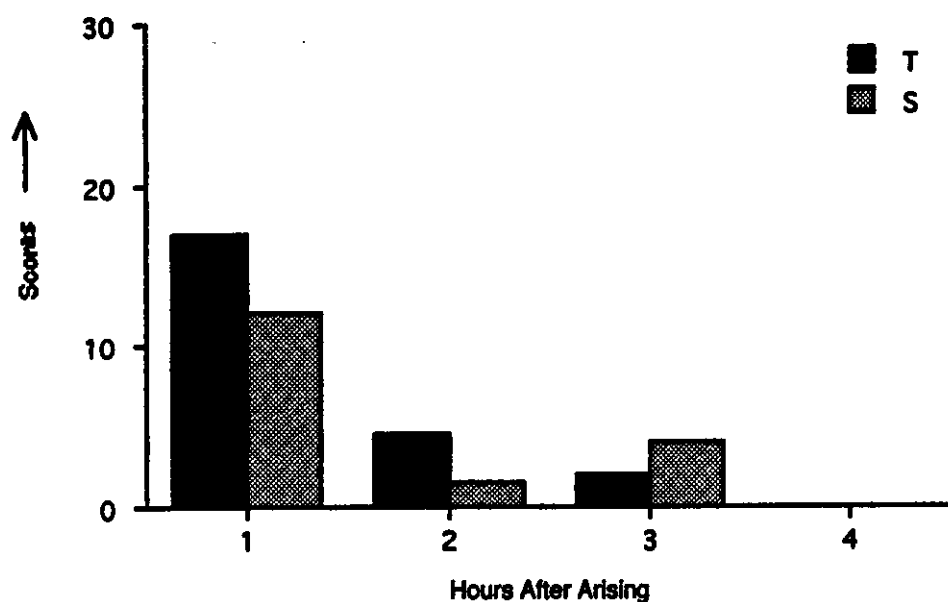
Fig. 6.15 Number of 'Tiredness' and 'Sleepiness' items selected by subjects in the WOS group after Baseline.
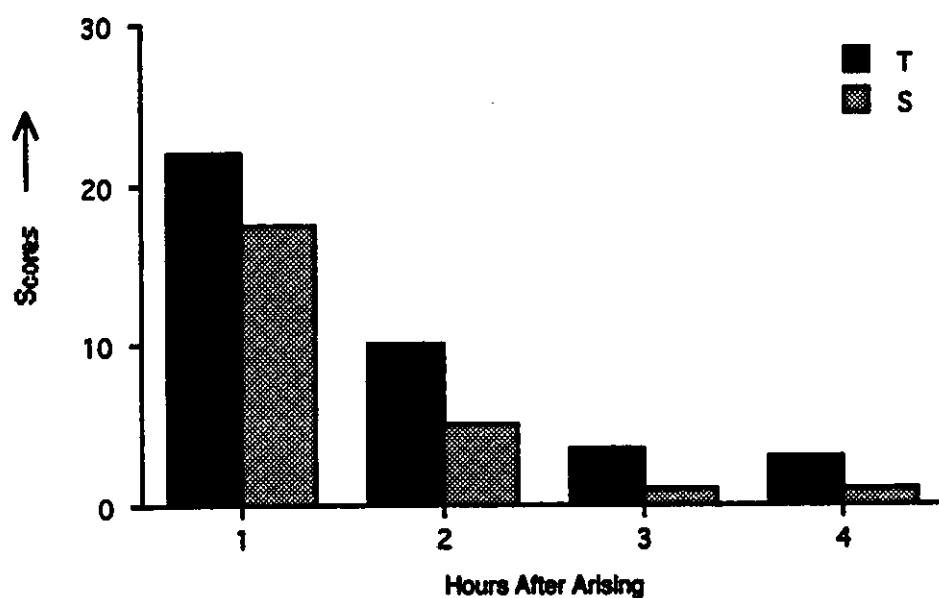
Fig. 6.16 Number of 'Tiredness' and 'Sleepiness' items selected by subjects in the NWOS group after Baseline.

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Sleep Extension

There was a distinct difference between the groups. The WOS group selected more Tiredness items relative to Sleepiness for all 4 hours. Chi-Square analysis revealed significant differences at:

a). Hour 1, Chi-square = 6.23 [df.1] $p < 0.05$
b). Hour 2, Chi-square = 7.76 [df. 1] $p < 0.01$
c). The difference at hour 3 approached significance,
   Chi-square = 3.77 [df. 1].

Compared to the NWOS group, subjects in the WOS group described elevated levels of Tiredness for each of the 4 hours tested [see figs. 6.17 and 6.18]. Relative to Baseline, the WOS group were adversely affected following Sleep Extension, and this was reflected in their enhanced Tiredness scores.

Examining the number of Sleepiness items selected, it was apparent that the WOS group described similar trends for both Sleep Extension and Baseline. In contrast, the NWOS group selected fewer items measuring Sleepiness after Sleep Extension than after Baseline, suggesting that additional sleep was beneficial; this was reflected by diminished ratings of Sleepiness.

It is evident that while subjects in the WOS group described enhanced fatigue after Sleep Extension, subjects in the NWOS group reported an improved state, and thus found the extra sleep beneficial.
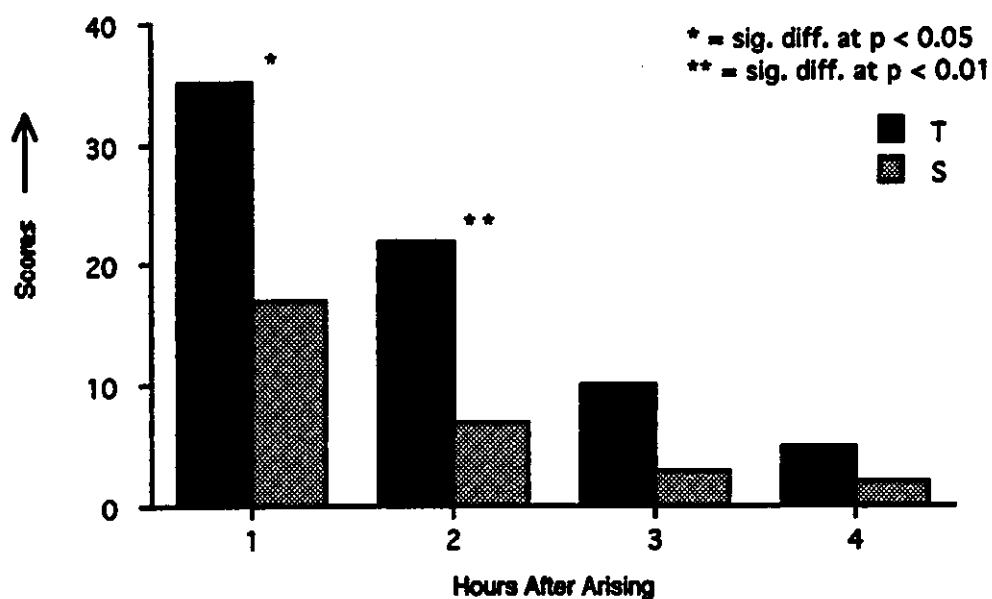
Fig. 6.17   Number of 'Tiredness' and 'Sleepiness' items selected by subjects in the WOS group after Sleep Extension.
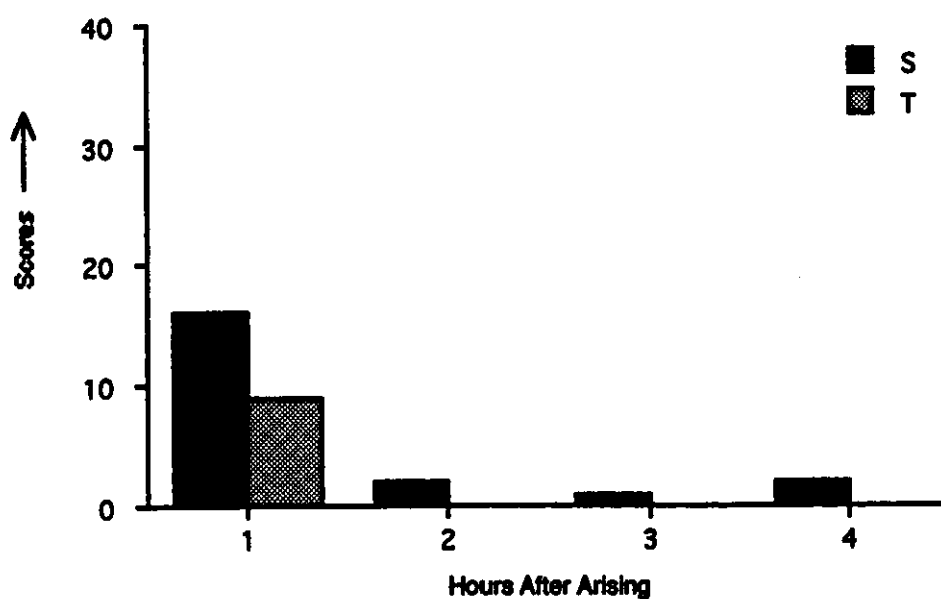
Fig. 6.18   Number of 'Tiredness' and 'Sleepiness' items selected by subjects in the NWOS group after Sleep Extension.

Sleep Reduction

Following Sleep Reduction subjects in the WOS group chose a similar number of items from both Tiredness and Sleepiness categories. Chi-Squared analysis failed to yield significant differences between the groups.

By contrast, subjects in the NWOS group exhibited divergent trends. For hours 2, 3 and 4, more Sleepiness items were selected than Tiredness ones by subjects in the NWOS group [see figs. 6.18 and 6.19]. These findings approached significance for hour 3, Chi-square = 3.56 [df. 1].

The WOS group exhibited similar trends in their selection of items from the Tiredness category following both Sleep Reduction and Sleep Extension. It is argued that the WOS group were adversely affected to a similar extent after both Sleep Extension and Sleep Reduction.

Paradoxically the NWOS group selected more Sleepiness items following Sleep Reduction than after both Baseline and Sleep Extension. This supports the hypothesis, subjects are expected to be more sleepy than tired after Sleep Reduction.

Subjects in both the WOS and NWOS groups displayed similar trends in their selection of Sleepiness items after Sleep Reduction. Both groups described elevated levels of subjective sleepiness compared to Baseline or Sleep Extension.
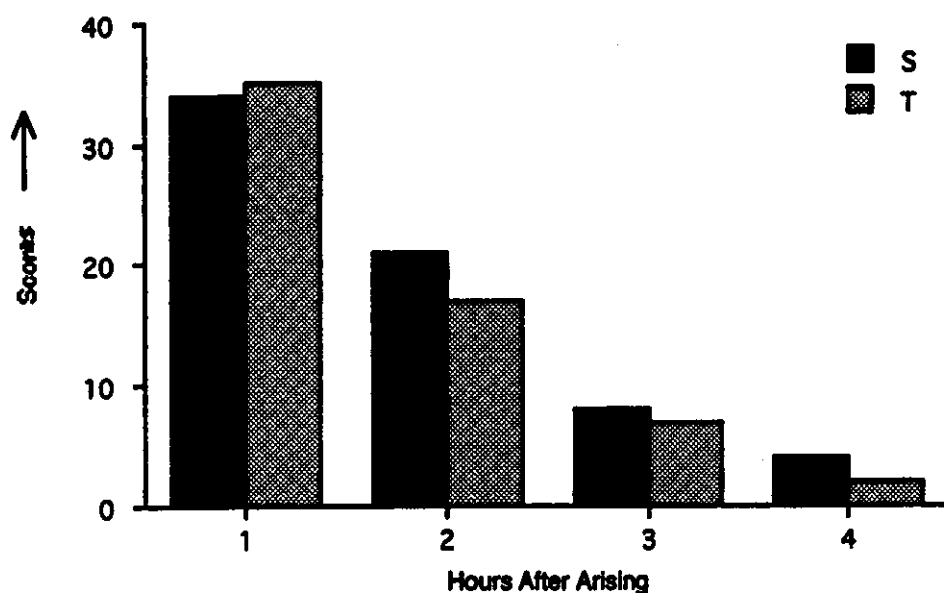
Fig. 6.18   Number of 'Tiredness' and 'Sleepiness' items selected by subjects in the WOS group after Sleep Reduction.
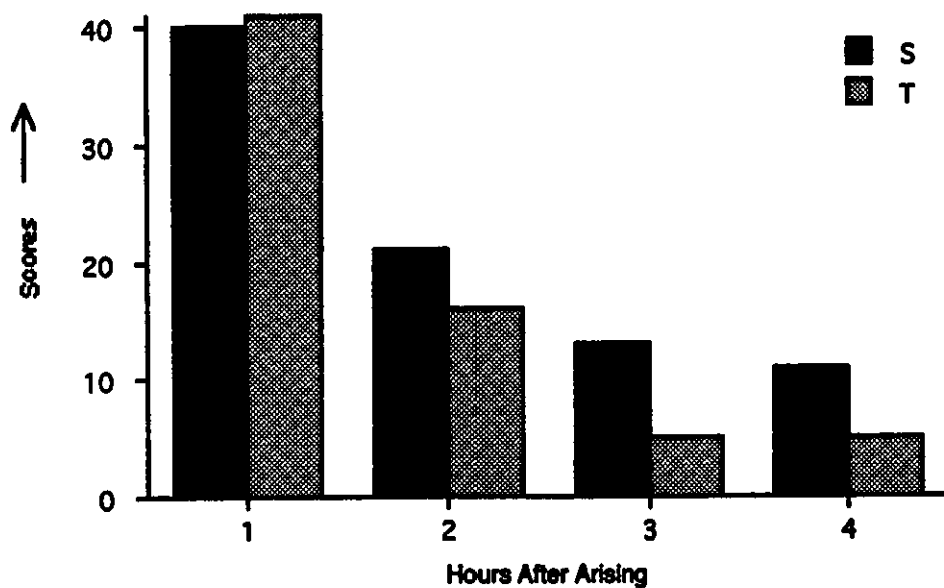
Fig. 6.19   Number of 'Tiredness' and 'Sleepiness' items selected by subjects in the NWOS group after Sleep Reduction.

Only after Sleep Extension was the diversity of the results in the WOS and NWOS groups apparent. The WOS group definitely described more subjective Tiredness than Sleepiness following oversleep. Subjects in the NWOS group reported less Tiredness and Sleepiness after Sleep Extension than after Baseline. Both groups were affected to a similar extent following Sleep Reduction. These findings provide evidence to support the theory that following oversleep, subjects are not sleepy, but are actually tired. The issue of individual differences is further highlighted since only certain subjects, specifically those in the WOS group, indicated enhanced Tiredness following Sleep Extension.

This analysis highlights the problems of using a measure of Sleepiness at face value. Current findings have indicated that the SSS is not a unidimensional measure of Sleepiness. It is argued that any results measuring subjective sleepiness using the SSS be interpreted with caution. It may be that subjects are responding to the Tiredness, rather than the Sleepiness dimension.

7

Discussion and Conclusions

7. Discussion and Conclusions

7.1 The Wornout Syndrome

Most people assume that the optimum amount of sleep is somewhere in the region of seven to eight hours per night. Nights when less than the *ideal* sleep duration is attained are generally viewed as 'bad' nights. Many of us believe that reduced sleep will have adverse effects upon subsequent mood and daytime performance. Furthermore, it is generally accepted that extra sleep will have beneficial effects on mood and general well-being. Several recent surveys [e.g. Webb and Agnew, 1975; Anch, 1988] have focused on sleep duration and the most common estimation of total sleep time falls between 7 to 9 hours per night. However, there is considerable inter-individual variability. This is reflected by accounts of extremely long and short sleepers, who appear to function without ill-effects.

The spontaneous lengthening of sleep at weekends and times when there are no external constraints to wake up have been well documented for both young [Webb and Agnew, 1975] and older adults [Lavie, 1981]. We frequently assume that a weekend lie-in will to some extent compensate for arising earlier than preferred during weekdays, when sleep duration is more dependent on external constraints (e.g. work schedules). We conform to a large number of social cues which operate as powerful zeitgebers in the organisation of work, social activities and sleep schedules. The voluntary increment in sleep duration at weekends and holidays, can be viewed as coincidental with the loosening of these zeitgebers.

Globus [1969] gathered retrospective information from students concerning their sleeping habits. He recognised a condition following oversleep, which he termed the 'wornout syndrome'. This syndrome was most likely to occur in individuals who had achieved greater than 10 hours of sleep and who were not making up for any prior sleep loss. Although Globus' work was significant, and has had considerable impact on later studies, it can be criticised on a number of issues: (i) the findings were based solely on retrospective accounts, which may have been distorted; (ii) no methods were employed to ensure that confounding variables were minimised, e.g. standardisation of daily activity levels; (iii) although the term most frequently used to describe their mood state was 'sleepy', no measures of the degree or even type of sleepiness were applied.

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Several studies have failed to find beneficial effects of oversleep upon performance or daytime alertness [e.g. Carskadon, 1986; Roehrs, 1989]. Clearly contradictory findings have emerged in studies relating performance decrements to manipulations of sleep duration. The findings reported in this thesis have assessed the relationship between mood and sleep duration in considerable depth. Previous research had neglected this important area, concentrating instead on establishing the association between sleep duration and simple performance.

In the first study reported in this thesis, the relationship between sleep length and subsequent self-reported mood was assessed. A simplified version of the POMS mood scale was devised, examining the mood variables fatigue, vigor and confusion, since these were deemed most sensitive to Globus' wornout syndrome. It was observed that four of the nine subjects experienced the wornout syndrome following oversleep. The detrimental effects of oversleeping were particularly pronounced in the first four hours after arising. This preliminary study provided evidence to support the existence of a wornout syndrome following increased sleep duration. The adverse effects on mood of extending sleep by two hours for one night were similar to those observed after a comparable reduction in sleep length. The pattern in the results was most evident when comparisons were made between the wornout syndrome [WOS] and non-wornout syndrome [NWOS] groups after extension and restriction of sleep. After sleep extension, only those subjects in the WOS group were adversely affected. However, after sleep reduction, subjects in both the WOS and NWOS groups were affected in a similar manner. As this was a preliminary study, personality factors and individual differences were not assessed. Hence, very little information was gained concerning the psychological state of the affected individuals.

The second study examined the area of sleep and mood in greater depth, with the inclusion of personality variables. Twenty healthy young adults were recruited. Mood states were compared to baseline after sleep extension and sleep reduction. Subjects were differentiated according to whether they were regular or irregular sleepers [based on actigraph readings and one-week daily diaries logging subjects' times of retiring and awakening]. In addition personality was assessed using the 16PF questionnaire. A more comprehensive mood questionnaire was devised, assessing six mood variables. Findings indicated that eight of twenty subjects were adversely affected by sleep extension. When compared to irregular sleepers, individuals maintaining regular sleep/wake schedules were significantly more fatigued and less energetic after sleep extension. In fact, 7 of the 8 subjects in the

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WOS group were regular sleepers. Interestingly, the regular sleep/wake schedules did not appear to have a significant impact on subjective mood states following sleep restriction. There were no differences in mood states between regular and irregular sleepers following a two-hour reduction in sleep length, both reported comparable levels of adversity. These observations suggested that regularity in sleep/wake schedules was a significant determinant of mood state, but only after excess sleep.

Individual differences were evident in the extent to which subjects were affected by changes in sleep duration. Certain personality characteristics were more prone to negative mood states following sleep extension. Introverts, emotionally tender minded and low impulsives described adverse effects of oversleep on mood. In contrast, subjects unaffected by oversleep were considerably more extroverted and high in toughness poise. Opposite trends were evident following sleep restriction. Subjects in the WOS group reported beneficial effects of sleep restriction. Morning types described less confusion and less depression than evening types after sleep restriction. Perhaps these subjects were more accustomed to arising earlier and therefore experienced fewer detrimental effects. Subjects who were unaffected by oversleep [NWOS] described adverse mood states following a comparable reduction in sleep length. This was reflected by accounts of enhanced fatigue, confusion, depression, anger and tension, in addition to a decrement in vigor.

Once these divergent trends in personality were disclosed, it was considered necessary to investigate further the precise role of personality in the increasingly complex relationship between sleep and mood. Study three examined such factors in more detail. It was hypothesised that the individual's personality modulates the effect of sleep duration on mood and therefore introverts, morning types, emotionally tender minded and cautious individuals are more likely to experience the wornout syndrome following sleep extension. To test the hypothesis thirty-four subjects were categorised into specific groups based on personality type. Mood responses to sleep extension and restriction were assessed and compared to normative baseline sleep. The results obtained did tend to support the hypothesis.

Thus individuals categorised as introverts, morning types, emotionally tender minded and cautious were prone to experiencing the adverse effects of oversleep on mood. By comparison, extroverts, evening types, tough and impulsive individuals reported either no change from baseline following oversleep, or in some cases, beneficial effects on mood. Gender differences were observed in certain personality

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groups: for example, 73 percent of cautious individuals were females, and 82 percent of extroverts were male.

Studies 2 and 3 explored the relationship between mood and personality. Wessman and Ricks [1966] proposed that individuals of a particular personality type were more prone to experiencing certain moods than others. Costa and McCrae [1980] observed a strong positive correlation between positive affect and extroversion, and between negative affect and neuroticism. The results documented in this thesis also provide strong evidence of an association between personality and mood.

The observation that subjects were quite easily able to extend their sleep by two hours might represent a convincing argument for those advocating more sleep at night. However, results from the current study do not support this notion. The adverse effects of oversleeping were observed in many individuals. Feelings of lethargy, thick-headedness, fatigue, low vigor and confusion following oversleep ought to prove a powerful deterrent to many individuals, particularly if they are concerned with maximising their potential and avoiding negative moods.

Subjects in the present study were able to tolerate the imposition of an extended sleep period at night and relied on alarm clocks to awaken them during sleep restriction conditions. Throughout the baseline week, actigraphs and subjective reports confirmed subjects' estimations of preferred sleep times. In contrast with previous investigations, subjects in this study slept in their normal home environment throughout, thereby avoiding the influence of any extraneous factors on mood.

Every effort was made to standardise conditions in this study. All subjects were assessed over four weeks. Experimental manipulations of sleep were always conducted on the same night of the week, and always on weekdays. This method was employed since activity during weekdays is more consistent from one week to the next, as opposed to weekends which are generally more variable, and guided more by social demands. Subjects kept a daily diary [completed hourly] to enable any deviations from normal activities to be registered, and to enable comparisons to be made across conditions. In addition, wrist actometers were worn, providing objective confirmation of subjects' activity levels and times of retiring and awakening. On the night preceding each study night subjects were asked to maintain their usual sleep schedule. This was to ensure that after the oversleep condition, subjects attained excess sleep and were not making up for any prior reduction in sleep duration.

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The findings reported in this thesis support Mc.Nair et al.'s [1971] portrayal of mood as multidimensional: each of the six mood variables were treated as monopolar and discrete factors. Earlier researchers [Blaney, 1986] have argued that mood is a difficult concept to measure, since it is a dynamic process. I have attempted to overcome this problem by administering mood questionnaires hourly, throughout the day. By employing this technique, diurnal variations in mood become apparent and any deviations in mood state are easily identifiable at specific times throughout the day.

Nowlis [1965] has argued that mood is "a multidimensional set of temporary reversible dispositions". This view has been supported by the current investigation. Subjects were affected in diverse ways following manipulations to sleep duration. Divergent responses were obtained to the independent mood variables measured. In most cases, the effects of altering sleep length upon mood were transient, but persisted for up to five hours. Many subjects were fatigued and confused upon arising after sleep extension, but most returned to normal 5 hours after awakening.

As mood is a subjective experience, individual reports of their feelings potentially have a personal validity. However, researchers [e.g. Cronbach, 1942] have criticised self-rating scales, casting doubt on the reliability of the informant. It has been suggested that subjects may either be defensive and reluctant to reveal their innermost experiences, or may exaggerate or overemphasise their feelings. Social desirability may influence scores, with subjects trying to depict themselves in a favourable light.

Previous sleep extension studies were specifically concerned with the consequences of ad-libitum extended sleep conditions (i.e. where subjects were allowed to gain as much extra sleep as preferred) by sleeping on in the morning. This includes Taub [1981] who documented enhanced subjective sleepiness following a night of ad lib extended sleep. The wornout syndrome described by Globus [1969] was attributed to taking extra sleep by sleeping later into the morning 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 awakening 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 detrimental effects on subjective sleepiness or mood the following day [e.g. Carskadon and Dement, 1979; Roehrs and Roth, 1989; Wehr et al., 1993].

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In contrast to these studies, subjects in the current study kept to their normal bedtimes. Manipulations of sleep duration were aimed at altering the time of awakening. Subjects awoke two hours earlier than normal for sleep restriction, and two hours later than usual for sleep extension conditions. It was necessary to standardise the duration of excess sleep, since subjects mood responses following sleep extension were to be compared to mood after a comparable reduction in sleep length.

The duration and structure of sleep is context dependent. It is influenced by a whole array of internal and external factors, such as the prevailing social, environmental, physiological and psychological conditions. Horne [1991] proposed that at least two types of sleepiness exist, resulting from a need for core or optional sleepiness. Optional sleepiness can be overcome more readily by motivational influences to maintain performance. Core sleepiness results in a more profound and overwhelming urge to sleep. Nonetheless, without sufficient alternatives or incentives to remain awake, optional sleepiness will also lead to the onset of sleep.

Changes in subjective states following the opportunity to extend sleep over 'long' periods have been reported. Wehr et al. [1993] compared the effects of 28 extended sleep periods (14h) to 7 normal nights (8h). Sleep extension produced improvements in self-reported vigor and fatigue and were viewed by the researchers as evidence of a pre-existing sleep deficit. However, Totterdell et al. [1984] observed that enhanced mood throughout the day was related to an earlier than normal sleep onset during the previous night and not to total sleep length. Therefore, the subjective consequences attributed to extended sleep were likely to be confounded by factors other than sleep duration. This was further highlighted in a repeated measures study of sleep duration and sleep quality in college students [Hawkins and Shaw, 1992]. Differences in sleep duration on weekday nights and at weekends were found to coincide with an improvement in subjective sleep quality during extended sleep at weekends. However, a corresponding reduction in sleep quality was not observed when sleep duration was restricted during periods when the subjects had reduced amounts of sleep (e.g. when studying for exams).

In the current study it was hypothesised that any alterations in mood and sleepiness would be observed after a single night of sleep extension. The effects of two hours excess sleep, for just one night, were examined in relation to subsequent mood. This resembled reality, since most individuals indulge in excess sleep only at weekends and not over prolonged periods. In addition, the potential disruption to subjects' work schedules was too great to examine long-term sleep extension.

Moreover, earlier studies of chronic sleep extension have documented little beneficial or negative effects upon daytime functioning [Wehr et al., 1993; Harrison and Horne, 1996].

The findings reported in this thesis provide evidence to support the theory that an improved psychological state, more specifically mood states, are highly contingent on a delicately maintained balance between sleep and wakefulness. There appears to be a rather narrow band for duration of sleep which is optimal in its physiological effects, with deviations on either side being disruptive. Hence, beyond given temporal limits, sleep itself can become a potentially debilitating and fatiguing process.

7.2 Circadian Rhythms, Mood and Personality

It has been argued that the wornout syndrome may be due primarily to a disruption of circadian mechanisms underlying sleep, rather than to mechanisms associated with the function of sleep. In other words, the detrimental effects of extended sleep on subjective sleepiness or self-rated mood, may have been due to the timing of the extra sleep. Taub and Berger [1976] observed that the inimical effects of sleep extension on subsequent waking behaviour, performance and overall mood were comparable to those documented after sleep restriction. Taub [1980] attributed the cause of performance decrements to phase-delays in accustomed awakening times, rather than to the effects of increased sleep duration. Taub and Berger suggested that impaired performance, which was associated with oversleep, was actually due to circadian desynchronosis, rather than actual sleep duration. In other words, subjects were incapacitated because they were sleeping during periods of accustomed awakening. Their hypothesis suggests that subjects were only adversely affected by extended sleep because they were sleeping at the wrong time of day. However, it is argued that if such a desynchronosis hypothesis is used to explain the wornout syndrome, then surely all subjects should be affected by oversleep to the same extent, because all subjects were increasing the duration of their sleep period by two hours. It is evident, from the findings presented in this thesis, that only certain individuals were adversely affected by oversleep. Therefore it is proposed that the wornout syndrome is directly associated with the duration of sleep per se.

Recent reports suggest that mood throughout the day is enhanced following an earlier than normal bedtime [Totterdell et al., 1994]. Furthermore, it has been proposed that the timing of final awakening, more specifically, the closeness to the

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circadian acrophase of the temperature rhythm, is associated with elevated ratings of sleep quality, feeling refreshed, and ease of awakening [Akerstedt et al., 1994]. However, subjects in the current study were not required to alter their time of going to bed. The manipulations to sleep in the case of sleep extension and sleep restriction were implemented in the morning. It was the time of awakening that was affected by two hours on experimental nights. In the present study, mood state was examined hourly. Taking circadian factors into account, analysis was conducted using two methods: as *real time [clock time]* and as *hours after arising*. Comparing both methods, there were no significant differences in the results. Differences between the present study and that of Totterdell's and Akerstedt's lay primarily in the fact that in the current investigation, subjects maintained their normal bedtime.

Some previous studies have demonstrated that there are probably circadian rhythms in alertness, which is increased in the morning, peaking around 1100-1400 hours, followed by a gradual decline [Folkard; 1985, 1990]. Monk [1983] also suggested that mood is controlled by internal oscillators. Kerkhof [1985] described a difference in morning and evening types such that alertness in morning types culminates much earlier in the morning compared to evening types. Thayer's [1987] model of mood proposes that endogenous rhythms influence our basic moods [e.g. alertness, tiredness, fatigue and energy]. A circadian rhythm was demonstrated in energetic arousal, peaking by late morning, subsiding in the late afternoon and ascending towards a secondary peak by early evening. Thayer observed differences in morning and evening types. Even components of extroversion, more specifically, impulsivity, appeared to affect these mood variables. Thayer attributes such differences to varying levels of energetic arousal. In a study of propensity towards morningness and personality, Adan and Guardia [1993] indicated that morning types revealed an earlier peak in arousal than evening types. A phase-advance was observed in morning types compared to evening types. A similar phase-advance was evident in introverts.

Circadian rhythms in alertness were also identified in the present studies. Differences were observed between morning and evening type subjects in levels of alertness, morning types showing earlier peaks in alertness than evening types. This was particularly evident in the sleep reduction condition. To allow for such circadian factors, all mood scores were evaluated as *change from baseline* rather than as actual values. Diurnal changes in mood were also observed in relation to personality type. For example, introverts reached a peak in alertness earlier than extroverts. Similarly, cautious and tender minded individuals presented an earlier peak in alertness compared to impulsive and tough-minded individuals.

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Considerable overlaps were detected between personality categories in the current study. For instance, there were large overlaps between morningness and introversion and between eveningness and extroversion. Furthermore, substantial overlaps were noted between introverts, emotionally tender minded and cautious individuals and between extroverts, highly impulsive and tough individuals.

In the present studies, it was observed that individuals who maintained regular sleep / wake schedules were more likely to be morning types and introverted, whereas irregular sleepers were more likely to be evening types or extroverted. Similarly, Patkai [1971] reported a significant positive correlation between morningness and introversion and between eveningness and extroversion. Webb and Bonnet [1978] further demonstrated a relationship between morning types and regular sleepers. Furthermore, Revelle [1980] hypothesised that high impulsivity was associated with eveningness. In contrast to Revelle's hypothesis, Larsen [1985] could not demonstrate any correlation between morningness and impulsivity. Meccaci [1986] noted a positive correlation between morning types and neuroticism and between evening types and psychoticism.

7.3 Sleep Inertia and the Wornout Syndrome.

The wornout syndrome is characterised by a period of relatively intense fatigue, diminished vigor, and a reported increase in confusional state. For most individuals, the effects persist up to five hours. However, some individuals report adverse effects upon mood throughout the day following sleep extension.
The wornout syndrome must be differentiated from sleep inertia. This is a transient phenomenon persisting for 5-30 minutes, characterised by feelings of disorientation and light-headedness. In the present investigations, the effects of sleep extension persisted for up to five hours. Clearly, sleep inertia does not last for this length of time and should not be confused with the wornout syndrome.

It is proposed that the degree of disorientation experienced in sleep inertia is directly proportional to the depth of sleep. Many of us can relate to the ill-effects of sleep inertia following a daytime nap. Although subjects in the present study may have experienced sleep inertia upon arising, as the effects should have been transient, they therefore should not have confounded the more long-term effects of the wornout syndrome. In order to allow for any ill-effects of sleep inertia, all subjects completed Mood Logs and Post-Sleep Questionnaires 30 minutes after arising, rather than immediately upon awakening.

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There is no scientific evidence to suggest that feeling rested immediately on arising is a necessary or even likely indication of sleep satiation. For example, Naitoh et al. [1993] described a period of sleep inertia following the termination of sleep as part of the awakening process. Akerstedt et al. [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 awakening in relation to the circadian phase was 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 reported to be improved with reduced sleep efficiency. It was also negatively correlated with total sleep time. The researchers concluded that reduced sleep inertia following more superficial sleep might account for this.

Therefore it cannot be assumed that the ability to wake and feel refreshed immediately will improve with increased prior sleep. Conversely, the process of going from wake to sleep at night is not instantaneous. Hence, it may seem unnatural to expect someone who is sound asleep one moment, to be fully awake and refreshed the next. In conclusion, it is argued that the effects of the wornout syndrome persist for longer than the transient effects of sleep inertia. In the present study, most individuals experiencing the wornout syndrome following sleep extension were affected up to 5 hours. Others, however, were incapacitated to varying degrees for the whole day.

7.4 Are subjects Sleepy or Tired after excess sleep ?

Both sleepiness and tiredness are subjective terms that are often used synonymously in everyday context. However, the terms have quite diverse connotations: sleepiness can be viewed as a characteristic of wanting to sleep, of irresistible drowsiness. Tiredness, however, can mean physical exhaustion, which is a physical awareness of discomfort or mental fatigue, which is often manifested as poor concentration and irritability. It is possible to be tired without necessarily being sleepy: indeed after a hard day's work, we often have a desire to unwind and relax, and quite often this is sufficient to alleviate the sensations of fatigue. On the other hand, it is possible to feel sleepy without being physically or mentally tired. It is important to differentiate between the terms and specify precisely the sensations being experienced.

Although subjective measures of sleepiness have been deemed sensitive enough to gauge alertness/sleepiness, there is still ambiguity surrounding certain measures currently employed. For this reason, it was decided to investigate the structure of the Stanford Sleepiness Scale [SSS]. In the current study, two forms of the SSS were administered: the original version and a 23-item version [after MacLean, 1989]. It was hypothesised that the SSS measures two variables: Sleepiness and Tiredness. Items pertaining to these variables were identified and the number of such items selected by subjects after each experimental condition was compared.

Assessing subjects' responses to the original version of the SSS Questionnaire indicated interesting findings. In study 2, comparable levels of subjective alertness were observed in both the WOS and NWOS groups after baseline sleep. After sleep extension, the WOS group described elevated levels of sleepiness compared to the NWOS group. However, a divergence was exhibited in the NWOS group: a reduction in sleepiness was documented. Following sleep restriction, similar values of enhanced sleepiness were observed in both the WOS and NWOS groups.
The results from the 23-item Sleepiness/Tiredness Questionnaire in study 2 revealed significant findings. Following baseline sleep, the WOS and NWOS groups indicated trends towards selecting more 'tiredness' items than 'sleepiness' ones. However, following sleep extension, the WOS group selected more tiredness items than sleepiness ones at all 4 hours tested, while the NWOS group selected more tiredness items at hour 1. Therefore, the WOS group appeared to be more tired than the NWOS group. In addition the WOS group presented more subjective tiredness than sleepiness. Following sleep reduction, similar levels of tiredness and sleepiness were documented in both the WOS and NWOS groups at hour 1. These values declined rapidly for the WOS group but more gradually for subjects in the NWOS group.

Findings from study 3 also indicated that subjects identified as experiencing the wornout syndrome after oversleep selected more items pertinent to the scale measuring tiredness than sleepiness. It could be argued that if subjects were genuinely sleepy after sleep extension, they would have selected a greater number of sleepiness items. Instead, more tiredness-related items were chosen, thus providing further evidence for the notion of a wornout syndrome following oversleep. The divergence in selection of tiredness and sleepiness items between subjects in the WOS and NWOS groups was significant after sleep extension. The WOS group preferred more tiredness items after oversleep. In contrast, after sleep reduction, subjects in the NWOS group selected a higher number of both tiredness

and sleepiness items, relative to the WOS group. Subjects in the NWOS group described a decline in tiredness and sleepiness after sleep extension relative to baseline. Both groups were affected similarly after sleep reduction.

Dinges [1989] acknowledged the multidimensionality of sleepiness, this was reflected by the diverse measures of assessment currently employed. Considerable ambiguity surrounds the various appraisal measures used for sleepiness. Sleepiness is predominantly viewed only from an operational standpoint, for example in terms of sleep onset latency [MSLT studies] and performance decrements in simple psychological tasks. Although these indices are extremely sensitive to sleepiness, this standardisation may have inadvertently constrained our thinking regarding sleepiness. The aim of the present discussion is to provoke further thought about the presumption that sleepiness is such a direct and unitary concept. In fact, as the data presented shows, *tiredness* is frequently mistaken for *sleepiness*.

Laverne and Johnson [1992] observed considerable overlaps between the POMS and measures of subjective sleepiness. They perceived that subjects with enhanced ratings of sleepiness also judged their mood as poorer than individuals with depleted estimates of sleepiness. Laverne and Johnson support Broughton's [1992] view of different states of sleepiness. Broughton proposed that sleepiness from undersleeping and sleepiness from oversleeping produced qualitative differences in subjective states and in performance. This finding was supported by results obtained in the present study: after oversleeping subjects were actually more tired than sleepy.

Carskadon and Dement [1986] observed distinct shifts towards enhanced sleepiness, following oversleep, as reflected by the SSS. It is argued that the SSS confounds at least two independent factors described in POMS terminology as vigor and fatigue. Therefore the reason why oversleeping bias SSS scores in a direction towards greater sleepiness is because participants respond to the 'fatigue' descriptors more than they do to those associated with 'sleepiness'.

MacLean et al. [1989, 1992] examined the structure of the SSS. They focused on two issues: the extent to which the component items of which each level of the SSS is constructed are homogenous and the factorial structure of these items. A 24-item version of the SSS was constructed, examining each item on the SSS separately. This scale was administered to subjects alongside the original. Considerable heterogeneity was observed in subjects responses. MacLean et al. proposed that the

SSS constituted two factors: an activation factor and a sleepiness factor. They concluded that the descriptors defining each level of the SSS were not homogenous. In addition they proposed that there was little relationship between the SSS and the two factors and speculated that factor 1 [activation] was closely aligned to Horne's core sleep and factor 2 [sleepiness] to optional sleepiness.

The divergence in selection of sleepiness and tiredness items noted in the present study, provides support for MacLean's theory that the SSS is not a unidimensional measure of sleepiness. If this were the case, variation would not have been observed. Instead, the disparity was evident, especially after sleep extension. Analyses of the responses indicated that endorsement of items on the revised SSS scale was not consistent with the SSS level endorsed, indicating that the descriptors at each scale level are not equivalent. It was apparent that subjects in the WOS group were responding to the tiredness dimension rather than to the sleepiness one. Dement [1976] distinguished between sleepiness and physical tiredness; this distinction was supported by Moldofsky [1989] following clinical observations of patients.

Analysing the structure of the SSS revealed that some of the adjectives employed were not synonymous with the alertness-sleepiness dimension. Several studies of oversleep have reported that ensuing MSLT scores do not support there being an increase in sleepiness [Carskadon et al., 1986]. If anything, scores on the MSLT lengthen, seemingly in the direction of enhanced alertness. Paradoxically, subjective ratings of sleepiness, reflected by the SSS, reveal distinct shifts towards increased sleepiness. Analysis of the structure of the SSS has highlighted its ambiguous nature. Several of the statements relate to the wornout syndrome and to the POMS fatigue dimension rather than to the more traditional alertness-sleepiness one [synonymous with POMS vigor]. Various statements are identical to fatigue, which is independent of vigor. After sleep extension, subjects responded to the fatigue scale more than they did to the sleepiness one. This could explain why MSLT scores remain unchanged or improve. Such scores are independent of fatigue or the wornout syndrome. However, after sleep reduction, where there must be real sleepiness, subjects respond more to the sleepiness descriptors of the SSS. This is endorsed by decrements in MSLT scores.

It is argued that the propensity to oversleep in the morning is not necessarily indicative of chronic sleep loss, but may simply be a manifestation of Horne's optional sleepiness. The additional sleep results in little substantive improvement to

daytime alertness. Current findings have indicated that for many individuals, excess sleep may be counterproductive for mood, manifesting in the wornout syndrome. The multifaceted nature of sleepiness has been recognised by several researchers. Although Dement [1976] maintained that sleepiness was "an independent feeling state" he remarked on the overlap between sleepiness, physical tiredness, and depression and lack of initiative. MacLean et al [1992] identified two factors, tentatively named activation and sleepiness. Present results provide evidence to support MacLean's findings relating to the sleepiness / tiredness ambiguity of the SSS. Thus distinguishing between the scales should facilitate and enhance our understanding of the nature of subjective sleepiness.

The findings reported here have revealed that sleepiness induced by oversleeping appears to be qualitatively different from that induced by sleep restriction. A wornout syndrome characterised by distinguishable feelings of fatigue, exhaustion and lethargy was observed after sleep extension. By contrast, sleep restriction induced sensations of increased sleepiness, irritability and diminished vigor. These findings provide support for the view that sleepiness is a multifarious state [e.g. Broughton, 1982]. In conclusion, the interaction between sleep duration and mood is extremely complex, and highly influential in our everyday lives.

C

A Clinical Dimension

8

Chronic Fatigue Syndrome and Sleep

8. A Clinical Dimension

Chronic Fatigue Syndrome and Sleep.

8.1 Introduction

Chronic Fatigue Syndrome has symptoms similar to the Wornout Syndrome, particularly the intense fatigue experienced by many 'healthy' individuals after arising from Sleep Extension. One area of particular interest, and of relevance to the Wornout Syndrome, is the amount of sleep chronically fatigued patients obtain. It is well documented that many CFS sufferers are advised to avoid expending energy unnecessarily and to take plenty of bedrest [Spracklen, 1988; Dawes, 1991]: inadvertently, these chronically fatigued patients may be indulging in excessive sleep. It is argued that this excess sleep may produce symptoms characteristic of the Wornout Syndrome, which in turn, may confound or exacerbate the symptoms of CFS.

8.2 Psychopathology and the Chronic Fatigue Syndrome.

The major symptom reported by CFS sufferers is one of overwhelming fatigue. However, this symptom is prominent in various medical, psychiatric and sleep disorders. Patients with CFS also complain of psychiatric symptoms such as depression, anxiety and phobias [Behan and Bakheit, 1991]. The paucity of physical signs in patients with CFS and the lack of objective laboratory tests to confirm the diagnosis has led some to conclude that some psychiatric illness is the cause of this fatigue seen in these patients. For example, sporadic cases of the disorder such as the outbreak which occurred at the Royal Free Hospital in 1955 have been attributed to mass hysteria [McEvedy and Beard, 1970].

Researchers have argued that if CFS patients with psychiatric diagnoses are excluded, then CFS is a very rare condition [e.g. Manu, Lane and Matthews,1988]. The majority of cases of fatigue can be attributed solely to a psychiatric disorder, only 2.7 percent received a partly medical diagnosis [Manu et al., 1993]. Furthermore, it has been suggested that CFS may merely represent a somatization disorder in patients with a predisposition to psychiatric disease [Cluff, 1991]. In accordance with these findings, Llewellyn [1996] argues that patients with true CFS are rare, and that depression is the most common cause of fatigue in clinical practice. Llewellyn suggests that these 'non-depressed, non-somatizing, fatigued patients' be reclassified as 'neurasthenic'.

In terms of psychiatric diagnoses of chronically fatigued patients, David et al. [1991] suggest that 50 percent have major depression, 6 percent dysthymia, 13 percent somatoform disorders, 8 percent anxiety disorders and 23 percent have no psychiatric diagnosis. Some investigators have suggested that there has been an over-emphasis on the psychiatric morbidity associated with CFS. Indeed, Hickie et al. [1990], concluded (in contrast to David et al. [1991]) that the pattern of psychiatric symptoms in CFS patients was no higher than in general community patients. Shanks and Ho-Yen [1994] observed a prevalence in chronically fatigued patients of 45 percent for psychiatric disorder. They suggest that the frequency and type of psychiatric symptoms associated with CFS will depend on the population characteristics and duration of fatigue.

More recently, Farmer [1995] has pointed out that 50 percent of all CFS patients also have depressive or anxiety disorders, but 15 percent of all CFS patients have no psychiatric diagnosis at all. Most CFS studies attain that between two-thirds and three-quarters of all patients with CFS have psychiatric disorders [e.g. Kroenke et al., 1988; David, 1991; Lane et al., 1991; Katon and Walker, 1993]. It is argued that if the fatigue syndrome is a depressive disorder, it is more likely to be a vegetative atypical depressive illness [White, 1992]. Many of the symptoms of depression are very similar to those documented in the CFS; infact several investigators have proposed that the two conditions are very closely linked [e.g. Wessely and Powell, 1988; Manu et al., 1993]. A study investigating 405 chronically fatigued patients revealed the major diagnoses to be depression, panic disorder and somatization disorder [Ray, 1991].

Disturbances of memory and concentration are frequently reported by patients with CFS. Straus [1988] documented lack of concentration in 90 percent of his patients. Smith [1991] perceived that CFS patients were slower at psychomotor tasks, had problems with selective and sustained attention, certain memory impairments and also enhanced sensitivity to visually disturbing patterns. In contrast to this, Riccio et al. [1992] noted that CFS patients were only impaired on the ability to recall information from a story and on a paired associate learning task. Patients with CFS generally complain of depression and other forms of psychopathology. These psychiatric symptoms *per se* may lead to reduced arousal and attention, lapses of concentration and impairment of memory [Weingartner, 1986]. It is important to understand whether problems of memory and concentration in patients with CFS are independent of, or secondary to, psychopathology.

Smith et al. [1993] investigated behavioural problems and mood in CFS and realised that, compared to controls, patients rated themselves less alert, more feeble, less clear-headed, more clumsy, more lethargic, more discontented, more mentally slow, more dreamy, more incompetent, sadder, and more depressed than controls. Patients also reported significantly higher levels of depression, anxiety, physical symptoms and cognitive failures. The 57 patients were slower on psychomotor tasks, showed increased visual sensitivity and impaired attention. Digit span and free recall were not impaired but retrieval from semantic memory and logical reasoning were slower. Smith et al. concluded that the performance differences between patients and controls could not be attributed to differences in psychopathology. They maintained that it was necessary to consider the nature of the neurological dysfunction underlying these effects.

Taerk [1987] indicated that 16 of 24 CFS patients he investigated were also suffering from major depression. Twelve of the 24 had a history of affective disorder prior to the 'fatiguing illness'. However the diagnostic criteria used by Taerk is criticised. He included fatigue as a symptom of psychiatric disorder, thus introducing an unwarranted circularity into the results.

Wessely and Powell [1989] compared a group of CFS patients [n=47] to a group of depressed patients [n=26] and a third group of patients with peripheral neuromuscular fatiguing illnesses (myasthenia gravis, myopathies, Guillian-Barre syndrome) [n=33]. Findings revealed that all patients were severely physically fatigued, but that mental fatigue was equally prominent in the CFS and affective group, but markedly less in the neuromuscular controls. Ninety-six percent of CFS patients experienced fatigue following physical effort compared to 56 percent of depressed patients and 91 percent of neuromuscular patients. However, following mental effort, 89 percent of CFS and 80 percent of depressed patients experienced fatigue compared to 45 percent of neuromuscular patients. Overall, physical fatigue was prominent in CFS but also in depression and neuromuscular disorder, implying that subjective complaints of physical fatigue were of little use diagnostically. Mental fatigue, however, was equally common in both depression and CFS but only occurred in peripheral neuromuscular disorders if there was coexisting psychiatric illness. The researchers concluded that CFS was defined by mental and physical fatigue, since mental fatigue was found in CFS irrespective of psychiatric disorder. However the same pattern of fatigue was observed in the depressed controls, suggesting a considerable overlap between CFS and affective disorder. Wessely and Powell argued that the majority of cases of CFS could be

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explained by disorders of mood; 47 percent were cases of affective disorder, 25 percent had other psychiatric diagnoses, and the remaining 28 percent had no psychiatric disorder. However 10 of the 13 without psychiatric disorder had disturbances of sleep and / or appetite.

Kroenke et al. [1988] also found a close association between unexplained fatigue and emotional disorder. Symptoms of depression were recognised in 56 percent of the fatigued sample, but no diagnoses were made. Manu et al. [1988] studied 100 patients whose principle complaint was chronic fatigue. They concluded that a psychiatric disorder was a major cause of the fatigue in 66 percent of cases. In 5 patients, the chronic fatigue was related to a medical condition and in the other 31 it remained idiopathic.

The role of depression and other psychiatric features in CFS is ambiguous. The syndrome may be multifactorial in origin, with somatic and psychological factors acting together to produce fatigue and other symptoms. Evidence of the organic nature of CFS has accumulated in the last few years but is still extremely controversial. One series of studies has been concerned with abnormalities in the muscles of CFS patients [Gow and Behan, 1991]. Additional research has focused on neuroendocrine and immunological abnormalities [Behan and Bakheit, 1991]. Many viruses have been implicated in the aetiology of CFS, notably the Coxsackie viruses [Gray, 1984], but also varicella, influenza and Epstein-Barr viruses [Tobi et al., 1982].

Fibromyalgia and CFS have fascinating parallels, especially as regards fatigue, myalgia, muscle weakness, headaches and sleep disturbance [Goldenberg, 1989]. Fibromyalgia was often considered to be a manifestation of hysteria and was equated with psychogenic rheumatism in the 1950's and 1960's. However, recent controlled studies pointed out that patients with this syndrome had uniform, stable and reproducible symptoms and signs, in contrast to the bizarre and changeable symptoms of hysteria. Psychological profiles of patients with fibromyalgia demonstrated elevated scores for depression, hypochondriasis and hysteria on the Minnesota Multiphasic Personality Inventory (MMPI), [Ahles et al., 1984].

The most compelling evidence for psychopathology in fibromyalgia and CFS has been the recognition, in recent reports, of a prevalence of 20 to 70 percent of major depression [Hudson and Pope, 1985]. These researchers explored three possible hypotheses to account for the association between fibromyalgia and depression.

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The first was that fibromyalgia was simply a manifestation of depression. However, many patients with fibromyalgia were neither depressed nor met criteria for psychiatric diagnosis at the time of their fibromyalgia. The responsiveness to antidepressant medication has also provided a link with depression. It has been suggested that this may be secondary to sleep or other effects. A second hypothesis was that depression was caused by fibromyalgia. This could be consistent with a reactive depression; moreover, Goldenberg demonstrated that depression generally antedated symptoms of fibromyalgia by many years. A family history of depression was also documented in most fibromyalgia cases, providing further evidence for a psychobiologic but not a cause and effect association of fibromyalgia and depression. A third hypothesis was that a common pathophysiological abnormality could account for the overlapping features of fibromyalgia, CFS and depression. In addition, irritable bowel syndrome was considered to be intimately related to the above mentioned conditions. Hudson and Pope [1985] observed that psychiatric diagnoses derived from the Diagnostic Interview Schedule were similar in patients with CFS and fibromyalgia, with a preponderance of mood disorders, anxiety disorders and somatization.

8.3 Sleep Disturbance and Chronic Fatigue Syndrome.

In addition to persistent debilitating fatigue, exacerbated after exercise, patients with CFS present with symptoms of myalgia, headache, sore throat and cognitive disability. Sleep disturbance is a prominent complaint in chronically fatigued patients. Studies examining sleep in CFS will be reviewed briefly. Moldofsky [1988] investigated sleep in 10 patients with CFS and 10 healthy controls for three nights in the sleep laboratory. Overnight changes in mood, fatigue and sleepiness were assessed using the Wahler Symptom Inventory, the Beck Depression Inventory and the SCL-90-R. All of the CFS patients described non-restorative sleep upon arising in the morning. Two patients exhibited sleep apnoea and one had nocturnal myoclonus. Nine of the ten patients exhibited an alpha non-REM sleep anomaly with significantly enhanced alpha EEG sleep observed than amongst the control subjects. This was identified as the presence of alpha wave intrusions during non-REM sleep [also referred to as 'alpha-delta sleep']. An identical sleep physiologic disorder, an alpha non-REM sleep anomaly was identified in fibrositis patients [Moldofsky, 1989] . This non restorative sleep physiology was associated with chronic fatigue and diffuse myalgia after a flu-like illness, it was related to unrefreshing sleep and overnight increases in muscle tenderness. Moldofsky suggested that such a febrile illness may trigger alterations in sleep-wake brain and immune functions in patients with fibrositis or CFS.

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Normally, during the day, a quiet state of wakefulness is accompanied by an alpha (7.5 to 11 Hz) EEG frequency distributed over the occiput of the scalp. With sleep onset the alpha wave pattern typically disappears. Stimulation's to induce behavioural arousal's are accompanied by the appearance of the alpha frequency in the sleep EEG. However, in patients with fibromyalgia, the alpha frequency appears over the central and prefrontal regions of the scalp during sleep. This alpha EEG sleep tends to be prominent during the first third of the night when delta sleep (0.5 to 2.5 Hz) predominates and usually occupies about 60 percent of total sleep time. It tends to abate over the course of the night and usually disappears during REM sleep. Subjectively patients report their sleep to be very light. Due to the overlap in symptomatology between CFS and Fibrositis Moldofsky suggested that the features observed may reflect such a common underlying psychophysiological arousal state during sleep.

Moldofsky [1993] has observed that total sleep deprivation, partial or selective sleep deprivation studies may induce many of the behavioural and somatic features characteristic of fibromyalgia and CFS. It is well documented that sleep deprivation causes sleepiness, fatigue, negative mood and impaired intellectual functioning. These disturbances in behaviour are reminiscent of the psychological difficulties described by chronically fatigued patients. Horne [1988] has reported that prolonged wakefulness, as well as resulting in sleepiness, fatigue and emotional distress, also leads to cognitive and temporal disorganisation. Additionally, both concentration and vigilance is impaired and subjects become forgetful, performing poorly on memory tasks. However, Horne has observed that these symptoms are reversible with slow wave sleep.

With selective deprivation of slow wave sleep, subjects become physically uncomfortable, less aggressive and manifest concern over vague physical complaints and changes in bodily feelings [Agnew et al., 1967]. Moldofsky [1979] observed that three nights of stage 4 sleep deprivation resulted in musculoskeletal aching, stiffness, generalised heaviness and unusual somatic fatigue. Some subjects experienced nausea, diarrhoea and loss of appetite, along with depression and irritability. All these symptoms subsided over the subsequent two nights of undisturbed sleep. In a later study, Moldofsky and Scarisbrook [1976] reported that behavioural symptoms could not be induced in physically fit individuals in whom stage 4 sleep was disrupted by noise.

In contrast to Moldofsky's findings of an alpha non-REM sleep anomaly in chronically fatigued patients, Guilleminault and Mondini [1986] observed hypersomnia associated with a normal sleep architecture in twelve patients with infectious mononucleosis. Cognitive problems associated with short periods of 'microsleep' were also documented during the daytime. The difference between the two studies was that Moldofsky's sample did not have a history of classic infectious mononucleosis.

The sleep profiles of ten patients (5M, 5F) with CFS were studied [Krupp and Mendelson, 1990]. In nine of the ten patients, a psychiatric or sleep disorder was identified, which may have contributed to their symptomatology (two patients had sleep apnoea, three patients had nocturnal myoclonus, two had a narcolepsy - like disorder and two were clinically depressed). In a later study, Krupp and Mendelson [1993] compared the sleep and fatigue characteristics of 72 patients with CFS to 57 Multiple Sclerosis (MS) patients and 40 healthy controls. Polysomnography was carried out in 16 patients, all had a sleep disturbance as one of their major symptoms in the initial interview. In 10 of the 16 patients polysomnography revealed clinically significant, but treatable sleep abnormalities (e.g. excessive daytime sleepiness, sleep apnoea and narcolepsy). In addition, they observed that although several patients had major depression or anxiety disorders, in most cases these were considered to be concurrent with the chronic fatigue rather than the primary or exclusive cause of symptoms. Contrasting the findings of the three groups, CFS patients had significantly elevated sleep disturbance, depression and fatigue (particularly striking since MS is a well recognised cause of persistent severe fatigue [Freal et al. 1984]). Krupp and Mendelson observed that sleep disturbance in CFS was not significantly correlated with fatigue severity - but the correlation of 0.26 between sleep disturbance and depressive symptoms suggested that depressive symptoms could account for seven percent of the variance of sleep disturbance. This emphasised the importance of taking psychological factors into account. Polysomnography revealed that two patients with CFS had alpha wave intrusion; this has been associated with fibromyalgia [Moldofsky, 1989], a condition which overlaps considerably with CFS [Komaroff and Goldenberg, 1989]. However, their study failed to produce polysomnographic data on healthy controls for comparison.

Morriss et al. [1993] investigated sleep abnormalities polysomnographically in 12 patients with CFS. Findings revealed that seven patients with CFS had a sleep disorder (four had difficulty maintaining sleep, one had difficulty getting to sleep,

one had difficulty in both initiating and maintaining sleep and one had hypersomnia). The authors concluded that it was most likely that it was these sleep disorders that were contributing to daytime fatigue.

Various research studies indicate that the amalgam of disordered sleep physiology, chronic fatigue, myalgia and cognitive and behavioural symptoms constitutes a non-restorative sleep syndrome that may follow a febrile illness, as in the CFS. Moldofsky [1993] indicated that both CFS and fibrositis have similar disordered sleep physiology that accompanies increased nocturnal vigilance and light, unrefreshing sleep. This may result in psychological distress, that re-enforces the perpetuation of sleep-related symptoms. Interestingly, Shimizu [1992] induced this form of sleep disturbance in healthy controls by depriving subjects of slow wave sleep. Healthy controls described myalgia, fatigue and stiffness upon awakening.

Manu et al. [1994] described polysomnographic findings from 30 chronically fatigued patients. Alpha-delta sleep was identified in 26 percent, depression in 67 percent, CFS in 50 percent and fibromyalgia in 13 percent of patients. In addition, 33 percent had a primary sleep disorder [sleep apnoea, periodic leg movements or narcolepsy]. The researchers concluded that alpha-delta sleep was not significantly correlated with fibromyalgia or depression. However, the sleep anomaly was significantly more common among patients who had chronic fatigue without major depression.

More recently, Buchwald et al. [1994] examined the prevalence of sleep disorders among chronically fatigued patients. Fifty-nine patients were studied polysomnographically, 81 percent had at least one sleep disorder, (sleep apnoea [44 percent] and idiopathic hypersomnia [12 percent]). In addition, 41 percent of patients had abnormal results for a multiple sleep latency test. They concluded that chronically fatigued patients with suggestive symptoms may have potentially treatable coexisting sleep disorders, that are not associated with meeting criteria for CFS.

Vercoulen et al. [1994] developed a multidimensional approach to the assessment of CFS in 298 patients. The questionnaires enquired into sleep disturbances in addition to other areas and revealed that 43 percent of patients reported sleep disturbances spontaneously. However, when asked specifically, 61 percent documented sleep disturbances [of those patients, 56 percent had difficulty falling asleep, 67 percent had restless sleep and 27 percent complained of early morning awakening]. An interesting finding emerged regarding duration of sleep in chronically fatigued patients. The average duration of sleep at night was 8 hours

[range 3-14] and during the day, 2 hours [range 1-11]. These findings indicate that during any 24 hour day, CFS patients obtained an average of 10 hours of sleep. This is clearly indicative of a longer than normal sleep duration. It is well documented [e.g. Webb and Agnew, 1975] that healthy young adults sleep on average 7-8 hours. All the information for Vercoulen et al's study relied on self-report and was conducted by questionnaires. No objective measures, for example actometers or polysomnographic data, were used to validate the findings.

Hypersomniac's frequently complain of chronic fatigue and sleepiness throughout the day, prolonged deep nocturnal sleep and difficulty in arousal. The daytime drowsiness may be interpreted as a feature of depressive illness, particularly since hypersomnia fluctuates and is associated with mild cyclical mood changes. Roth [1980] observed that 79 of 167 patients with hypersomnia had psychological problems, and 24 were depressed. The characteristic feature of hypersomnia is that of deep nocturnal sleep with few awakenings. This can be compared to the multiple arousals typical in depressed patients. Roth suggested that patient's with idiopathic hypersomnia, sleeping 8-12 hours per night and obtaining lengthy non-refreshing daytime naps, had an excess of slow wave sleep at night with a high arousal threshold. This was possibly associated with a lower setting of the ascending reticular activating system (ARAS).

In a multicenter study of 2,234 patients presenting with a complaint of excessive somnolence [Coleman et al., 1982] fewer than five percent were attributed to psychiatric disease after a sleep disorders centre evaluation. Admittedly chronic fatigue and excessive somnolence are not one and the same. Since patients often have trouble making the subtle distinction between fatigue and sleepiness it is likely that these studies evaluated similar patients. Huller [1990] stressed the importance of exploring the possibility of a sleep-wake disorder in addition to other medical and psychiatric disorders in patients presenting with chronic fatigue, since sleep-wake disorders may explain the overwhelming fatigue in certain cases. Patients complaining of chronic fatigue are frequently given different labels by different specialists. Examples of labels include fibromyalgia, chronic viral or fungal illness, hypoglycaemia, irritable bowel syndrome and sleep disorders. The unifying assumption is that a primary physical disorder causes chronic fatigue, with resultant psychological distress.

Although the above mentioned researchers considered specific sleep abnormalities in CFS, the area of sleep duration and its association to fatigue has not been

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investigated thoroughly. This is an area of direct interest to the present investigations on the relationship between sleep and mood. The studies reported here may have a bearing on the Chronic Fatigue Syndrome. Results of my previous study investigating the 'wornout syndrome' in healthy adults indicated that sleep length, can in certain personality types, be highly influential on subsequent mood. Oversleeping can have adverse effects on mood; this has been reflected by reports of elevated fatigue and confusion associated with diminished vigor. Clearly, many healthy adults experienced symptoms very similar to CFS following sleep extension. Oversleeping in healthy adults proved, in some individuals, to be more detrimental to mood than sleep restriction. Relating these findings to clinical studies in depressed patients, it has been illustrated that sleep deprivation enhances mood [Pflug, 1976] to such an extent that the depressed feelings subside until the following sleep period. Undoubtedly there is a strong association between sleep and mood. My next study will investigate the sleeping habits of CFS sufferers. It is hypothesised that one explanation for the overwhelming fatigue may be oversleeping. More specifically, it is proposed that the symptoms of CFS may be confounded by the effects of the Wornout Syndrome.

Subjects

Participants with CFS were recruited via the local ME Association [Leicestershire, England] and through advertisements placed in the local paper. Although a large number of subjects volunteered, care had to be taken in screening subjects due to the controversial nature of the illness and lack of objective testing for the diagnosis of CFS. All respondents were initially interviewed over the telephone; only those who had been diagnosed by their General Practitioner or other health professional were approached for a second interview. One stipulation was that symptoms had to be present for at least six months. Subjects were required to be free from major medical conditions and those subjects receiving medication affecting the CNS were eliminated. Six subjects met the criteria for CFS, two were depressed and one had general aches and pains (all nine subjects believed they had ME). In addition, three healthy subjects who were free of any complaints of daytime sleepiness and with healthy, regular nocturnal sleep patterns were recruited as controls. The group of subjects consisted of ten females and two males, with an age range of 24-52.

Table 8.1 presents details of subjects sleep patterns, age, gender, duration of illness and medication. It also identifies events surrounding the onset of their illness. The

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clinical group consisted of 7 females and 2 males with an average age of 39 years
[range 24 to 52]. The duration of illness ranged from 10 months to 8 years with an
average duration of 3.5 years.

Table 8.1 Details of Illness and Sleep Patterns

Ss / Group	Age / Sex	Duration of Illness	Illness at Onset	Usual Sleep Pattern	Medication
1 CFS	26 / F	2 y	Fever for 2 days	2300 - 0800 + Daytime naps	Vitamins, garlic capsules, germanium.
2 CFS	26 / F	10 m	Non-specific "flu-like" illness	2200 - 0800 + Daytime naps	Nystatin, Codeine.
3 CFS	38 / F	5 y	None	2400 - 1200 + Daytime naps	Vitamin C, essential fatty acids
4 CFS	48 / M	5 y	Non-specific "flu-like" illness	2200 - 0600 + Daytime naps	None
5 CFS	24 / F	4 y	Glandular fever	2100 - 0800 + Daytime naps	Vitamins A + C, germanium, zinc.
6 CFS	43 / F	18 m	Gastric infection	2300 - 0900 + Daytime naps	Vitamins, garlic, evening primrose oil.
7 Depression	52 / F	8 y	None	2400 - 0900 + Daytime naps	None
8 Depression	40 / F	3 y	Fever	0100-1200	Codeine
9 Aches / Pains	52 / M	2 y	Intestinal Disorder	2300 - 0600 + Daytime naps	Temazepam [discontinued before study].
10 Control	40 / F	N /A	N / A	2300 - 0700	None
11 Control	26 / F	N / A	N / A	2400 - 0800	None
12 Control	40 / F	N / A	N / A	2300 - 0600	None

There was a great deal of overlap in the symptomatology reported by subjects, and
this is noted in table 8.2 overleaf, which compares the symptoms described by
subjects at the time of the study.

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Table 8.2 Symptoms exhibited at time of study.

Symptom	CFS N=6	%	Depress-ion N=2	%	Aches/ Pains N=1	%
Muscular Fatigue	6	100	2	100	1	100
Leg/Neck Pain	6	100	2	100	1	100
Headache	5	83	1	50	0	0
Nausea	5	83	2	100	1	100
Diarrhoea	4	66	2	100	0	0
Blurred Vision	4	66	2	100	1	100
Hearing Difficulties	2	33	2	100	1	100
Sweating	4	66	2	100	1	100
Hot/Cold Sensitivity	5	83	2	100	1	100
Inability to Concentrate	6	100	2	100	1	100
Impaired Memory	6	100	2	100	1	100
Mental Confusion	5	83	1	50	1	100
Sore Throat	4	66	0	0	0	0
Dizziness	5	83	1	50	0	0
Depression	6	100	2	100	1	100
Speech Difficulties	4	66	1	50	1	100
Vivid Dreams	3	50	1	50	0	0
Loss of Balance	6	100	2	100	0	0
General Fatigue	6	100	2	100	1	100
Breathing Problems	3	50	1	50	0	0
Feverishness	3	50	1	50	0	0
Bouts of Crying	6	100	2	100	1	100

Design and Aims of the Study.

The primary objective of the study was to examine the sleep patterns of CFS sufferers, using objective and subjective measures. One hypothesis was that patients with CFS would exhibit an alpha non-REM sleep anomaly, comparable to that identified in Fibromyalgia (due to the similarity in symptoms). It was also hypothesised that CFS patients were oversleeping and that this was one cause of the overwhelming fatigue. This hypothesis was based on the findings of the previous study.

Apparatus

i). Ambulatory Activity Monitor

Actometers were worn on the wrist throughout the study [Type: Z80-32K V1; available from: Gaehwiler Electronics, Eichtalstrasse 20, CH-8634 Hombrechtikon, Switzerland]. They monitored subjects' diurnal levels of activity and their sleep/wake patterns. Actometers were set to sample at 1-minute epochs, and were attached to the dominant wrist.

ii). Medilog 4-24 Recorder

Ambulatory sleep monitoring devices were utilised to record 3 channels of data: electroencephalography [EEG], electromyography [EMG] and electrooculography [EOG]. Electrodes were applied to the subjects scalp 1 hour before retiring and removed upon awakening the following morning. The medilog recorder logs 4 channels of data [EEG, EMG and two EOG's] continuously for up to 24 hrs on a C-120 tape. Sleep is scanned at high speed on a Page Mode Display - the PMD-12 to give clear results in the form of hypnograms and sleep statistics.

Measures

The measures included:-   i)   ME Questionnaire
ii)   General Sleep and Health Questionnaire
iii)   Beck Depression Inventory
iv)   Pre-Sleep Questionnaire
v)   Post-Sleep Questionnaire

i). ME Questionnaire

This enquired into:-   a. Events surrounding onset of illness.
b. Whether CFS was preceded by any viral or other illness.
c. Present status of the illness.
d. Symptoms experienced presently.
e. Changes made to lifestyle due to illness.

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ii). General Sleep and Health Questionnaire

This determined:-
    a. Subjects' usual quality and quantity of sleep including timing and duration of daytime naps.
    b. Medication used currently.
    c. Detailed history of illnesses and/or medical problems.

iii). Beck Depression Inventory [BDI]

This scale [Beck et al. 1961] was used to assess depressive symptoms in patients.

iv). Pre-Sleep Questionnaire

This determined:-
    a. Time of retiring and going to sleep.
    b. Subjective sleepiness, measured on the SSS.

v). Post-Sleep Questionnaire

This determined:-
    a. Time of awakening.
    b. Quality of sleep.
    c. Severity of night-time pain.
    d. Subjective sleepiness, measured on the SSS.

Procedure

Subjects were interviewed in their own homes to elicit information concerning the illness, its onset, duration and severity of symptoms. Subjects were required to complete the ME Questionnaire, the Sleep Questionnaire, and the BDI. Those deemed suitable for the study [free from CNS medication and willingness to participate] were recruited. All participants were required to wear an actometer and complete daily diaries for one-week to determine their usual sleep patterns.

Following screening, subjects underwent polysomnography for three consecutive nights, in their own homes. An ambulatory monitoring device was used and electrodes applied 1 hour before the subjects usual bedtime. Electrodes were attached in accordance with the standard method described by Rechstaffen and Kales [1968]. A1-C3 and A2-C4 EEG, EOG and EMG recordings were made for

each subject. Skin was cleaned with methylated spirits, prior to attachment of silver-silver chloride electrodes to the scalp, using collodion glue, dried with pressurised air. For application of skin electrodes, double-sided adhesive discs were used and secured with micropore tape. Saline 'neptic' gel was inserted into the electrode cavity using a syringe with a blunted needle to increase conductivity. A blunted disposable 'orange stick' was used to scarify the skin. Electrode resistance was reduced to less than 5 kΩ prior to commencing the recording, and checked using a resistance meter. Subjects were instructed to switch on the Medilog just before going to bed and to switch it off after arising the following morning. Electrodes were removed each morning by the experimenter and reapplied the following evening. Actometers were worn throughout the study and subjects were required to complete pre and post-sleep [30 mins after awakening] questionnaires everyday for the duration of the 3-day study.

Results

Sleep Quality
Subjective ratings of Pre and Post-Sleep Quality are displayed below.

Table 8.3 Pre and Post Sleep scores on the SSS [Mean scores].

Category	Pre-Sleep SSS	Post-Sleep SSS
CFS [N = 6]	5.0	4.2
Depression [N = 2]	5.5	3.0
Aches/Pains [N = 1]	3.0	3.0
Controls [N = 3]	3.0	2.3

key:
2 = Functioning at high level, but not at peak, able to concentrate
3 = Relaxed, awake, not at full alertness, responsive
4 = A little foggy, not at peak, let down
5 = Fogginess, beginning to lose interest in remaining awake, slowed down

Due to the small sample size, the above data was not subjected to statistical analysis. However, certain trends emerged, suggesting that Depressed individuals reported a state of greater sleepiness prior to nocturnal sleep [SSS score = 5.5]. This was followed closely by CFS sufferers whose pre-sleep score was 5.0. Both these groups described increased subjective sleepiness at night compared to the Controls and the Aches and Pains subjects. Following a night of sleep, CFS sufferers presented elevated sleepiness upon awakening [SSS = 4.2]. This was in contrast to the remaining subjects, who reported levels of sleepiness between 2.3 and 3.0.

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Comparisons were made on the night-time sleep / wake profile across conditions [see table 8.4]. In terms of difficulty getting up, subjects in the CFS group found it difficult. In contrast subjects in all other groups described their level of difficulty as moderate to easy. In terms of difficulty getting to sleep, all groups reported that it was the same or easier than usual. When asked about restlessness during sleep, the CFS group claimed it was the same as usual. The CFS group described the quality of their sleep to be normal whilst the other groups reported an improved sleep quality. Subjects were also asked about the severity of night time pain. Controls reported none, depressed patients described very mild pain, CFS sufferers detailed mild pain, and the patient with aches and pains presented moderate pain.

Table 8.4 Subjective Sleep / Wake Profile [Mean and standard error].

	CFS	Depression	Aches / Pains	Controls
Difficulty Getting to Sleep 2=harder than usual 3=same as usual 4=easier than usual	3.1 (.2)	3.5 (.5)	3.0 (0)	3.0 (0)
Restlessness During Sleep 2=more restless 3=as usual 4=less restless	3.0 (.2)	4.0 (.2)	3.5 (.5)	3.3 (.3)
Quality of Sleep 2=better than normal 3=normal 4=worse than normal	3.1 (.2)	2.0 (.4)	2.5 (.5)	2.7 (.3)
Difficulty Getting Up 2=difficult 3=moderate 4=easy	2.2 (.2)	3.3 (.2)	3.5 (.5)	4.0 (.6)
Severity of night time pain 1=none 3=mild 5=moderate 7=severe	3.2 (.5)	2.0 (.5)	5.0 (0)	1.0 (0)

Polysomnography

The standard method of scoring episodes of electroencephalography in terms of wake and sleep stages developed by Rechstaffen and Kales [1968] is perhaps the most widespread and frequently used approach to the scoring of electroencephalograms. Following the recommendations of techniques for the detection and recording of EEG sleep, records are separated into discrete epochs of an arbitrary duration - usually between 20-30 seconds. A single score [e.g. wake, movement, sleep stages one to four, REM sleep] is assigned to each epoch

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according to criteria relating to both frequency and amplitude of the generated signal. In healthy young adults, sleep should consist of the following proportions: 5% is wakefulness after sleep onset; 2-5% is comprised of Stage 1; 45-55% is Stage 2; 3-8% consists of Stage 3; 10-15% is Stage 4; and 20-25% is REM sleep. EEGs were scored into sleep stages in 1-min epochs using the Oxford Medilog sleep stage software (based on the standardised criteria for the scoring of human sleep developed by Rechstaffen and Kales, 1968). Hypnograms detailing all this information were obtained for each night [see Appendix]. Table 8.5 shows the quality of sleep experienced by all 12 subjects following 3 nights of polysomnography. Table 8.6 displays the mean polysomnographic findings of the 4 groups, CFS, Depression, Aches and Pains, and Controls.

Table 8.5 Polysomnographic findings of CFS, Depression, Aches and Pains and Control Subjects.

Group	Ss	Age / Sex	TST [mins]	TSWS [mins]	REML [mins]	TREM [mins]	WASO [mins]
CFS	1	26 / F	446	126	66	100	41
CFS	2	26 / F	525	127	88.5	89	86.5
CFS	3	38 / F	544.5	106	79.5	118	53.5
CFS	4	48 / M	467	artefact	artefact	artefact	21
CFS	5	24 / F	531	123	63	206.3	41.5
CFS	6	43 / F	474.5	47.5	artefact	artefact	44.5
Depression	7	52 / F	367.5	68	67	109.7	35
Depression	8	40 / F	431	67	74	182.5	24
Aches/Pains	9	52 / M	359.5	0	51.5	192	26
Control	10	26 / F	390	88	80	80	0
Control	11	40 / F	409	34	109	95	6
Control	12	40 / F	405	65	69	89	20

[TSWS - Total slow wave sleep per night ; REML - REM latency (time before first period of REM sleep); TST - total sleep time; WASO - wake after sleep onset; all values are mean values calculated from 3 nights EEG recording].

The results indicate that the CFS group slept longer than other groups [an extra 100 minutes], had more SWS and were awake for a longer period of time following sleep onset than the other groups. In addition, all the CFS patients were taking

daytime naps [range 40-200 minutes]. These findings would strongly suggest that CFS patients were oversleeping when compared to controls.

Table 8.6 Mean Polysomnographic findings of CFS [N=6], Depression [N=2], Aches and Pains [N=1] and Control [N=3] Subjects.

Group	TST [mins]	TSWS [mins]	TSWS %	REML [mins]	TREM [mins]	TREM %	WASO [mins]
CFS	498.0	105.9	21.3	74.3	128.3	25.8	48.0
Depression	399.3	67.5	16.9	70.5	146.1	36.6	29.5
Aches / Pains	359.5	0	0	51.5	192.0	53.4	26.0
Controls	401.3	62.0	15.5	86.0	88.0	21.9	8.7

Discussion

Although only a small number of subjects were studied, all six met accepted criteria for the chronic fatigue syndrome. They were chosen to participate in the study only if they had been given a diagnosis of CFS by a health professional. Their selection was not based on reported problems with sleeping. Enquiries were made relating to subjects' sleep patterns only after they had been selected to participate in the study. An additional three patients were recruited: two were clinically depressed and one had aches and pains comparable to those observed in fibromyalgia. All subjects were monitored electroencephalographically for three nights. Their subjective reports of sleep correlated well with their electroencephalograms.

Patients in the CFS group clearly slept longer than controls. Chronically fatigued patients obtained an additional 100 minutes of nocturnal sleep, a further 40-200 minutes of sleep was acquired in daytime naps. Their polysomnograms indicated that they obtained twice as much SWS as controls. As a percentage of TST, the amount of SWS gained by CFS sufferers was 21.3 percent compared to 15.5 percent in controls. This is a striking finding, providing evidence to suggest that enhanced levels of SWS in CFS sufferers could be a significant contributory factor to the condition. REM latencies were comparable across chronically fatigued and depressed patients, though slightly lower than controls. The total amount of REM

sleep attained was 25.8 percent in the CFS group, 36.6 percent in depressed patients and 21.9 percent in controls. In addition to enhanced levels of SWS, chronically fatigued patients presented more time awake after sleep onset; 48 minutes compared to 29.5 minutes in depressives and 8 minutes in healthy controls. Comparing these objective measures of restlessness after sleep onset to subjective restlessness during sleep, as measured on the Post-Sleep Questionnaire, reveals divergent findings. Subjective reports showed that restlessness in CFS patients was the 'same as usual' [see Table 8.4]. Although EEG data indicated increased restlessness compared to controls, this was not confirmed by subjective reports.

From subjective responses on the Post-Sleep SSS [completed upon awakening] it was evident that CFS sufferers described elevated levels of sleepiness compared to controls. They reported difficulty getting up compared to subjects in the other groups who described considerably less distress. Contrasting these findings to those obtained in the previous study [examining healthy adults following sleep extension], both CFS patients and subjects who experienced the wornout syndrome complained of difficulty getting up in the morning. In addition both groups reported elevated sleepiness upon arising, as measured by the SSS.

Findings from this study have indicated that CFS patients sleep for a considerably longer duration than controls. It was proposed that excess sleep could exacerbate the symptoms experienced by chronically fatigued patients. It could be argued that because excess sleep was obtained, these patients were also experiencing the wornout syndrome. This could account for the 'sleepiness' [as measured by the SSS] that they described upon arising. The ambiguity of the SSS has already been discussed in relation to both tiredness and sleepiness dimensions. Hence it is proposed that CFS patients were checking the tiredness items rather than those pertaining to sleepiness. Unfortunately, the revised version of the SSS was not administered to chronically fatigued patients in this study. However, prior studies investigating daytime sleepiness in chronically fatigued patients using MSLT techniques have yielded contrasting findings. Researchers observed that MSLT records were normal [e.g. Whelton et al., 1988; Mahowald et al., 1988]. Additionally, these researchers examined nocturnal sleep and found no indication to suggest that chronically fatigued patients were obtaining more than the usual amount of sleep.

Since chronically fatigued patients are generally too exhausted to do anything, they are often advised that the best treatment for them is rest. However, if this is

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interpreted as bedrest, patients are presented with an increased opportunity to nap and sleep whenever they are so inclined. It is proposed that this leads to the accumulation of excessive sleep, both at nightime and then possibly at irregular intervals throughout the daytime in the form of naps. Daytime naps often result in sleep inertia, the ill-effects of sleep inertia coupled with the wornout syndrome may exacerbate the CFS. Due to the considerable overlap in symptomatology between the wornout syndrome and the chronic fatigue syndrome, it was difficult to ascertain precisely the aetiology of the multitude of symptoms presented by these individuals.

Excessive sleep is frequently documented in the milder neurotic depressions, particularly among young adults, as a defensive response which accomplishes withdrawal from painful reality. This could lead to a vicious circle, whereby excessive sleep, initially a defensive manoeuvre, begins to maintain the depression and anergia as part of the syndrome associated with excessive sleep.

An alpha non-REM sleep anomaly was identified in earlier studies of chronically fatigued patients [Moldofsky, 1989]. In alpha-delta sleep, stage 4 sleep is generally absent. REM sleep is largely unaffected although the time between successive REM periods is increased. Alpha-delta sleep replaces delta sleep, early in the night and opposite to REM sleep. Alpha activity [7-11 Hz] usually occurs during relaxed wakefulness when the eyes are closed, and is related to basal metabolic rates. This abnormality in sleep has also been observed in the slow wave sleep of patients with fibromyalgia. Moldofsky proposed that the muscular pain and stiffness presented in fibromyalgia may be mediated by a specific slow wave sleep disturbance. This sleep anomaly was interpreted as a physiological indicator of a state of partial wakefulness, or an arousal type of disorder within sleep. Alpha-delta sleep was first observed in neurotic depressives who complained of somatic malaise and fatigue. It has been suggested that alpha-delta sleep disrupts the restorative role of slow wave sleep, and this is why subjects report enhanced fatigue upon awakening.

The alpha non-REM sleep abnormality described in CFS patients [Moldofsky, 1989] was not observed in the current sample of chronically fatigued patients. Whereas Moldofsky [1989] documented increasing amounts of alpha activity associated with diminishing levels of delta sleep in CFS patients, current findings have indicated an increase in the amount of slow wave sleep. However, the one patient with aches/pains in this study indicated an absence of SWS, in addition to excessive amounts of REM activity [53.4%].

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The question remains as to the origins of the excess SWS and its precise role as a contributory factor in fatigue. It is generally accepted that SWS is associated with enhanced tissue restitution, it is only during SWS that the cerebral cortex can 'relax and recover' [Horne, 1985]. It was suggested that increasing brain work during wakefulness, by raising brain temperature [e.g. as in exercise], or by sustained arousal, leads to elevated SWS. Horne suggested that the effects of exercise on sleep were probably an artefact of the accompanying increase in cerebral temperature and metabolism. The increase in cerebral metabolic rate somehow initiates these SWS increases. Horne suspects that more 'wear and tear' is caused by more 'brain work', thus requiring an increased need for cerebral restitution; or there is a build up of SWS-promoting substances in the brain.

In the present study, CFS patients obtained twice the amount of SWS, compared to controls. The reason for this is unclear. However, in studies of sleep extension, the reappearance of SWS 12 hours after sleep onset has been well documented [e.g. Webb, 1986; Horne, 1988; Gagnon and De Koninck, 1990]. Webb suggests that SWS is an endogenous programmed system, and the resurgence is due to a REM/SWS interaction. Horne [1988] argues that the reappearance of SWS might be a secondary effect of the increased wakefulness after sleep onset found mainly in the last 3 hours of extended sleep. Horne postulates that the additional REM sleep resulting from oversleep encourages the generation of SWS (since REM sleep closely resembles wakefulness). In addition, he argues that one reason for the resurgence of SWS in the delayed sleep extended condition is that subjects are awake for an extra 4 hours and thus require the extra SWS. However, Gagnon and DeKoninck [1990] indicated that the delayed SWS peak 13.5 hours after sleep onset could not be explained by greater prior wakefulness; they ascertained that the less the amount of prior wakefulness or light sleep (and the greater the prior SWS) the larger the magnitude of the delayed SWS peak. Results from the present study indicated that CFS patients slept on average, for 10 to 12 hours per day. The sleep period was not continuous, however; it comprised of daytime naps in addition to nocturnal sleep. Therefore, it is argued that the secondary peak in SWS, observed after 12 to 13 hours of sleep, is not directly relevant to the present study.

The amount of SWS varies with maturation, peaking in neonates and infants. At the age of 2.6 years, stage 4 sleep amounts to 17.3 percent of total sleep time, thereafter decreasing with age [Monti, 1981]. SWS is altered in many disease states, it is reduced in unipolar depression, hypothyroidism and schizophrenia. However, in hyperthyroidism and Addison's Disease, it is increased, compared

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with normal controls. It is possible that the alterations in the amount of SWS are due to some metabolic or hormonal factors. Since the aetiology of the CFS remains unclear, maybe some endogenous factors are contributing to the increase of SWS in CFS patients. Further research aimed at reducing the amount of SWS in CFS patients, perhaps by administering low doses of aspirin [see Horne and Shackell, 1987] may yield interesting findings.

The results presented in this thesis strongly suggest that chronically fatigued patients have a sleep abnormality. Although the chronic fatigue syndrome is extremely perplexing, it is proposed that reducing the total time spent asleep may help these sufferers. This is a difficult task though, since most chronically fatigued patients seem to be caught in a vicious circle. They feel exhausted, lack energy for even mundane tasks and are constantly advised by health-care workers to rest as much as possible. However, by following their own instincts and others guidelines, some of the time spent resting is inadvertently spent asleep. As well as disrupting their sleep pattern, which leads to a whole area of nocturnal sleep problems in itself, more deleterious excess sleep is acquired. Earlier studies investigating the effects of excess sleep on mood have indicated that this excess sleep can produce symptoms characteristic of the wornout syndrome. Rather than producing beneficial effects, mood is adversely dampened.

Findings from the present study provide a compelling argument implying that excess sleep may be important in the aetiology of the chronic fatigue syndrome. The subjective symptoms characteristic of the wornout syndrome observed in healthy adults comprise sensations of tiredness and weakness. Additionally, subjects complain of muscular aches, poor concentration and feeling unrefreshed after oversleep. These sensations closely resemble many symptoms described by sufferers of the chronic fatigue syndrome. In conclusion, it is suggested that chronically fatigued patients regulate and reduce their total sleep time, say to within normal limits and eliminate daytime napping behaviour. Perhaps this modification in sleep schedules will aid in curtailing the suffering of these individuals, who are afflicted with such a perplexing constellation of symptoms.

Relationship between the Wornout Syndrome and the Chronic Fatigue Syndrome.

The association between sleep and mood is extremely complex and the studies presented in this thesis were undertaken to further our understanding of the nature of this alliance. The first three studies compared the differential effects of sleep extension and sleep reduction on mood in healthy adults. Following a preliminary analysis of the differences in mood states between baseline, extension and restriction of sleep, mood changes were re-assessed in subgroups of subjects [e.g. introverts vs extroverts] following manipulations in sleep length. Findings indicated that certain subjects were adversely affected by changes in sleep length: some by sleep extension, others by sleep reduction, some subjects were impaired by both conditions, and a small number of subjects were immune to the effects of sleep manipulations altogether.

The results from studies 2 and 3 indicated the significance of personality and individual differences in the relationship between mood states and sleep duration. Certain trends and significant findings emerged, suggesting that introverts, emotionally tenderminded, morning types and cautious individuals were more susceptible to the adverse effects of oversleep. However, these findings must be interpreted with caution, due to the relatively small number of subjects in each study. Nevertheless, it is clear from the present findings that the effects of sleep duration on subsequent mood is modulated by the personality of the individual.

Mood states were compared after sleep extension and sleep restriction and contrasting forms of 'sleepiness' were observed after oversleep compared to sleep restriction. Sleep extension led to a Wornout Syndrome, characterised by distinguishable feelings of fatigue, exhaustion, lethargy and impaired concentration. In contrast, sleep restriction produced symptoms of increased sleepiness, diminished vigor and irritability.

There is considerable overlap in the symptomatology in both the Wornout Syndrome and the Chronic Fatigue Syndrome, specifically the intense fatigue, lethargy and impaired concentration. Importantly, the effects of the Wornout Syndrome only lasted 4 to 5 hours in most cases, compared to the long-term suffering endured by chronically fatigued patients. This common symptomatology does suggest a possible link between the two conditions. CFS sufferers are commonly advised to rest, and spend much of their time in bed. Thus, in Study 4, it

was hypothesised that these patients were indulging in excessive sleep. On this basis, it was proposed that the Wornout Syndrome was a confounding factor that contributed to the Chronic Fatigue Syndrome.

Assessing the sleep patterns of CFS sufferers yielded interesting findings. When compared to age-matched controls, these patients were obtaining, on average, 100 minutes of additional nocturnal sleep, and a further 40 to 200 minutes of sleep was acquired in the form of daytime naps. Due to the small sample size, individual differences were not assessed, and mood states were not examined, primarily because this study was conducted in parallel with Study 1 and the mood questionnaires were still in the process of being developed. However, overall findings strongly support the notion that excess sleep promotes the symptoms of the chronic fatigue syndrome, manifesting in the wornout syndrome. Chronically fatigued patients may be better advised to regulate their sleep habits and reduce their total sleep time, to avoid the possible effects of the Wornout Syndrome.

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10

Appendices

10. Appendices

Appendix 1: Questionnaires

1. Sleep Questionnaire

NAME............................   ADDRESS ....
                                                  ....
AGE ....

SEX ....   CIGARETTES SMOKED/DAY.......

HEIGHT ....   ALCOHOL INTAKE/DAY.............

WEIGHT ....   PRESENT OR MOST RECENT
                                          OCCUPATION ....
MARITAL STATUS ....

Please answer all the questions EITHER by circling the letter OR by answering
the questions as fully as possible.

1. How well do you normally sleep at night? ....

      a. Very well
      b. Satisfactorily
      c. Some problems
      d. Poorly

2. What is your usual time of settling down to go to sleep?

      a. During the week ....

      b. At weekends   ....

3. How long does it usually take you to fall asleep after turning out the light?

      a. Less than 10 mins.
      b. Up to 30 mins.
      c. No more than 1 hour
      d. More than 1 hour - please specify ....

4. How would you describe your level of wakefulness prior to going to bed?

      a. Active
      b. Alert
      c. Lively
      d. Stimulated
      e. Drowsy
      f. Sluggish
      g. Sleepy

5. How many times do you wake up during the night, on average?

    a.   Never
    b.   1-2 times a night
    c.   2-4 times
    d.   4-6 times
    e.   More than 6 times - please specify ....

6. If you wake up during the night, what usually causes this?

    a.   I don't know. Awake spontaneously
    b.   Nervous tension, worries
    c.   Need to pass urine
    d.   Shortness of breath or coughing
    e.   Noise
    f.   Pain(s) felt in the (specify) ....
    g.   Other causes (please specify) ....

7. If you wake up during the night how long does it usually take you to go back to sleep?

    a.   Less than 10 mins.
    b.   Up to 30 mins.
    c.   No more than 1 hour
    d.   Longer - please specify ....

8. Do you lie awake worrying at night?

    a.   Every night
    b.   Most nights a week
    c.   Several times a month
    d.   Once a month or less
    e.   Never

9. Do you have difficulty waking up in the mornings?

    a.   Never
    b.   Occasionally
    c.   Frequently
    d.   Always

10. What time do you usually wake up in the mornings?

    a.   During the week ....

    b.   At the weekends ....

11. Which of the following best describes how you feel in the mornings?

    a.   Peaceful
    b.   Contented
    c.   Calm
    d.   Tense
    e.   Uneasy
    f.   Distressed

12. Do you regularly wake up with any of the following ?

    a.   Aching - Where? ....
    b.   Pain - Where? ....
    c.   Stiffness - Where? ....
    d.   Tenderness - Where? ....
    e.   Cramps - Where? ....
    f.   Headache
    g.   Bad temper
    h.   Lethargy

13. Do you ever have difficulty staying awake during the day?

    a.   Never
    b.   Most days a week
    c.   Several times a month
    d.   Once a month or less

14. If you suffer from daytime sleepiness then when is this sleepiness likely to start and for how long?

    Starts about .... (time) until about .... (time)

15. Do you take daytime naps? YES / NO

    If YES, then   a) What time do you usually take these naps? ....

                       b) On average how long do these naps last for?............................

16. How much does the quality of your sleep vary from one night to the next?

    a.   Very much
    b.   Moderately
    c.   Slightly
    d.   Not at all

17. Do you take sleeping pills ? YES / NO

    If YES, Which pills ? ....

    How often do you take them?

    a.   Every night
    b.   Most nights a week
    c.   Several times a month
    d.   Once a month or less

18. Are you currently taking any prescribed pills or medicines?

    If so, which medicines? ....

    How long have you been taking them? ....

    What are they for? ....

19. Please indicate if you have experienced any of the following using the code below.

In the past	P
At present	N
Regularly	R

Asthma	....
Hay fever	....
Eczema	....
Allergies	....
Thyroid problems	....
Migraine	....
Breathing problems	....
Undue anxiety	....
Stammering	....
Sleepwalking	....
Snoring	....
Nightmares	....
Teethgrinding	....
Difficulty reading/writing	....
Arthritis	....
Rheumatism	....
Fibrositis	....
Depression	....
Heart problems	....
Stomach problems	....

2. Daily Diary

NAME .... DATE ....

0600		1400		2200	
0700		1500		2300	
0800		1600		2400	
0900		1700		0100	
1000		1800		0200	
1100		1900		0300	
1200		2000		0400	
1300		2100		0500	

IN THE MORNING

What time did you go to bed last night ? ....

What time did you go to sleep last night ? ....

What time did you wake up this morning ? ....

What time did you get up this morning ? ....

Which of the following best describes the quality of your sleep last night ?

        a. Much better than normal
        b. Better than normal
        c. Normal
        d. Worse than normal
        e. Much worse than normal

AT BEDTIME

Have you had any naps today ? Yes / No

If YES, please indicate TIME and DURATION of NAPS below

      ....

      ....

And finally, if you removed your ACTIGRAPH today, please indicate

TIME and DURATION below

      ....

3. The Mood Questionnaire

This version was employed in Study 1

THE SLEEP LABORATORY   LOUGHBOROUGH UNIVERSITY

NAME -------------------------   DATE ------------------

PLEASE COMPLETE THIS FORM EVERY HOUR USING THE SCALE IN THE KEY.
PLEASE RECORD YOUR FEELINGS FOR THE PAST HOUR.

TIME (hrs)	0600	0700	0800	0900	1000	1100	1200	1300	1400	1500	1600	1700	1800	1900	2000	2100	2200
ALERT																	
WORN-OUT																	
CONFUSED																	
TENSE																	
ENERGETIC																	
HAPPY																	
DROWSY																	
CLEAR-HEADED																	
SAD																	
CALM																	

4. The Mood Questionnaire [Studies 2 and 3]

KEY
0 Not At All
1 A Little
2 Moderately
3 Quite A Bit
4 Extremely

NAME ------------------------   DATE ------------------

PLEASE COMPLETE THIS FORM EVERY HOUR USING THE SCALE IN THE KEY.
PLEASE RECORD YOUR FEELINGS FOR THE PAST HOUR.

TIME (hrs)	0600	0700	0800	0900	1000	1100	1200	1300	1400	1500	1600	1700	1800	1900	2000	2100	2200
ALERT																	
WORN-OUT																	
ANNOYED																	
TENSE																	
ENERGETIC																	
HAPPY																	
DROWSY																	
CLEAR-HEADED																	
FATIGUED																	
SAD																	
IRRITABLE																	
ACTIVE																	
HOPELESS																	
BAD-TEMPERED																	
CONFUSED																	
WEARY																	
LONELY																	
CALM																	

5. Post Sleep Questionnaire

NAME ....   **DATE** ....

Please complete this form upon arising in the morning.

1. What time did you go to bed last night ? ....

2. What time did you go to sleep last night ? ....

3. How difficult was it getting to sleep last night ?

    a. Much harder than usual
    b. Harder than usual
    c. Same as usual
    d. Easier than usual
    e. Much easier than usual

4. How restless were you last night ?

    a. Much more restless than usual
    b. More restless than usual
    c. As usual
    d. Less restless than usual
    e. Much less restless than usual

5. Which of the following best describes the quality of your sleep last night ?

    a. Much better than normal
    b. Better than normal
    c. Normal
    d. Worse than normal
    e. Much worse than normal

6. What time did you wake up this morning ? ....

7. What time did you get up this morning ? ....

8. How difficult was getting up this morning ?

    a. Very difficult
    b. Difficult
    c. Moderate
    d. Easy
    e. Very easy

6. The SSS Questionnaire

NAME ....

DATE ....

TIME ....

CONDITION ....

Listed below are a set of feelings which reflect various degrees of ALERTNESS and DROWSINESS. Read them carefully and indicate your present state by placing a tick in the appropriate box.

1. ACTIVE, VITAL, ALERT AND WIDE AWAKE .... ☐

2. FUNCTIONING AT A HIGH LEVEL, NOT AT PEAK,

   BUT ABLE TO CONCENTRATE .... ☐

3. RELAXED, AWAKE, NOT AT FULL ALERTNESS, RESPONSIVE .... ☐

4. A LITTLE FOGGY, NOT AT PEAK, LET DOWN .... ☐

5. FOGGINESS, STARTING TO LOSE INTEREST IN REMAINING AWAKE,

   SLOWED DOWN .... ☐

6. SLEEPINESS, PREFERRED TO BE RESTING, FIGHTING SLEEP,

   FEELING WOOSY .... ☐

7. ALMOST UNABLE TO STAY AWAKE, STRUGGLING TO STAY

   AWAKE .... ☐

7. Sleepiness Questionnaire

Listed below are a set of descriptors related to feelings.
Please read each one carefully and indicate your present state
by placing a tick in the appropriate box.

	YES	NO	CAN'T SAY
1. FIGHTING SLEEP	☐	☐	☐
2. A LITTLE FOGGY	☐	☐	☐
3. RELAXED	☐	☐	☐
4. FOGGINESS	☐	☐	☐
5. NOT AT PEAK	☐	☐	☐
6. RESPONSIVE	☐	☐	☐
7. WOOZY	☐	☐	☐
8. PREFER TO BE LYING DOWN	☐	☐	☐
9. SLEEPINESS	☐	☐	☐
10. VITAL	☐	☐	☐
11. SLEEP ONSET SOON	☐	☐	☐
12. ACTIVE	☐	☐	☐
13. ABLE TO CONCENTRATE	☐	☐	☐
14. WIDE AWAKE	☐	☐	☐
15. ALMOST IN REVERIE	☐	☐	☐
16. ALERT	☐	☐	☐
17. FUNCTIONING AT HIGH LEVEL	☐	☐	☐
18. AWAKE	☐	☐	☐
19. SLOWED DOWN	☐	☐	☐
20. LET DOWN	☐	☐	☐
21. LOSING INTEREST IN REMAINING AWAKE	☐	☐	☐
22. LOST STRUGGLE TO REMAIN AWAKE	☐	☐	☐
23. NOT AT FULL ALERTNESS	☐	☐	☐

8. ME Questionnaire

NAME ....   ADDRESS   ....
                                                      ....
AGE   ....

SEX   ....   CIGARETTES SMOKED/DAY   ....

HEIGHT ....   ALCOHOL INTAKE/DAY..........................

WEIGHT ....   PRESENT OR MOST RECENT
                                          OCCUPATION ....
MARITAL STATUS..................   ....

1. How long have you suffered from ME?

2. Did your symptoms of ME occur after a particular illness? If YES, please state illness, how
long it lasted and treatment received.

3. In the 6 months prior to ME, did you suffer from a viral infection? If so, please give details.

4. Would you regard the present status of your illness as

    a) Getting markedly worse
    b) Slightly worse
    c) No change
    d) Improving slightly
    e) Improving greatly

5. Does your G.P. recognise your illness to be ME?

6. Have you sought medical advice through sources other than your G.P.? Please specify.

7. Does any close family member also suffer from ME?

8. ME is said to be characterised by a medley of symptoms. Using the following key indicate those which you consider to be your main symptoms.

Never .... N
Occasionally .... O
Frequently .... F

muscular fatigue after exercise	....	mental confusion	....
leg/neck pain	....	sore throat	....
headaches	....	dizziness	....
nausea	....	depression	....
diarrhoea	....	speech difficulties	....
blurred vision	....	vivid dreams	....
hearing difficulties	....	loss of balance	....
sweating	....	general fatigue	....
sensitivity to heat and cold	....	breathing problems	....
inability to concentrate	....	feverishness	....
impaired memory	....	prone to bouts of crying	....

8. Please state any tests you have had for ME.

9. In the 6 months prior to falling ill, were there any major upheavals in your life? (e.g. moving house or death of a close relative).

10. In the few weeks leading upto the illness were you under any emotional strain, either at home or at work?

11. Since you became ill, what changes have you had to make in your lifestyle? (e.g. have you changed or given up your job?).

12. With regard to hobbies and interests please state any which you currently enjoy and those which you have had to give up due to ME.

Current interests: ....
....

Interests prior to ME: ....
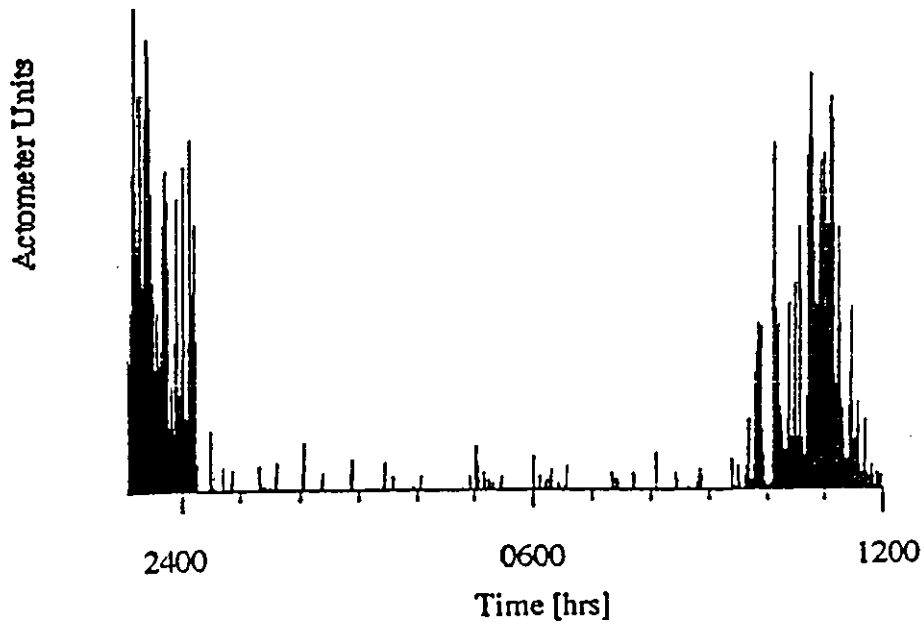..

13. Before becoming ill, did you play any sports or take regular exercise?

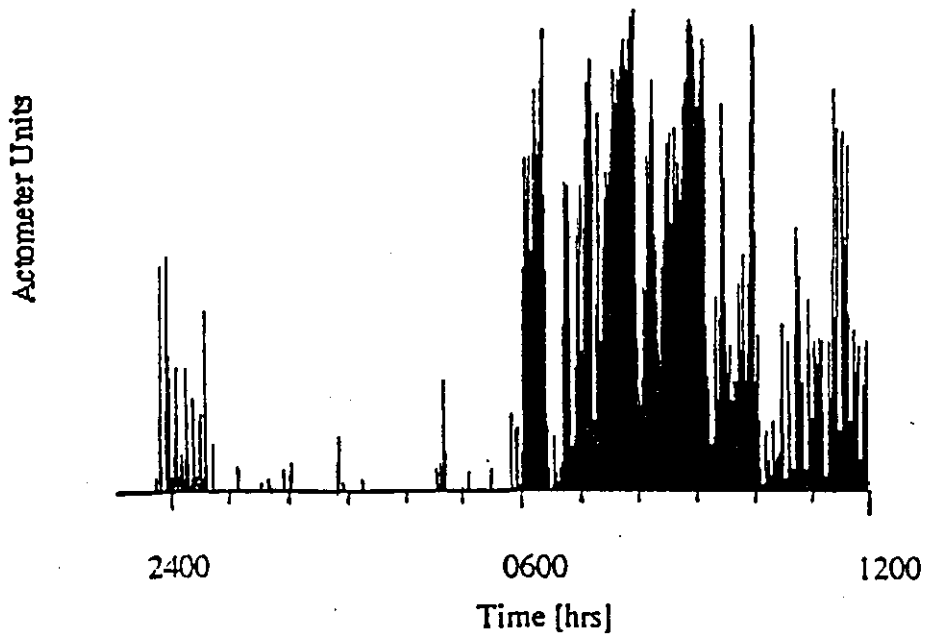14. What exercise, if any, do you take now?

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APPENDIX 2

The following actometer readings were obtained for subject 5 in study 2 after Sleep Extension and Sleep Restriction.
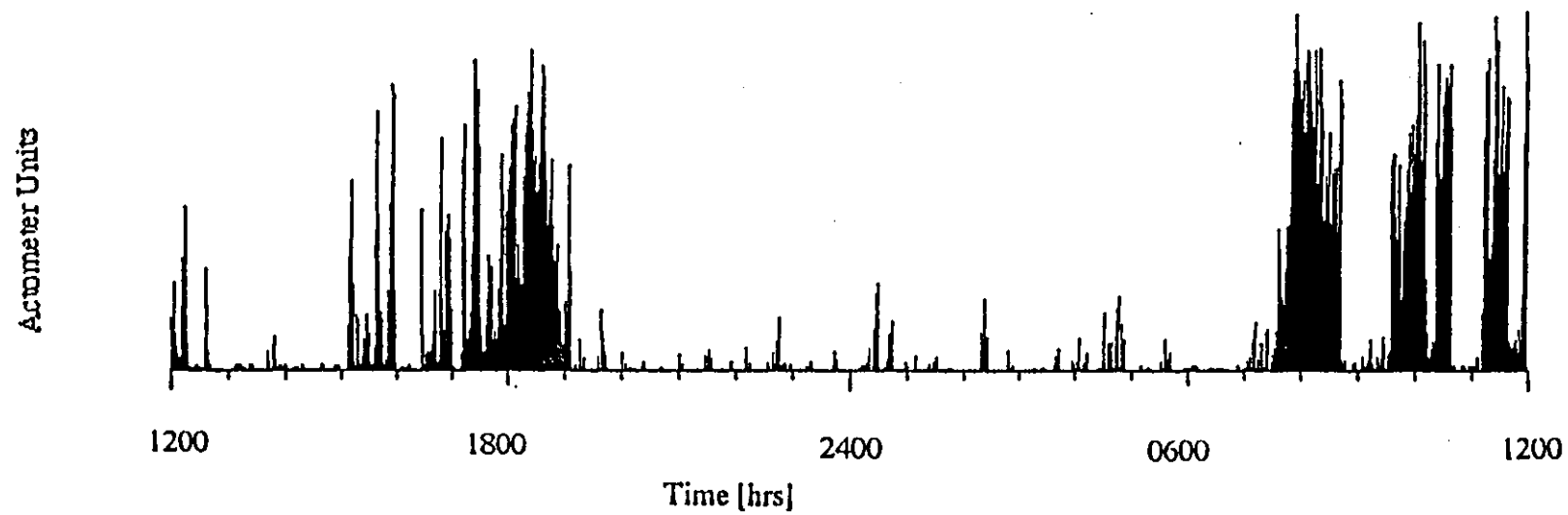
(i) Sleep Extended to 9 hours.
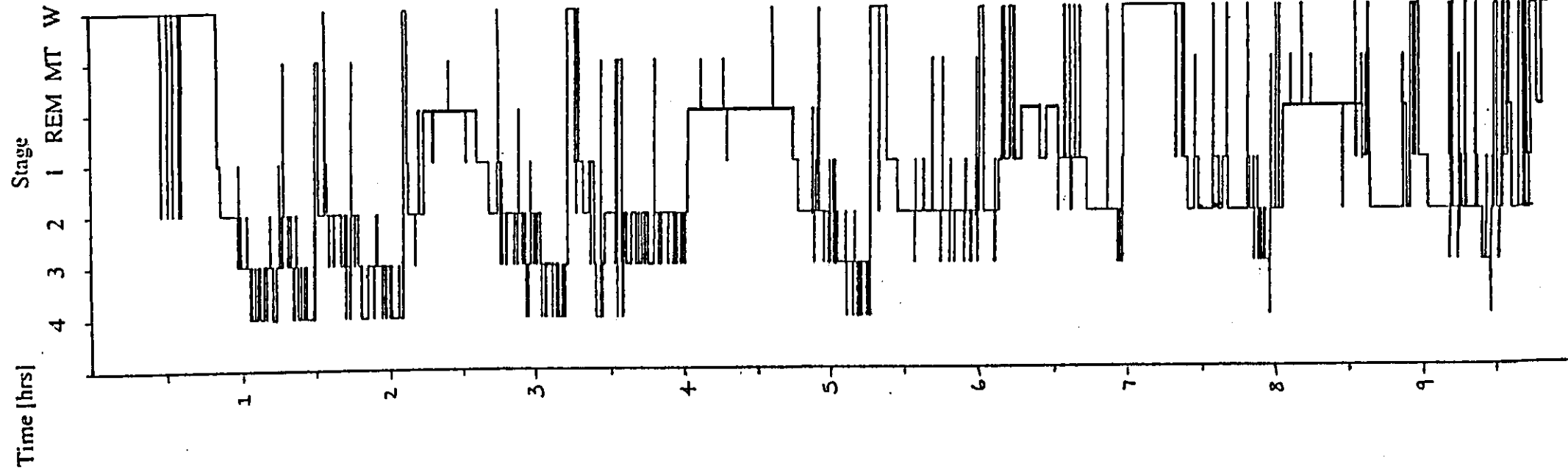

(ii) Sleep Restricted to 5 hours

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The following actometer readings were obtained for a chronically fatigued patient in study 4. Readings were obtained for a 24 hour period.
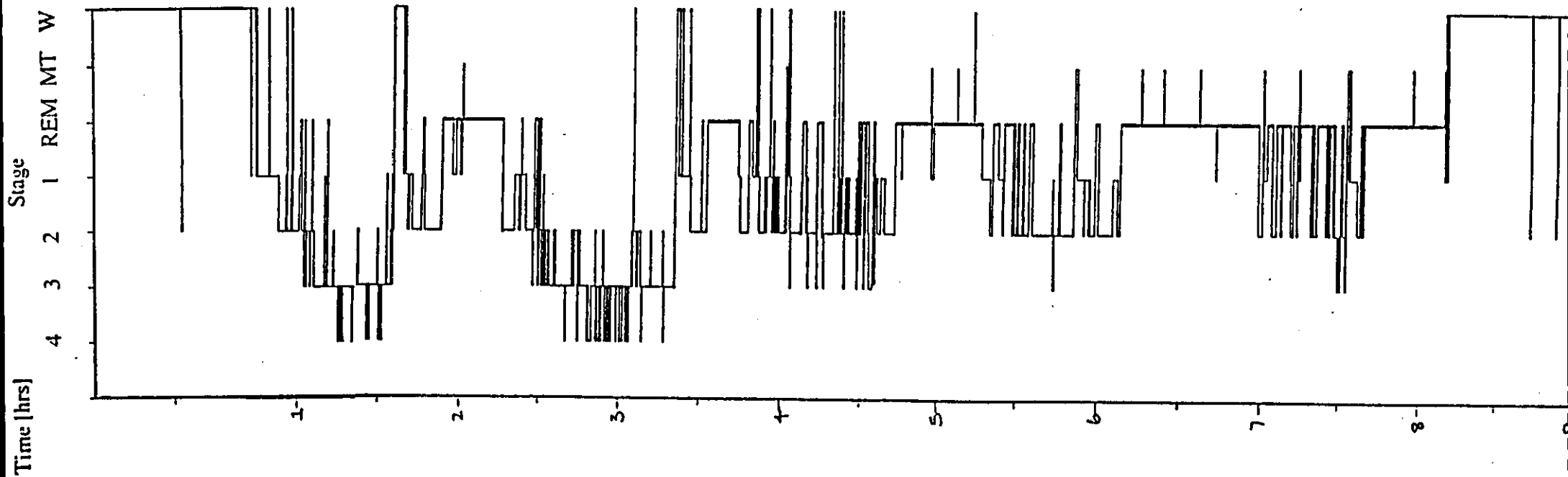
(iii)   Chronic Fatigue Syndrome

Polysomnographic data displaying nocturnal sleep patterns in a Chronically Fatigued patient.

Polysomnographic data displaying nocturnal sleep patterns in a Depressed patient.

Polysomnographic data displaying nocturnal sleep patterns in a healthy control.