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The Afternoon Pressure For Sleep

by

Vijay Mavjee

A Doctoral Thesis

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

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Abstract

Halfway through the waking period, around early afternoon, there is a strong propensity for sleep to occur again. This second sleep tendency is part of a 12-hr. rhythm of sleepiness, that I have termed "The Afternoon Pressure for Sleep" (APS). Evidence for this bicircadian sleepiness, include siesta cultures, short sleep latencies in the afternoon, a midday peak in pathological sleep attacks, and the concentration of napping to this afternoon window. The flexible nature of the human sleep/wake cycle allows for napping to be easily suppressed, as necessitated by social and occupational demands, and so the APS is readily masked.

The work reported in this thesis, represent a concerted effort to 'unmask' this inherent sleepiness. In the first experiment, the afternoon trough was contrasted with the circadian peak of alertness, prominent in the early evening. Forty-eight volunteers were exposed to room temperatures that were either warm or cool, and they were presented with boring or interesting stimuli, at 2 times of day; "afternoon": 1200h – 1600h or, "evening": 1800h – 2200h. There were 8 possible conditions, with 6 subjects assigned to each condition. The experiment was conducted in a climatic chamber. Sleepiness was measured by reaction times, subjective sleepiness scales and mood ratings. Heart rate and body temperature were monitored continuously. Results indicated that boredom-induced sleepiness, was mainly confined to the afternoon, and not the early evening, even though conditions were identical for both groups. Room temperature produced only minor effects. The afternoon trough maximised at 1500h, and the transient nature of this lull, was clearly evident, as subjects showed signs of recovery by late afternoon.

A second study using 48 subjects, examined the post-prandial effects of lunch: a big meal vs. a small meal, after subjects had suffered varying degrees of sleep loss the night before. Three groups of 16 subjects were allowed either: i). a full night sleep; ii). a 2hr. sleep reduction; or, iii). a 4-hr. sleep reduction. Half the members of each group were presented with either a big meal, or a small meal to consume at midday. In addition to the reaction times and sleepiness scales, subjects were also given the Multiple Sleep Latency Test; an EEG based probe of day-time sleepiness. The sleep inducing property of a large meal was only apparent in females, unused to such large meals. All subjects were most sleepy at 1300h – 1330h, irrespective of meal type. Prior night sleep restriction did not modulate the post-prandial trough.

The experimental designs were ecologically valid and innovative, insofar as common elements of the environment were employed in a controlled (and contextually correct) fashion, to unmask the secondary component of the biphasic sleep tendency.

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Dedication

This thesis is dedicated to my mother.

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1

The Afternoon Pressure For Sleep
— A Review —

1 ○ The Afternoon Pressure For Sleep

1. A Review

1.1 Monophasic Sleep

On any summer afternoon, Trafalgar Square, in the heart of London, will be full of people; the throng of tourists and pigeons merging with the din of incessant traffic. If we were to return to this landmark 12 hours later, hardly a soul would be in sight, neither tourist nor satiated pigeon. A nightly curfew has been imposed — decreed by the laws of circadian biology. In the short space of time between the afternoon snapshot with the pigeons, and the desolate hours of the night, the earth has turned on its axis, and light has given way to darkness. London, and its population of 8 million people are asleep. They will wake up at a predictable hour of the morning, and sleep at a predictable hour the following night — all part of the grand circadian phenomenon.

This pattern of sleep can be described as *monophasic* — one consolidated period of sleep at night, followed by a period of wakefulness. The evolutionary roots of this pattern can be deduced from studies of our closest common ancestors, the non-human primates. It is believed that prehistoric man probably enjoyed a polyphasic sleep-wake pattern and it was not until Neanderthal Man (70,000-40,000 B.C.) that the transition from polyphasic sleep to monophasic sleep began¹. The emergence of a fully-fledged monophasic sleep-wake pattern probably occurred during the Neolithic period (since 10,000 B.C.). It is also likely that monophasic sleep arrived by way of a well developed biphasic pattern of sleep, as seen in the chimpanzee. This genetically close primate succumbs to a second period of sleepiness with a lengthy midday nap.

But to what extent is this monophasic pattern of sleep an inherent characteristic of modern man. Aubert and White² realised that whilst sleep was a biological necessity, its structure and patterning had sociological underpinnings. The rules and conditions of sleeping behaviour are largely prescribed by society, through the process of cultural learning. These rules are based on environmental and economic factors, together with beliefs and value-systems that form the custom of the social group, inherited from past generations. The process of retiring to bed is for instance, a highly ritualised affair: each culture with its own particular idiosyncrasy. This may involve changing ones attire

to specified garments, modifying ambient lighting, a pre-sleep religious act of worship and in certain human groups, the adoption of correct postures during sleep: the Chagga tribe of Tanzania will always sleep with their heads pointed towards Mt. Kibo and the Navaho Indian will not allow their infants to sleep with its head upon its hand. The social lore governing sleep behaviour may have been gained through the wisdom of 'grandmother' or through the writings of modern social gurus, the likes of Dr. Spock and others. Taken together, the anthropological evidence would tend to suggest that sleep behaviour is a highly institutionalised affair; a mundane physiological event that is made to acquiesce in the face of tradition.

A universal finding across cultures is the location of the the large consolidated period of sleep, to the dark hours of the night. In terms of human physiology, darkness is not a necessary prime mover for the occurrence of sleep. Yet, it becomes clear that as the infant matures, "culture operates to tilt the [sleep] pattern into a desirable preponderance of night sleeping"³. Such a patterning is not without good reason as Aubert and White have argued. Their reasons can be summarised, thus:

1. Visual isolation prohibits any work, either for the agriculturist or the hunter-gatherer.
2. The 'quiet' of the night facilitates sleep.
3. The fall in night-time temperature favours the withdrawal indoors.
4. Domiciliary confinement not only protects against climate and animals, but it also protects one from social enemies and competitors – "the norm of nightly sleep must be enforced in order to ensure mutually protected rest" – an agreed nightly truce amongst men.
5. A coordinated pattern of simultaneous night-sleeping amongst social groups, somewhat akin to the simultaneity of eating and leisure serves to foster solidarity amongst group members.

For early Man and pre-industrialised cultures, night time sleep may be perfectly valid for the reasons outlined above. But do these reasons still hold for modern Man? Clearly, the regulation of sleeping behaviour, even in a complex society as ours is advantageous especially in regulating the economy, transport needs and other essential services. The somnolent hours of darkness may serve as a time-reservoir when the machinery of society can pause to draw its breath.

Artificial lighting and other technical innovations in our dwellings and cities free us from the physical constraints imposed by nature. With the development of shift-work, computerised and automated control-systems, our world is able to operate on a 24-hour basis. Our needs can be met by day or by night. However, the daylight hours remains as our period of wakefulness — viewed from Kleitman's (1939)^{see 3} evolutionary

perspective: we have progressed from the cyclical “wakefulness of necessity” to a “wakefulness of choice”. The need to synchronise our activities to the changing environment diminishes as we develop controls to ‘master’ our environment. To all intents and purposes, it is now possible to regard the entire 24-hour day as our ‘biological niche’.

Though we may be free from temporal constraints and able to engineer our lifestyles accordingly, it is worth remembering that we are still circadian animals. As in many organisms, a large number of our internal physiological processes are also attuned to a 24-hour endogenous rhythm, e.g. temperature, plasma cortisol, blood pressure and intraocular pressure. These examples of our internal milieu remain tied to a circadian pacemaker, but if there is a separate ‘biological clock’ governing our sleep-wake behaviour, then it seems that in this respect at least, we have “transcended our circadian inheritance of evolution.”⁴ Through the process of cultural conditioning, we have gained the capacity to ignore or overrule our biological clocks at will.

1.2 Biphasic Sleep

Sleep is regulated by society: that much is clear from the discussion above. The influence of social cues is just as potent as the physical zeitgebers of light, or other geophysical phenomena in the entrainment of the sleep rhythms. The culture-bound monophasic system of sleep has an endearing utility to modern day society. But the scientific evidence would suggest that it does not reflect the true biological tendencies of the human sleep-wake organisation.

Halfway through the waking period, around early afternoon, there is a strong urge or propensity for sleep to occur again. This second sleep tendency with its 12-hour rhythm is variously referred to as the Biphasic Sleep Pattern, the Circasemidian Sleep Rhythm, or the Hemicircadian Biorhythm. Essentially, two periods of activity will span from dawn to dusk separated by two troughs – a lengthy nightly sleep and a briefer quiescent period around midday. The same holds true for nocturnal animals, allowing of course for a 180 degree shift in events; so midday quiescence will read midnight quiescence. Whether this 12-hour cycle should be recognised as an aspect of the shorter “ultradian rhythm” or be considered as part of the larger “circadian rhythm” which is characterised by a bimodal pattern, is still subject to debate^{5,6}. Roger Broughton, a Canadian worker from Ottawa has been one of the most prominent scientists of recent times to propound the idea of a midday break in the circadian cycle, which culminates in a secondary period of sleepiness^{5,7,8}. He has assembled a convincing case for this biphasic theory of sleepiness.

In the next few sections of this introduction, I will review the literature which might have a bearing on this proposition of a biphasic sleepiness rhythm – using at times a skeletal framework first employed by Broughton to summarise his ideas⁷. I will expand upon the evidence and present additional data. To begin, a consideration of the diversity of this biphasic pattern will serve to place it within an evolutionary context.

1.2.1 EVOLUTION OF THE BIPHASIC RHYTHM.

The 12-hour secondary sleep tendency is best recognised as a behavioural process that is very much part of our *natural* biology, derived through a process of natural selection in line with the rest of our biology. To view such an aspect of human behaviour in a biological sense, inevitably leads one to consider its wider evolutionary context, and as Lea⁹ has aptly stated; “an evolutionary explanation is often a plausible starter as a way of accounting for behaviour.” Such an explanation harking back to our animal ancestry, brings to the fore the incongruous nature of our monophasic sleep pattern, when one is presented with the fact that most mammals display a polyphasic pattern of rest and activity.

Ethological studies of our non-human primates has led us to draw analogies, and so deduce early man’s probable behaviour. The claim that Neanderthal Man exhibited polyphasic sleep, and the dating of biphasic sleep to the Neolithic era, is based on this deductive paradigm. Our close neighbours on the phylogenetic tree, the apes and Old World Monkeys, have presented us with models by which to consider the evolution of human behaviour (instinctive behaviours not confounded by the impact of complex cultures). The behaviour we are interested in here, is sleep, or more precisely the brief period of sleep that supervenes around midday.

If the original code for the biphasic rhythm was programmed in the remote past of evolutionary history, then it should not be too surprising to find examples in our common ancestors. Indeed, such is the case. This biphasic pattern of inactivity is not unique to humans, examples abound elsewhere in the animal kingdom.

In his much cited paper; ‘Circadian Activity Pattern with Two Peaks’, Aschoff¹⁰ surveyed the activity schedules of numerous species and discovered widespread examples of a daily bimodal pattern of activity. Dawn to dusk activity was punctuated with a midday quiescent period – the biphasic pattern. He noted that some forty-five mammalian species exhibited this pattern, and he even extended his list to include examples of insects, crustaceans, reptiles and birds. Mammalian examples included the elk, the moose, the mule deer, giraffes and the howling monkey. Other authors have also commented on similar observations. The chimpanzee, whose EEG is more akin to humans than to any other mammal, are also accustomed to midday napping according

to ethological observations by Goodall, conducted in the Gombe Reserve¹¹. She describes the enthusiasm with which the chimps' build their 'day nests' prior to their midday nap. Even the slothful adult gorilla is at great pains not to miss its afternoon siesta, and will often stir from its recumbent and contemplative posture to build a day nest, before slipping into a full nap, or simply adopting a semicomatose stupor for the rest of the afternoon, as described by Schaller¹². The more active howler monkeys on Barro Colorado Island, whose day spans from 5–6 am. until its nightly retirement between 7–8 pm., also partake in a substantial midday siesta¹³. The diversity of mammalian sleep patterns has been extensively reviewed in a recent papers by Irene Tobler^{14, 15}. The problem with much of the animal sleep data, concerns the perennial question of the artificiality of laboratory studies, versus field studies. Performing EEG recordings in the latter scenario is of course very difficult – what constitutes sleep for the observing ethologist may not equate with the rigours of the EEG trace. The African elephant (*Laxodonta africana*) has a major sleep period between 0400–0700 h. and usually rests between 1200–1400 h, “when it appears to be dozing”^{see 14}. Are we to class this sleep pattern as monophasic or biphasic? The paucity of EEG data makes this a debating issue for the moment, though modern telemetric advances may one day settle the argument.

Interestingly, the prosimians which are a lower order of primates are neither monophasic nor biphasic, but clearly polyphasic in their sleep-wake behaviour. The ‘exotic’ mammals; elephants, apes and giraffes are found in ‘exotic’ places, with warm climates to match, which makes the midday somnolent retreat appear as quite a natural response to the unpalatable temperatures. As the soaring heat renders all activity impossible and unwise, the ensuing ‘spare time’ would constitute a likely period for the placement of sleep, in the Meddis¹⁶ proposition. However, Aschoff took issue with investigators, who at the time, simply dismissed the midday quiescent period as a response to high temperatures, and/or low humidity values. In his experiments with finches, Aschoff exposed the birds to artificially controlled light and dark cycles – some under constant lighting conditions. Despite the changing conditions, the bimodal activity pattern persisted, surprising even Aschoff by its stability. For these species it was concluded that the biphasic pattern was under the control of an endogenous self-sustaining oscillator. If heat was the all-important factor responsible for the midday lull in activity, how could we explain a similar trough at midnight for nocturnal animals. Whilst the environment may serve to influence the pattern, it does not *produce* the pattern. In evolutionary terms, the utility of such an endogenous biological clock contributes immensely to the fitness of the species. A genetically programmed clock will anticipate the changing environment (eg. light and temperature levels) and allow the animal to adapt in advance. For an animal to survive it must adapt to a “temporally programmed world.”^{see 13}

Although tempting, it would be unwise to proffer a universal pattern, depicting the evolutionary change to sleep structure, in a manner resembling: POLYPHASIC → BIPHASIC → MONOPHASIC. Though, such a linear schematised progression seems likely for Man – accepting the qualification that the monophasic pattern is an artificial socio-cultural construct, undermining the truer innate biphasic proclivity of man. It is interesting to note that during ontogenic development from infancy to old age, we cycle through all, returning once more to polyphasic sleep at the close of life.

Both a biphasic and a polyphasic pattern of sleep have adaptive value; they are more pliant to sudden changes in the environment, so contributing to the species fitness. Sleeping patterns are responsive to, and intertwined with, the changing demands of the environment. Thus, grazing ungulates have to endure an almost permanent state of drowsiness as they remain vigilant to a potentially hostile biotic and abiotic environment, unlike the not-so-timorous carnivores whose consummate, but infrequent feeding habits, ensure much leisure time to nap¹⁷.

To summarise, surveys of animal sleep data, point to the ubiquitous nature of the biphasic sleep pattern. Deductions from our close primate ancestors, present clues to the likely history of this pattern. The role of the environment cannot be underrated in any discussion on phylogenetic factors. Although sleep researchers are “untroubled by any fossil record...[we are nevertheless] ..troubled by an astonishing diversity of sleep patterns among extant species¹⁶.” With the cerebral advancement of hominidae, came the development of sophisticated cultures. For the culture of *today*, this midday vulnerability to sleep is not adaptive – it is being suppressed and phased out at the societal level. It is on the wane.

1.2.2 THE SIESTA: A Cultural Phenomenon

One of the most striking examples of the call to sleep at noon, is the existence of the ‘siesta culture’. Millions of people around the world engage in a cultural afternoon nap, affectionately known in Hispanic countries as the siesta^{see footnote}. Such customary forms of nap-taking are to be found world-wide; from the European states of France, Italy, Spain and Greece; extending to the Indian subcontinent, Africa and South America.

* SIESTA: I shall use the term to refer specifically to the taking of an afternoon nap, and not include simple resting. The term ‘siesta’ is a Spanish word with origins in Latin: *sexta (hora)* - sixth hour [from the Concise Oxford Dictionary, 1990]. One can add further meaning to this by turning to The Bible. The first century Jews adopted the count of 12 hours for dividing ‘daytime’ – Jesus: “There are 12 hours of daylight are there not” [John 11:9]. Sunrise was at 6 in the morning and sunset at 6 in the evening. Therefore, the ‘third hour’ is at 9 in the morning and the ‘sixth hour’ is at noon. Thus, siesta = ‘sixth hour’

A New York writer, taking up residence in India was pleasantly surprised to find that the locals' in her neighbourhood, were "fond of long afternoon naps" and even more surprised to find a clause in her tenancy agreement "...prohibiting 'banging noises' between the hours of 1pm. to 4pm."⁷⁶. The siesta hour is clearly sacrosanct and well protected in some cultures. If the universality of this pan-cultural phenomenon needs further credibility, in terms of *numbers* engaged, then the inclusion of China with its constitutionally enshrined 'xiu-xi' ('time to rest' : Article 49 of the constitution), should prove conclusive.

It is somewhat astonishing then, to discover that the literature on cultural napping is so sparse. The bibliophile, Wilse Webb, is a chronicler of publications by the growing sleep research community, and is a researcher with strong interests in cross-cultural napping behaviours. His most recent survey, published in 1991¹⁸, updates his earlier 'publications chronology', which surveyed the period, 1801–1980¹⁹. In a recent paper on the siesta²⁰, this doyen of the 'sleep bibliography' could only find 4 studies on the cultural siesta.

Perhaps as a consequence of this dearth in the scientific literature, Webb & Dinges²⁰ turned to a novel source of information on cultural napping. They assessed napping behaviours across cultures, using the 'Human Relations Area Files' (HRAF). This is a data bank from a cross-cultural survey, conducted under the aegis of the University of Yale since 1937; designed to gather information from samples of people around the world. Some of the information in the survey relates to sleeping and napping behaviours. The information is essentially excerpts taken from the literature of various countries, which are then translated into English and coded. This survey can boast over 200 participating universities.

Table 1. (overleaf) lists citations from siesta countries (with siesta timings). For the purpose of example, I have assembled a selection from the numerous listings to be found in the Webb/Dinges paper. The source of the HRAF are in the main selective anthropological observations, pooled from a diverse literature. Granted that it is highly questionable to extrapolate directly from anthropological sketches to the human condition, I do nevertheless think that the data – *though not scientific* – present a useful insight.

The HRAF data also show the time for napping across cultures to be remarkably consistent — see Table 1.1 on the next page. From the figures we can see that the time frame for the siesta window extends between 1200 h. to 1600 h.

Table 1.0 An abbreviated list of citations referring to siesta occurrence and timing.
Adapted from Webb/Dinges [20]. Data abstracted from HRAF.

Country (subgroup)	Date	Setting	Time
Burma	1943	Agricultural	1200 – 1400
Cambodia	1938	Agricultural	1200 – 1500
India (Bhil)	1954	Agricultural	1200 – 1400
India (Gujarati)	1928	Agricultural	1300 – 1500
Tibet	1940	Agricultural	1400 – 1500
Kenya (Nairobi)	1967	Urban	Afternoon
Nigeria (Nupe)	1922	Village	1300 – 1500
Sierra Leone	1945	Village	1200 – 1500
West Africa (Dogon)	1935	Agricultural	1200 – 1500
Oman	1945	Urban	1200 – 1600
Egypt (Fellahin)	1878	Urban	Midday
Yugoslavia	1939	Agricultural	1300 – 1400
Papua (Orokaiva)	1925	Village	1200 – 1400
Colombia (Kogi)	1949	Village	1200 – 1400
Haiti	1966	Agricultural	1200 – 1400
Jamaica	1954	Village	1200 – 1400
Paraguay (Guana)	1947	Agricultural	1200 – 1400

Table 1.1 Nap time across cultures assessed by % time mentioned in the HRAF [20].

Nap Time	% Citations in HRAF
Noon – 1400 hr.	76%
1500 – 1600 hr.	17%

A more 'scientific' approach to the question of the human siesta, was adopted by Taub in his seminal paper; 'The Sleep-Wakefulness Cycle in Mexican Adults' ²¹: the first major quantitative report. In the town of Hermosillo, Mexico, a cross-section of the residents [N=257], were questioned on their sleeping habits. With respect to the frequency of naps, the results are summarised below; see Table 1.2.

Table 1.2 Napping Frequency: Results from the Taub Mexican Study [21].

Study	Location	N	Age	Napping Frequency
J. Taub [1971]	Hermosillo [Mexico]	257	20 – 89	<p>79 % = napped 4 or more times per week</p> <p>50 % = napped all year * round Jan-Dec.</p> <p>40 % = napped only in the summer</p> <p>10 % = napped during autumn, winter and spring</p>

* For the older age group (50+), 67% napped throughout the year.

A Hollywood image of the siesta, is typified by the archetypal Mexican 'bandito' or peasant, usually draped in white, with a broad-brimmed sombrero, and crouched under a shading tree, in happy sleep. Not surprising then, that Taub's Mexican study on napping frequency, while remarkable in itself, failed to raise much interest. However, the true forte of this telling study lies elsewhere; in its choice of experimental location. Hermosillo has a wide annual variation in temperature, from a low of around 60°F (15°C) to a scorching 90°F (32°C) in summer. The fact that such a large percentage of the sample engaged in siestas during the *entire* year, would seem to nullify the entrenched belief that the siesta is merely a response to the intense heat of summer. Additional factors were at play. Taub drew heavily upon Tunes data^{see 30} on English sleep behaviour, to emphasise the role of sociocultural factors. We now of course acknowledge that napping does occur in cooler countries, with latitudes far from the equator.

Because siesta studies are thin on the ground, we may cautiously draw upon the copious bank of research data on 'daytime sleepiness' as it may be reflective of the tendency to nap at midday. In one of the largest survey samples I have encountered, Billiard et al.²² questioned a massive 58,162 young army recruits on the prevalence of daytime sleepiness. They found a "particularly high" incidence of daytime sleep

episodes (14%) for this age group. In support, they also referenced similar Finnish studies which again found a high rate of daytime sleepiness in a comparable sample. If it was the oppressive heat which forced the young adults of Hermosillo [latitude 29°N] to nap in the afternoons, then to what cause can we attribute similar episodes of daytime sleepiness, found in the cooler climates of Paris [lat. 48.5°N] or Finland [lat. 65°N].

Webb has pointed out that the siesta cultures of the world are to be found principally between latitudes 45°N and latitudes 45°S. His data is weighted by the geographically biased HRAF records: casting his net wider (as other data would compel) might attribute the aetiology of the siesta to a human chronobiologic effect, and one not solely contingent upon equatorial heat. Dinges²³ has recently tried to take the 'heat out' of the argument by suggesting that high environmental temperatures may not be directly responsible for the siesta. The warmer climate of the tropics can in fact disturb and fragment nocturnal sleep; not encourage it. Dinges argues that high ambient temperatures inhibit the capacity to work, especially in agrarian cultures. The tropical heat may thus draw people indoors for shelter - the opportunity afforded by the ensuing inactivity and boredom, may allow the underlying daytime sleepiness to be expressed as naps. Environmental temperature, is going to be a pivotal issue throughout this thesis, and will be returned to and debated at various points.

Clearly, the final act of napping will only be expressed if the circumstances permit. This notion is best illustrated in a comparative study conducted between American and Guatemalan students by Lawrence and Shurley²⁴. They wanted to compare sleep patterns of students from a country where the "siesta was part of the cultural picture with students in a 'non siesta' society". Their sample included 196 American students from Oklahoma, and 196 university students from Guatemala City. Results showed that nearly 80% of reported naps occurred in the afternoon. The location of the naps to the afternoon period was expected. More surprising, however, was the discovery that only 52% of the Guatemalan students took naps in the afternoon, compared to 67% of the American students. Guatemala is a South American country that 'pauses' for a midday siesta — Middle America does not. Further analysis revealed that 79% of the Guatemalans were engaged in part-time employment, as against only 29.6% of the American students. It seems the impecunious students of this Latin American enclave are denied the *opportunity* to indulge in the traditional siesta, compared with their more affluent American counterparts.

Maintaining this theme of daytime activity and napping propensity, a study was also conducted by Okudaira et al.²⁵ on the daily activity levels of a group of elderly subjects in Vilcabamba, Ecuador and the proclivity to nap. Twenty subjects with a mean age of 88 years, had their daily activity levels and sleep patterns monitored. Although there was no custom of taking siestas in Vilcabamba, over half of their sample group took

afternoon naps between 1300 h and 1600 h. The volunteers were classified into three activity groups; high, moderate and low. Their results showed a negative correlation between activity levels and napping behaviour.

The sedate and passive lifestyle of the elderly could explain the high incidence of napping in this group, whilst for the American students, described earlier, the answer probably lies in 'replacement napping' to make up for previous night revelry [see page 14]. For the elderly, the maintenance of a healthy sleep pattern may prove to be very important; napping has been positively linked with a reduction to the risks associated with cardiovascular disease. A recent Lancet paper by Greek researchers, entitled: 'Does a Siesta Protect From Heart Disease?'²⁶ raised the issue. Their work suggest that a short, 30-minute siesta can reduce the incidence of coronary heart disease by almost 30%. A much earlier paper by Soldatos et al.²⁷ also indicated that a siesta may be linked to an improved mental health status as well.

The case of the Guatemalan students is a poignant reminder that culture and dispensable biological drives must remain suppliant to modern day market-forces. The sociologist, Rudolph Rezsoschazy^{see 20} was interested in the way people used and valued their time; in particular, those in the developed nations and the developing countries. He noted that one of the first victims of industrial progress was the siesta. There was a negative correlation between the prevalence of the siesta break and the industrialisation of countries. The interlinked money-markets of the world cannot benefit those who break from the stream of events - *to sleep* - whilst the New York Stock Exchange ripples by the second. In Spain, the siesta is disappearing; in Chile, it is being outlawed by governmental decree; and in China, a resolution by the State Council has placed restrictions on afternoon napping. In his paper 'Early Afternoon Napping: A Fading Greek Habit', Soldatos²⁷ laments the passing of the siesta from Greek culture, as encroaching urbanisation transforms a once rural nation out of old habits.

In northern European countries and the United States, there exist strong sanctions against the siesta. The pervasive ideology which predominates in these countries, was described by Max Weber as, the 'Protestant Ethic'^{see 2}, which seemingly condemns all human excesses in its puritanical drive against the wastage of time. He writes: "*Loss of time through sociability, idle talk, luxury, even more sleep than is necessary for health, six to at most eight hours, is worthy of absolute moral condemnation.*" The Catholic ascetics, St. Francis of Assisi, St. Teresa and the methodists, Fletcher and Wesley, saw sleep as an unnecessary luxury – they tried to eliminate it; but failed³. These religious ascetics date back to the 12th century; and today, sixty years after Weber first published his great tome, our beliefs about sleep have not changed very much. Dement²⁸ recently asked his audience in a lecture, to raise their hands if they

thought that taking a nap projected a negative image of their persona. Almost half the hands in the auditorium shot up in the air.

In summation, the evidence would point to the universality of the afternoon nap. The prevalence of the siesta in cooler climates would mitigate the view that high temperatures alone are the *raison d'être* for napping behaviours. A real chronobiologic tendency to nap must exist – albeit, one that is easily masked in the face of strong social sanctions. The fact that our performance and cognitive abilities dip at midday [see later] would attest to the limitations of our behavioural plasticity. Our 'cultural nature' cannot invalidate our 'animal nature', programmed over aeons of evolutionary history.

The siesta is being speedily abolished through a process of 'cultural evolution'. The anthropologist, Vernon Reynolds, sees "modern urbanized man as caught up in an anthropogenic world that is subjecting him to various new stresses and strains"²⁹ and he questions whether we have the ability to cope with this new 'cultural environment'. Dement²⁸ has recognised that biology and culture often conflict in human affairs. In addressing the question of napping, he remarks: "...it seems that nature definitely intended that adults should nap in the middle of the day.." and calling for a wider public discussion, he appeals, "It is my strong belief that there should be a clear professional/ societal/cultural positioning on napping." It is an issue the sleep research community has yet to address.

1.2.3 NAPPING: A *Global Phenomenon*

This section can be considered an addendum to the above discussion on the siesta. I have described the siesta as a form of 'cultural napping', prescribed by custom and primed by definitive social zeitgebers. But people can (and do), lapse into an afternoon nap for a multitude of reasons, totally unconnected with culture. From the standpoint of this thesis, the crucial reality is this: when such voluntary naps do occur (for whatever reason), the likelihood is that it will manifest itself during the early or middle part of the afternoon^{24, 25, 30}. For the proposition of a Biphasic Theory of Sleep, this is significant.

In non-siesta countries, the recreational time afforded at the weekend will permit afternoon naps, whilst the non-working, unemployed and retired elderly, will nap *ad libitum*, usually out of boredom. But, there is another segment of the population, whose napping behaviour has engrossed the attention of sleep researchers out of all proportion. I refer of course to the superabundant *students* found on campuses, and seen as a temptingly convenient 'data-pool' for the university sleep laboratories. The unstructured, sometimes bohemian lifestyle of students, allows for the free expression of napping behaviours. The studies are plentiful, and the data good, but there is a caveat; we cannot readily extrapolate our findings to the general populous. Students

commonly fall into the category known as ‘replacement nappers’³¹, as they attempt to compensate for lost sleep, due to previous night excesses. Irrespective of motivations occasioning such naps, the point at issue is the temporal placement of that all too likely nap. The majority of studies would place the napping time of students from mid to late afternoon, whilst for the general populous, it would probably be early afternoon²³. As if to emphasise the chronobiologic nature of this second sleep tendency, researchers have discovered that the timing of naps are largely influenced by the temporal placement of the major nocturnal sleep. For instance, if we note the time of the mid-nocturnal sleep, and calibrate this circadian phase as 0 degrees, then we have a metaphoric point of reference. Dinges²³ found that the midpoint of the afternoon nap was likely to be 180 degrees out of phase with the midpoint of the nocturnal sleep (24 hr=360°). So, if an individual goes to bed at 12 midnight and rises at 0700 h, then the midpoint of this nocturnal sleep will be at 0330 h. From this we can predict that the midpoint of the afternoon nap will most likely occur at 1530 h. There are indications that such a well-programmed biological clock, as seen here, has lost its synchronicity in the case of narcoleptic patients. Mullington and her Canadian team³² have recently reported that the midnap point of narcoleptics, occur at phase angles which are typically earlier (150°–170°) than the 180° normally found in healthy subjects. Such is the robustness of this midnap timing phenomenon, that it is proving a useful benchmark in the diagnosis of sleep disorders. Godbout and Montplaisir³³, found that in tests of performance, narcoleptic patients showed greatest improvement following an afternoon nap taken at 1400 hr. and 1600 hr. (compared to naps at other times), so alluding to some beneficent property of sleep at this time. In a recent experiment involving an ultrashort sleep-wake paradigm that has become his trademark, Peretz Lavie³⁴ has also found afternoon naps (coincident with his ‘secondary sleep gate’) to be more efficient and restorative, with longer SWS duration and lower post-nap sleepiness, compared with later evening naps.

There is a growing modern-day philosophy that we ought to return to a more ‘natural’ way of life; for instance, by changing our diet to include ‘organic whole foods’ rather than artificially refined foodstuffs. The underlying presumption of this ethos, is that ‘things that are natural – products of nature – are good for us.’ By the same corollary, if afternoon naps are in the ‘blue-print’ of Man, an evolutionary legacy laid down by nature, then they ought to be good for us. Does the scientific evidence bear this out? Are naps good for us? On the whole; yes. Research findings such as that of Lavie’s noted above, and many others, are consistent in highlighting the improvement in both mood and performance following naps, although controversy surrounds the merits of simple bed-rest versus *actual sleep* during naps. These studies are documented and reviewed elsewhere^{23, 35, 36, 37}. A small percentage of nappers do report momentary impairment immediately upon rising from sleep - a condition known as ‘sleep inertia’ or

sometimes, 'sleep drunkenness'. It's a transient effect, lasting between 5 to 15 minutes, though soon giving way to improved mood and performance, relative to prenap values.

If we have developed an inflexible monophasic sleep-wake cycle, then afternoon napping – "*that deviant and unwanted form of sleep*"³⁸ – may be expected to thwart the well balanced nocturnal sleep budget. In fact, for subjects with a healthy sleep hygiene, napping has not proved to be deleterious to either the quality, or the onset of nocturnal sleep. Recently, Soldatos et al.³⁹ presented new data on the effects of afternoon napping on nocturnal sleep. In a comparison between matched groups of 10 habitual nappers (NPRS) and 10 non-nappers (N-NPRS), they found very little difference in the amount of nocturnal total sleep time (TST): NPRS = 389.5 ± 11.9 vs. N-NPRS = 391.0 ± 20.4 . Karacan⁴⁰ discovered that the infrastructure of nocturnal sleep may change in response to a homeostatic redistribution of SWS, accumulated during the afternoon. But, there were no indications of any deleterious effects resulting from napping per se' — infact, the afternoon sleep latency of Soldatos's subjects, was half that of the nocturnal sleep latency, indicating the ease with which afternoon naps are engaged in.

In a survey of 430 students, Evans et al.³¹ found 60% of their sample reporting some form of napping. They proceeded in a Linnaen fashion to classify the nappers into two distinct families:

- i) Replacement Nappers: napping to recover lost nocturnal sleep
- and, ii) Appetitive Nappers: napping for some obscure psychological benefit, unrelated to the physiology of sleep.

This binomial nomenclature of nappers has gained acceptance in the parlance of sleep research. However, I am troubled by the simplistic subdivision of napping; the accredited terminology of nappers when examined closely would appear to countermand the notion of an endogenous biphasic sleep pattern. Replacement nappers are simply fulfilling a homeostatic sleep requirement; a daily quota of sleep has to be met and no reference to circadian factors need be made. There are problems with this line of reasoning. While accepting that a sleep debt is paid off, Webb⁴¹ accurately challenges the assumption that the sleep debt is the *agent* for the nap. As part of the endogenous biological rhythm, a *natural* propensity to sleep during the 'nap time' emerges *anyway*. Other questions need addressing too: why is the replacement nap 'time bound' to the mid afternoon, a debt which could arguably be settled at any other time of the nycthemeron; and why is there 'an apparent recovery without sleep' that is evident much later in the afternoon. Finally, what is the nature of that 'debt' which could be so quickly paid off in the brief 'forty winks' that is the afternoon nap (cf. the 7-8 hours of concerted 'sleep effort' at night). There are also valid objections to the loose and nebulous distinction characterising the so called Appetitive nappers. To claim

that the ‘appetitive nappers’ are regularly satiating an ‘*undefined*’ psychological need, other than sleep, goes against the evidence of chronobiology and the tenets of human nature. We are not simple ‘creatures of habit’; our every action is entrusted with meaning, the motivations of which are not always apparent, but imperceptibly present. In an excellent chapter entitled; ‘Adaptive Daily Strategies in Behaviour’⁴², Serge Daan observes:

“Daily routines in the behaviour of individual animals can be viewed as strategies to cope with the time structure of the environment. Such routines, like other elements of behaviour are rough products from the mold of natural selection polished by individual experience.”

I am not suggesting that our call to a collective nap is a characteristic fixed action pattern indicative of other animals; our cerebral advancement ensures a behavioural repertoire that is not tied to the vagaries of the environment. But, we are products of a slow evolution; 99% of our existence has been as hunter-gatherers,²⁹ and we do carry vestiges of an earlier time. Our tendency to nap at midday is just such an inheritance, whose fulfilment may belie the vulnerable group classed as appetitive nappers by Evans et al.. We may ignore or overrule this chronobiologic urge to nap at midday, but for the many who do succumb, the pressures to sleep are simply too great.

In a bid to highlight the ‘naturalness’ of naps, and to accord it the full status of a true biological rhythm, Webb⁴¹ has enumerated a set of qualifying criteria, which he claims, human naps do satisfy. These criteria are summarised below:

Criteria For Biological Rhythms

Conditions Met By Naps

- | | |
|--------------------------|----------------------------------------------------|
| 1. Temporally repetitive | Its occurrence halfway during the day at 1-4pm. |
| 2. Species specific | Each species has a distinctive intermittent sleep. |
| 3. Developmental | A developmental pattern of naps is evident. |
| 4. Innate and unlearned | It's world-wide, pancultural spread exemplifies. |
| 5. Endogenous | Naps occur in isolation chambers free of cues. |
| 6. Adaptive | A control system to avoid heat & save energy. |

In the daily pageant of the sleep-wake cycle, the taking of naps may be seen as a response to the chronobiological demands. But naps may also fulfil a vital homeostatic function; as in the case of the perennial sleep-deprived student, naps may even redress the sleep deficit. If we do have a genetically determined ‘sleep stat’ as advanced by Meddis¹⁶, with a programmed species-specific sleep quotient, then it seems that the afternoon nap may play an integral part in the management of such a sleep economy.

1.2.4. THE ONTOGENY OF SLEEP PATTERNS

In trying to understand this midday sleep need, we need to adopt a multidisciplinary approach, drawing from all aspects of biology and even social anthropology. The evolutionary approach discussed earlier has made an important contribution to the understanding of mammalian sleep, including human sleep. An equally successful research strategy, commanding much interest, is the 'developmental' course of sleep entrainment. Clues to the origin of the biphasic rhythm may lie somewhere in the ontogenetic data. The utility of this approach has been emphasised by Lea⁹: "For any behaviour of any complexity, we also need....a 'developmental' explanation, which will centre on the way the behaviour emerges and is perfected within the lifespan of the individual."

Investigators have identified a physiological state in the unborn foetus approximating to sleep – as we know it. By week 30, distinct periods of activity and quiescence are apparent, occurring at regular intervals, making the notion of a 'foetal day' appear quite tenable, even at this stage⁴³. Sleep patterns resembling REM sleep, are to be found in the womb; its supporting brainstem structures are early to develop. The cerebral structures important for non-REM sleep will mature after birth. The relation between the changing brain structures and sleep, has prompted J. Allan Hobson⁴³ to opine: "...sleep is of the brain and by the brain", a view that has been extended recently into a bolder concept, declaring sleep to be very much *for the brain* as well (see Horne¹⁷). The drama of the developing brain, before and after birth, is exemplified by the proliferation of cells in the cortex, together with the growing interconnections of the serotonergic and noradrenergic systems. All this neural activity will ultimately be reflected in the infants changing behaviour.

Outwardly, it might seem that the only behavioural repertoire the infant is blessed with, is the capacity for prodigious sleep. A newborn will sleep for upwards of 16 hours, waking intermittently to feed. The sleep will be evenly dispersed throughout the 24 hours, with large blocks occurring during the daytime. This is a good example of the polyphasic pattern of sleep, the *sine qua non* of many lower order animals. Through a process of entrainment, a rhythm will be established (imposed!) by the mother, and at 3 months or so, the infant will confine the larger portions of its sleep to the nighttime. As the infant develops, the levels of daytime sleep will gradually fall.

Kleitman and Engleman^{see 44} closely observed a group of infants, aged around 6 months, through the entire span of daytime (8.00 am.–7.00 pm.). They discovered two distinct periods of sleep tendency, occurring between 10.00 am. and noon and again between 2.00 pm. to 4.00 pm. A clear binary pattern of daytime sleep was evident. Between the

ages of 2 to 4 years, most infants will continue to nap, although the binary pattern of napping will soon be replaced by a single midday nap. Webb has cited data which indicate that between 50% to 70% of 2-year old children succumb to daily bouts of napping – at least 3 to 4 times per week. Similar reports by Hellbrugge of young (hospitalised) children indicate a marked propensity to sleep between 1pm. to 3pm^{see 13}. It has been suggested by Hobson that the stubborn adherence to the afternoon nap, may serve to offset the “disabling trough of irritability and dysfunction” that is likely to occur late in the afternoon, if naps are missed. As the child enters primary school, the afternoon period set aside for the nap is finally lost.

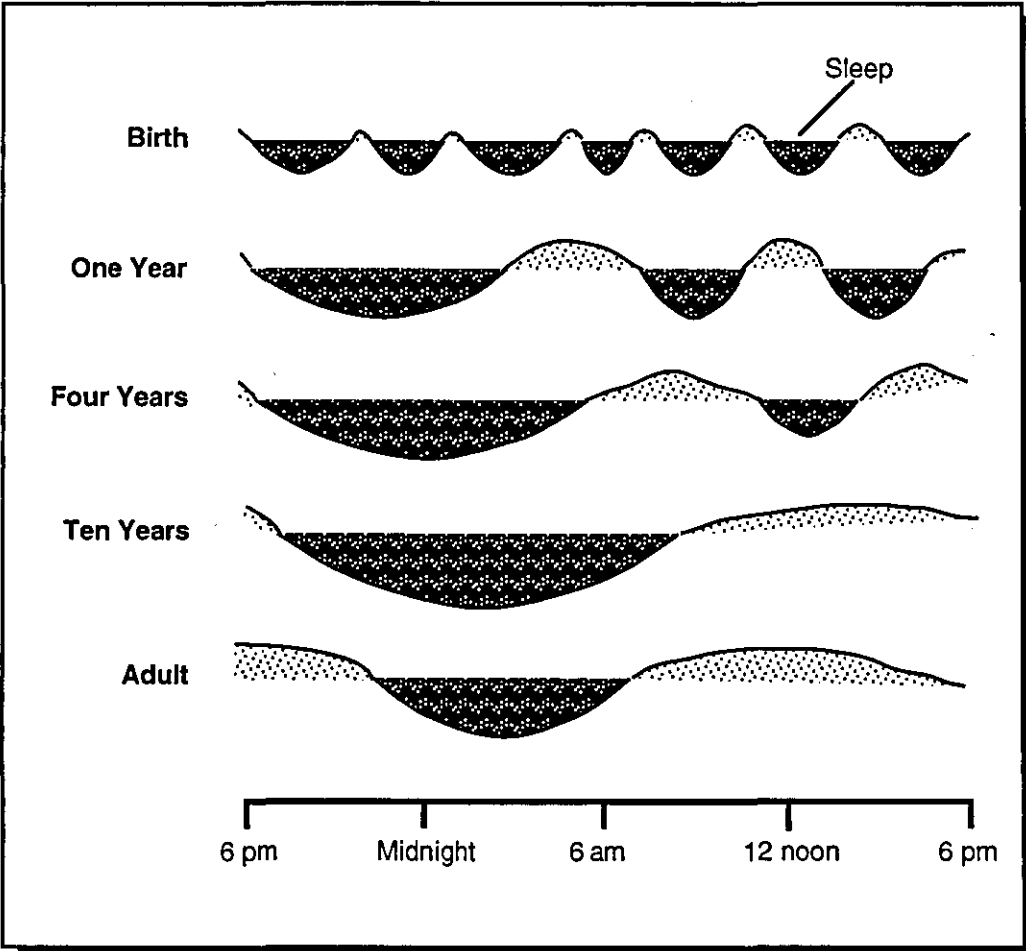


Figure 1.0 The ontogenetic sleep profile highlighting the change from polyphasic sleep to biphasic and monophasic sleep. Adapted from Borbely [77].

This ontogenic tale of the emergent sleep patterning raises a few pertinent questions in the context of the larger debate on biphasic sleep. In the multiplicity of naps, scattered throughout the nycthemeron of early childhood, why, it must be asked, is the afternoon nap always the last to be surrendered⁷. Can we confer any special biological importance to a nap taken at this particular stage of the day? If we accept the hypothesis that there exist a strong primordial urge for a second sleep tendency at midday, then this additional ontogenic idiosyncrasy provides grounds, supportive of such a proposition. If this midday nap has its roots in infancy, then it appears to be carried over into adulthood in an almost neotenuous fashion. Horne¹⁷ muses upon the uncanny resemblances of the human adult to the human infant, in examples of our 'behaviours' and physiognomy, and speculates that : "...the functions of human sleep, particularly in relation to the cerebrum, may not change substantially from infancy to adulthood." Likewise, it would also seem, the patterning and chronobiologic directives for sleep, do not alter much in the course of the evolving life span. Given the opportunity, childhood naps will reappear in adolescence and early adulthood as Carskadon's surveys and MSLT data would attest [see later].

With advancing years, nocturnal sleep generally becomes shorter and fragmentary, and the waking day is often characterised by a slower and sedate pace, in the wake of retirement. These particular circumstances appear to favour the re-emergence of the afternoon nap. This situation, reminiscent of the childhood napping described earlier, is not a regressive step backwards, but rather, a natural expression of the true biological drive to secondary sleep, that is given a freer reign as the limiting controls and constraints of acculturation diminishes during the retirement years. Perhaps it is inaccurate to talk of a 'consolidated sleep' breaking down in old age (see Tune³⁰), as if this monophasic pattern represents the de facto state of affairs. Clearly, the afternoon nap, so much a part of childhood, is returned to in later years of life, and given half the chance is wilfully indulged in, at any point during the intervening years.

1.2.5 THE ENDOGENOUS NATURE OF THE MIDDAY NAP.

In the unfolding discussion on this midday sleep tendency, the overall view presented so far, has been of a rhythm that is independent – and not passively tied to the changing environment. The conclusion affirmed has been of a secondary sleep cycle generated from 'within'; driven by an endogenous oscillator. Supporting evidence for this view has not been discussed. We really need to observe this endogenous rhythm directly. The classical way of uncovering such internal oscillators has been to place volunteers in situations devoid of external cues and influences. Fortunately, these conditions prevail in natural caves, or can be artificially instituted in specially constructed isolation units.

The study of napping in 'time-free environments' has been the forte of Scott Campbell and Jurgen Zulley — their efforts, in recent years, have largely established the endogeneity of this secondary sleep cycle⁴⁵⁻⁴⁹.

Since the discovery of endogenous rhythms by De Mairan – over two centuries ago – chronobiologists have been inspired to descend into the depths of caves, or to construct elaborate underground bunkers in order catalogue an array of endogenously controlled rhythms. Whilst they succeeded in the removal of nychthemeral cues, we now realise the many failings in their methodology. Typically, subjects were required to live a 'normal life', with three sequential meals, and the circumscribed day to be concluded with one nightly sleep period. Daytime naps were strictly forbidden. Unfortunately, in prejudging the monophasic pattern to be *de rigueur*, the researchers missed out on discovering the full complexity of human sleep patterns.

Campbell and Zulley were to appreciate, that for the true physiological sleep tendency to surface, volunteer subjects had to be free from cultural demands and experimental restrictions. In their experimental paradigm, no 'daily structure' was imposed, napping was allowed and barriers to sleep were removed⁴⁹. By minimising behavioural options (reading, writing, music, etc.) they provided conditions which – they believed – would allow for the true expression of sleep propensity. In their view, sleep must be offered a "course of least resistance", drowsiness if present, should be given free reign, yielding to naps or sleep and not be actively fought.

Their results were unequivocal in suggesting that the placement of naps across the nychthemeron was not a random process. Subjects under [72-hrs.] isolation, tended to spread their sleep episodes across the 24-hr. day. However, on closer examination, they discovered a clear bimodal distribution of sleep onset times: major sleep episodes occurred around 2400h, with shorter naps clustering around 1400h. They also found that naps were most likely to occur around a "phase position corresponding to the time of maximum values in body core temperature," (see figure 2, overleaf). In general, the maximum body temperature occurred in the middle of the waking period; so if one awoke at 0700h and retired at 2300h, then the point of maximum temperature would occur around 1500h. In contradistinction, the major sleep episode occurs at the temperature nadir.

I must add that the temperature maximum is part of a broad peak (from midafternoon to 8 pm in the evening). An analysis of the *duration* of sleep, revealed a further alliance between temperature and sleep. Daytime sleep episodes would not exceed 3 hrs. – a tentative suggestion of a circadian control mechanism imposing an upper limit. However, if sleep was initiated after 20.00h. (a point at which body temperature begins to fall), then the restrictions on sleep duration was summarily lifted.

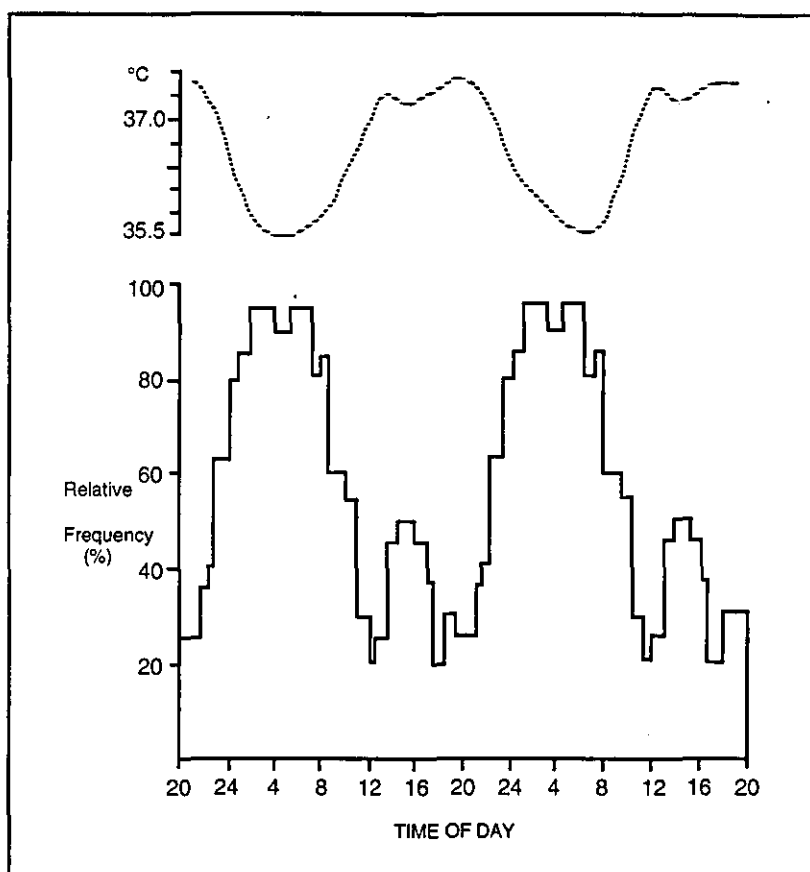


Figure 2. Distribution of sleep tendency and core temperature (average) against time of day. Adapted from Campbell and Zulley [46]

The experimental methodology employed by Campbell and Zulley has been termed “disentrainment” by the authors. There is however, an extreme form of disentrainment in which subjects are actually ‘confined to bed’ in experiments lasting up to 60hrs. Maintaining recumbency in an isolation chamber for such a length of time, with no viable alternatives to engage the human mind, results in the taking of frequent naps. Zulley and Wever,^{see 48} employed this protocol and discovered the re-surgence of a polyphasic sleep-wake cycle of 4–6 hours. A similar four hour sleep rhythm was found by Nakagawa⁵⁰ in subjects confined to bed for 12 hours a day. Interestingly, neonates display a similar 4–6 hour cycle, prior to social entrainment.

In another investigation, Campbell and Zulley changed their experimental strategem by instructing subjects to pursue normal activities and to *avoid* taking naps⁴⁵. Remarkably, over 50% of their subjects found it impossible to suppress the urge to nap, bestowing a strange omnipotence upon the afternoon nap. The fact that we all do not succumb to a universal nap in daily life, is more a reflection of our social and occupational demands ‘masking’ our innate rhythms. Sleep, is after all highly plastic, as shift workers and sleep deprived students would corroborate.

The sociocultural environment exert a (exogenous) control that is just as influential as the endogenous component. Campbell and Zulley perhaps underplayed the environmental factors – their isolation chamber, providing no viable alternatives to sleep, was an abnormal setting that may have told us more about boredom than circadian sleep. Such healthy scepticism should not colour the fact that their isolation studies have provided us with valuable data on the endogenous nature of naps. The value of their work can best be enunciated in a re-adaptation of one of their analogies: If a hydrologist or geographer is interested in the true course of the river Thames, he should realise that after the imposition of the Thames Barrier, numerous dams and reservoirs, yachting marinas and countless bridges (in an effort to structure the river for our benefit) what he will find will probably not reflect the accurate, natural dynamics of the river. Likewise, experimental sleep studies geared around modern industrialised man, living a 9-5 existence, bears no relation to the true dynamics of the sleep-wake organisation, modified by countless behavioural controls in the service of society.

1.2.6 THE BIPHASIC RESURGENCE OF SWS

A. THE DATA

The free-running experiments of Campbell and Zulley were conducted in the wider context looking at the endogenous nature of the human sleep-wake organisation. It was fortuitous that their results proved so germane to the issue of the biphasic rhythm; a fact recognised by the authors and subsequent investigators. By contrast, Gagnon and De Koninck, approached the problem of the 12-hour sleep rhythm directly – formulating their experimental goals in order to specifically test Broughton's circasemidian theory.

To recall; a major precept of the Broughton postulate^{see 7} was the evidential increase in stage 4 sleep during the afternoon. This increase was earlier described (and graphically illustrated) by Webb,⁵¹ and in a brief abstract some years later⁵² he was to note that two peaks of SWS were identifiable in subjects able to extend their sleep: one occurring shortly after nocturnal sleep onset (as expected) and a second (unexpectedly) 12 hours later. Bendrich Roth, in the early 1970's was also mindful of this phenomenon^{see 17}. This growing corpus of data was seized upon, and incorporated by Broughton, into his circasemidian model. The secondary resurgence of SWS was interpreted as a real "afternoon pressure for deepening NREM sleep⁵." Moreover, when viewed as a 12-hour ultradian rhythm, the post-lunch dip, he suggested, could be "a subharmonic of the normal circadian distribution of delta sleep."

Gagnon and De Koninck, fellow academics of Broughton at the University of Ottawa decided to put Broughton's circasemidian theory to the test⁵³. In their first study they found 6 volunteers (out of an initial 8), who were able to extend their sleep to 15 hours.

After 2 nights of 'normal 7–8 hours laboratory sleep', subjects were asked to extend their sleep on the third night. Polysomnographic data was collected throughout. For sampling purposes, the EEG data was divided into blocks of 3 hours: from 00–03, 03–06, 06–09, 09–12 and 12–15 hrs. The levels of stage 3 and 4 systematically dropped from the 00–03h peak values to a nadir at 09–12h. However, in the 12–15h time block there was a sudden increase in stages 3 and 4 (anova: $p > 0.01$ and $p > 0.05$ respectively). In essence, the SWS which was abundant at the time of sleep onset, was to fall sharply, only to reappear in the afternoon between 1200h and 1500h.

These results provoked further questions as to the nature of SWS distribution in sleep.

- i). Is the early afternoon period between 1300–1500 hrs., a natural phase position for the procurement of a second SWS quotient ?
- or ii). Are the two SWS peaks phase-locked into a bipolar (180°) conformation i.e. must 12 hrs. elapse before the reappearance of the second SWS block ?

These points were addressed in a second study by Gagnon and De Koninck, with the original protagonist, Broughton⁵⁴. Their experimental design was more complex, but essentially, it involved the phase shifting of the extended sleep period. Sleep was delayed by 4 hours – subjects slept at 0400h, in a design that included an adaptation night and baseline recording. The first SWS peak occurred in the first 3 hours after sleep onset. A second SWS peak was noticeable around 1300h and a third peak emerged 12.5 hours after the 0400h sleep onset. By the third day of the experiment, the middle SWS crest seen at midday finally disappeared, leaving just a secondary peak, separated by a 12.5 hour interval from the first. The fading midday peak was inconsequential, representing a “residual expression of the second peak prior to shifting”⁵, in much the same way, rhythms are realigned after jet-lag; slowly substituting new patterns for old. A 12-hour biorhythm of SWS was thought likely in the light of these results [NB. the actual interpeak interval was 12h. 20min in acknowledgement of our innate 25 hour (free-running) day]. These findings were taken as strong support for Broughton's original proposition of a circasemidian rhythm.

Working independently, Webb⁵⁵ produced further evidence to corroborate these results. Webb's extraordinary sample [N=56] was made up of 14 pairs of identical twins and 14 pairs of fraternal twins. The majority slept for between 8 to 9 hours and only 4 subjects were able to maintain sleep in excess of 11 hours. Analysis of this sample (albeit small) of extended sleepers, revealed a similar SWS peak occurring in the 12th hour. Taken together, the results of Gagnon et al.^{53, 54} and Webb⁵⁵ concur, but the conclusions drawn by the two camps, differ somewhat.

The Canadian workers set out to test Broughton’s postulate, and this is reflected in their inferences, which remained confined to the 12-hour circasemidian model. Webb, by contrast, sought an explanation from the much shorter (and primitive) ultradian rhythm concept. He frames his explanation in terms of the paradigm presented by Lubin and colleagues^{see 55}, which looks upon SWS as a “dampened ultradian rhythm, within sleep” — the fact that SWS is not manifested during the latter portion of nocturnal sleep, is attributed to its ‘dampening’. Though recognising that “the REM episode always coincided with a delta trough”, they were to reject the notion that REM sleep itself, was the dampening factor responsible for the suppression of SWS. Webb’s close analysis of his data did, however, point to a reciprocal relation between REM sleep and SWS.

The studies described so far, were published in the short span between, 1984 to 1986. In a paper published in 1989, Campbell and Zulley⁴⁶ confirmed the existence of a SWS biorhythm. However, they reserved two points of criticism towards these early studies. Firstly, they bemoaned the fact that only ‘sleep’ was used as a parameter and no other dependent measures taken. Secondly, they emphasised that on average, 40 minutes of wakefulness interjected towards the end of the 15-hour extended sleep; a point not missed by Horne either [see later]. Their first point of dissent is a cogent one, the latter remains a subject of an ongoing controversy. The extended sleep paradigm offers a foundation upon which we can possibly build a coherent biphasic theory of sleep, so these criticisms must be addressed. I will therefore attempt to clarify the various points of contention in the rest of this section.

The architecture of sleep is greatly affected by temperature: we know this from numerous reports,⁶⁴⁻⁷¹ and the affiliation between temperature and sleep is now widely accepted. It was thus befitting to include temperature as a dependent variable, which is what Campbell and Zulley did; and it proved to be the hallmark of their contribution. Their methodology, described earlier, was to isolate subjects [N=9], in a time-free environment, without any sleep or feeding restrictions. They found that naps occurring between 1400 and 1700 hrs. contained a greater proportion of SWS than naps occurring at any other time; see Table 1.3 below.

TABLE 1.3 Temporal Placement of Naps : Data from Campbell and Zulley [46]

	Naps between 1400–1700h [N=12]	Naps at other times [N=14]
Average SWS	19.4%	10.8%
Median SWS	14.8%	5.7%

The high variability in the SWS proportions prohibits statistical significance, but a trend towards a midday clustering of naps is seen. In a disentrained environment, sidereal time is rendered meaningless. In order to gauge the precise circadian phase position of the numerous nap episodes, they referenced each nap onset by its interval to the absolute maximum of body core temperature; i.e. the proximity of each nap onset to the maximum body temperature. Figure 3 shows that the closer the naps are placed to the temperature maximum, the greater the percentage of resultant SWS.

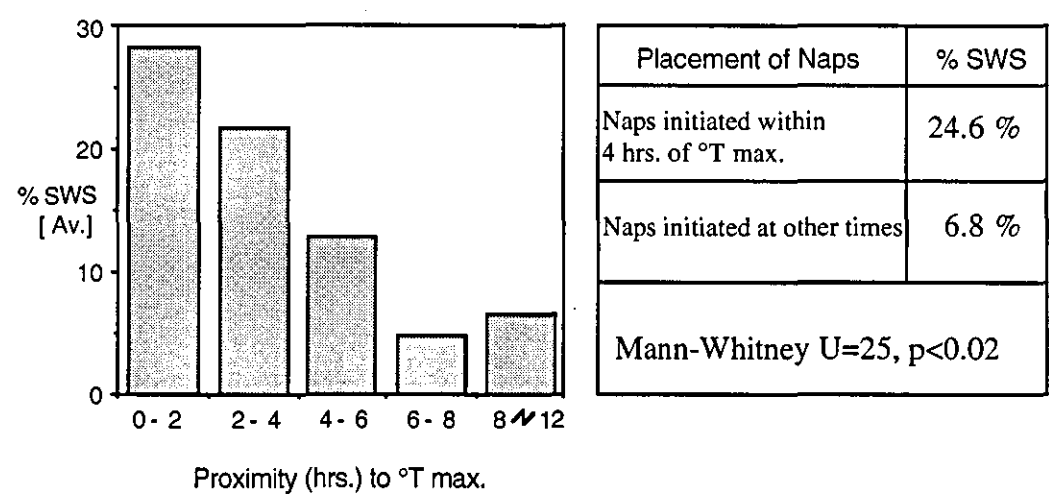


FIGURE 3. Placement of Naps : Relative to Core Temp. Maximum [Ref.46]

Naps occurring within 4 hours of the temperature maxima had over 3 times the amount of SWS per hour, compared to naps found outside this range (14.7 min/hr vs. 4.1 min/hour respectively). The time of maximum body temperature (and SWS surges) corresponded “to a time approximately 12 hours after the midpoint of the preceeding major nights sleep.” This 12 hour periodicity agrees with the earlier Canadian reports and with of Webb’s own findings. However, whereas Webb was to conclude that : “the reappearance of SWS was *sleep generated* rather than determined by a circadian time period” — Campbell and Zulley on the other hand, maintained that their results showed: “a strong relationship between the propensity for SWS and circadian factors *besides sleep itself* ”[emphasis mine]. Berger et al.⁵⁶, have produced data which has also led them to conclude that body temperature at noon, may : “also account for the reappearance of SWS after 12 h of extended sleep in humans, at the time of day when the circadian rhythm of Tb [body temp.] during wakefulness is on its upswing.”

We must view Campbell and Zulley’s findings of a relationship between midday SWS and temperature, as an important element, that has to be incorporated into the circadian model developed by Gagnon et al.. Weitzman et al.⁵⁷ conducting similar studies of napping under disentrainment have concluded that sleep stages, like other rhythmical processes, were: “...constrained by underlying periodic physiological mechanisms.”

In the light of the emergent zeitgeist, De Koninck and co-workers, have now included rectal temperature readings into their well-established research paradigm. Their recent findings indicate that subjects who showed a SWS resurgence under sleep extension, were also most likely to display a marked drop in core temperature during the first half of sleep; suggestive of a link between the circadian phasing of temperature and SWS changes⁵⁸. With the inclusion of these physiological measures into the extended sleep paradigm, Campbell and Zulley's first point of criticism is being addressed. I will now move on to the vexed question of intervening wakefulness found during the latter portions of extended sleep, and discuss the likelihood that it may be responsible for the second SWS appearance. This point has been the source of much controversy.

One of the first, and most damaging critiques against the conclusions drawn from extended sleep studies was articulated by Horne^{59, 17}. He argues against the current crop of interpretations tendered so far, and proffers an alternative explanation of the data. In the first instance, he regards the SWS resurgence as a compensatory action, assuaging the period of wakefulness that is observable in the latter fraction of oversleep. From the data of Gagnon et al., Horne has mathematically derived a quotient of 6.5 minutes of SWS per hour of wakefulness. However, by this calculation a two-third shortfall in the *Wakefulness:SWS* ratio results. The extra SWS is accounted for in his second line of reasoning. Additional REM sleep, which ensues from oversleep, Horne believes, serves to encourage SWS generation. Since REM sleep closely resembles wakefulness, it is argued, the attendant consequences of wakefulness (increasing SWS) is likewise mimicked. Finally, he takes to task the second experiment by Gagnon et al., in which extended sleep onset was delayed by 4 hours. The emergence of a secondary SWS peak in this case, was dismissed as a manifestation, or artifact of the experimental protocol, in which 4 hours of extra 'prior wakefulness' was obtained from the delay of sleep onset.

These reservations outlined by Horne in 1988, prompted Broughton, Gagnon, and De Koninck to re-analyse all their original data in a retrospective study⁶⁰. The authors specifically addressed the contentious point of the 4 hours prior wakefulness engendered by the phase delay of sleep onset to 0400h. Whilst accepting that a 35-minute increase found in SWS could be due to the extra burden of wakefulness, they do not see it as causally related to the second SWS peak. They argue instead, that the brief appearance of the fading SWS peak seen briefly during entrainment, and found between the two major SWS peaks (with a 12-13h separation), would indicate that SWS is under the governance of a 'circadian time-of-day factor', linked to a 'sleep dependent' 12-hr. rhythm. The transitory appearance of the third peak between the two major SWS peaks – explicable in terms of the time-zone travel analogy I invoked earlier – cannot be accounted for by the 'prior-wakefulness' interpretation. It is an explanation also refuted by Campbell and Zulley. The 26 multiple naps they examined were

preceded by 3.7 hours of wakefulness (on average). Despite experiencing the same amount of prior-wakefulness, afternoon naps were disproportionately abundant in SWS, compared to naps at other times. Extreme sleep deprivation studies have furnished us with ample evidence of the association between prior-wakefulness and SWS, but in the minutiae of everyday living, circadian factors cannot be denied an important modulating role in this respect.

B. THE FUNCTIONAL IMPLICATIONS

The issues raised in the foregoing discussion concerning the re-appearance of SWS will inevitably contribute to addressing questions of a teleologic nature. But, what is the 'ultimate' purpose of this resurgent SWS? I reiterate, not of SWS per se, but of SWS temporally placed at midday.

In terms of the current models of 'sleep function', there is a tangible utility to a 'prior wakefulness' explication of SWS resurgence, especially for proponents of 'restorative theories'. Simply put, they would argue that wakefulness results in a particular 'internal state' which is satisfactorily redeemed or modified by sleep. Their rosetta stone, of course, is the exaggerated proportions of SWS found in the recovery sleep of sleep deprived subjects. It is hard to believe that the very short spell of the midday nap can even begin to make an indent upon any putative recovery process. Kenneth Hume has remarked: "Common sense would suggest the system (restorative processes) has evolved to withstand a certain duration of depletion and take subsequently a relatively fixed duration for recovery."⁶¹ The large benefits accrued from a short nap are not proportionate to the fractional time spent asleep (cf. nocturnal sleep), nor can the relatively brief waking interval explain its necessity. If naps are important for tissue repair (cerebral or bodily), biology would not allow it to be discretionary or whimsical as is presently evident by our ability to offset napping.

The alluring theoretical base we are left with, comes under the eegis of the 'behavioural theories'. These ideas are ethologically based, and noteworthy in that they do take into account circadian factors, which are responsive to environmental periodicities. Sleep is seen as a behavioural adaptation to the dangers and vagaries of the environment. This is succinctly abridged by Moorcroft⁶² as: "*It's safer to be asleep.*" As I have recounted earlier, Webb and Meddis are distinguished spokespersons (if not architects) ascribing to such a view. If sleep offers safety, then where is the danger around the time of noon? The danger could be in the abiotic environment. Specifically, the torrid heat of midday. It is a time when the wise retreat to a shading shelter – the chimpanzees of Gombe withdraw to their shaded day nests; the farmers desert their fields, and only 'mad dogs and Englishmen venture into the noon day sun'. So, what is it about this scenario which invites the resurgence of SWS in naps taken at this unpalatable hour?

The escape from the midday heat is the escape from physiological stress. The brain has a very narrow working temperature threshold, at 37.5°C it is lower than most mammals (38.5°C). There is a manifest danger of overheating from environmental exposure, with its excessive cranial insulation, coupled with the circadian rise in body temperature and further amplified by high visceral and cerebral metabolic rates that are so characteristic of wakefulness. A withdrawal into (adaptive) sleep at this time not only ensures inactivity, but also conserves energy; and importantly, may offer “a cooling period for the brain”⁶¹.

If sleep must occur in the heat of midday, then part of its edict may be to check temperature: if so, SWS would clearly be propitious at this time, according one school of thought. It is argued that during SWS the brain can institute a “controlled reduction” in body temperature compared to the “uncontrolled” temperature regulation seen in REM sleep. The thermostat is tuned to a ‘lower setting’ during SWS, whilst in REM sleep it is totally “disconnected”⁶¹. With respect to thermoregulation, Benoit⁶³ believes that “...REM sleep represents a special time of risk, particularly if external conditions are far from normal (very cold or hot environments).” General support for the thermoregulatory role of SWS has been assembled from studies correlating pre-sleep body heating, with increased SWS generation^{17, 64-70}, though the most developed appraisal of this hypothesis has been presented by McGinty and Szymusiak¹⁷². A recent report entitled; ‘Human Sleep in Dry Tropical Africa’⁷¹ further implicates SWS involvement under conditions of a more natural thermal load from the climate.

The aforementioned teleological account which aims to explain the *raison d’être* of afternoon sleepiness, possesses the kind of appeal that characterises all such ‘complete theories’ of sleep. Nevertheless, the evidence for the thermoregulatory role of SWS is still subject to controversy. A more mundane and parsimonious explanation that perhaps lacks the same ‘armchair’ charisma of the above theory would simply state that afternoon sleepiness is a consequence of boredom combined with opportunity, that manifests during the afternoon withdrawal indoors (from the noon day sun). Simply put, the bicircadian urge to nap is given free expression when the environment permits.

1.2.7 THE POST-LUNCH DIP

The discussion so far has presented the second sleep tendency as a momentary interlude that interjects at a point, half-way through our waking day. It has been amusing to note how some cultures stop all activity around midday, to enjoy a brief nap, as do our own dissident students and elderly folk: some nap for recovery and some for recreation. In the event, the picture presented, may have unwittingly portrayed this afternoon sleepiness as a mere ephemeral state, of little consequence, and one that is easily offset by a cup of coffee, or leisurely stroll to the office mailroom. One feels ‘lousy’ at a

particular point in the afternoon, but by tea-time normal 'biological service' is resumed. There is however, a more serious side to this midday 'lull' which descends upon our daily stream of consciousness. During this period, our ability to perform certain tasks becomes impaired; cognitive and motor tasks. Examples to be presented later, will show a secondary afternoon increase in car-accidents, industrial accidents and near fatal lapses by radar operators, ICU nurses and other occupations demanding maximal vigilance. These are all examples of failures in *human performance*; in other words, impairments to tasks requiring "the use of cerebral processes in responding to specified sensory information by appropriate motor actions"⁷². These field observations have been replicated in the laboratory, where human performance measures have been discovered to display circadian rhythmicity. This early afternoon drop in human efficiency has been considered as further proof of our biphasic sleep tendencies, where the drop in performance is adjudged to be a manifestation of the increased sleepiness that is present in the afternoon.

Our understanding of these performance decrements stems largely from the pioneering work done over 100 years ago. Much of the research was carried out by educational psychologists', interested in the practical issue of 'mental fatigue' over the course of the school-day, and the appropriate timetabling of academic subjects. Diurnal variations in mental efficiency was reported by Ebbinghaus (1885), in studies involving the rote learning of nonsense syllables at various times of the day. Other reports by Lombard (1887), measured the changing intensity of the knee-jerk response over the day, whilst Dressler (1892) tested morse-key tapping (6 times per day, for 6 weeks). Dressler's data gave a curve showing an increase in tapping rate between 0800h–1200h, a fall in speed from 1200h–1400h, followed by a recovery until 1800h. A similar afternoon trough in performance was reported by the famous psychiatrist, Kraepelin in 1893 and by Gates (1916). A historical account has been written by Colquhoun⁷², but for a more detailed record, see Lavie's review⁷³ (from where the material above is sourced). The problem with a majority of these early studies (eg. Ebbinghaus, Lombard and Dressler) was that they only used one subject, namely, themselves; and on the whole, they failed to control for practice effects in their methodology. So the inclusion of their data, as additional evidence for the biphasic sleep model, must be treated with care.

The first rigorous study of recent times to which we can refer to with some confidence, was conducted by Blake in 1967⁷⁴. He studied a group of naval ratings performing a range of tasks at 0800, 1030, 1300, 1530, and 2100h. The tasks included a simple reaction time test, a card sorting task, an arithmetic test and a letter cancellation task. It was a repeated measures design in which subjects conducted each test on a different day — the five tests took five days. The results can be seen in the graph overleaf; fig.4.

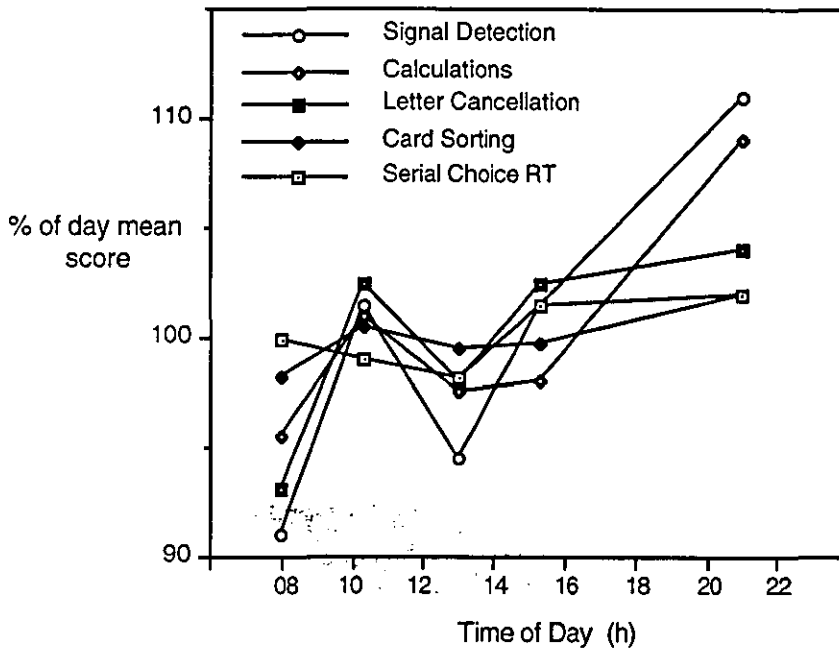


FIGURE 4. Variation in performance over the waking day [ref. 72]

It is clear from Blake's data that a marked fall in performance is observable at the 1300h test session. This transient drop in performance during the early part of the afternoon has been referred to as the "*post-lunch dip*".

Over the years, researchers have continued to point out this afternoon dip in their data. However, their attempts at *explaining* this dip have not proved so fruitful. The early 19th century efforts of Kraepelin, Griesbach and others, tended to veer towards an explanatory model based on 'mental fatigue'⁷³. The growth of 'industrial psychology' between the two world wars, was to usurp this line of research from the educational psychologists. Their input, in describing daily 'work curves', again drew heavily on ideas of 'fatigue' in concord with earlier pedagogical studies. New ideas, and a fresh approach, came from Kleitman "who bridged the gap between the early period of research and studies related to current interest in rhythmic aspects of human behaviour."⁷³ He saw a parallelism between diurnal changes in performance, and the course of body temperature. This idea was to be challenged by Colquhoun who presented an explanatory model based on 'arousal theory'. A fuller discussion of these opposing theories will be presented in section 2.

The leading exponent of the biphasic sleep theory, Roger Broughton, views the post-lunch dip as an aspect of circadian rhythmicity, based on a precise 12-hour interval, and timed by that elusive 'biological clock'. A fixed ultradian-like periodicity that is open to mathematical modelling is envisaged. The dip is seen as a clear expression of the 12-hr. circadian propensity for delta sleep. After reviewing numerous experiments on 'round-

the-clock' performance studies (where sleep deprivation also becomes a factor), he notes that between the hours of 1:00–3:00 a.m., a transitory dip in performance also occurs. This finding is juxtaposed with the classic afternoon dip, prompting the observation: “this approximately 1:00–2:00 p.m. decrease in vigilance is almost exactly 180° out of phase with the lowest nocturnal level of vigilance, which is at about 1:00–3:00 a.m.”⁷”

The ubiquitous nature of the post-lunch dip inevitably leads one to posit the central chronobiological question: “Is this post-lunch dip an endogenous rhythm?” An unqualified, yes, according to Broughton’s work. Isolation studies by Aschoff et al., (reported by Colquhoun⁷²) in which subjects experienced “an abrupt shift in the phase of the zeitgebers” were to clearly show that ‘afternoon’ impairment persisted, independent of external cues.

However, if one were to trace the etymology of the term ‘*post-lunch dip*’ the suggestion of food or lunch is implicit. The belief that a big lunch causes sleepiness is almost part of folklore; the big Sunday joint followed by the big Sunday ‘snooze’. This ancient wisdom goes back to the writings of Aristotle. But mythology and biology are paths that do not always meet. The evidence is mounting against the presumed association between food and post-prandial drowsiness. Food may have a role to play, but it has a limited influence on the ‘endogenous’ post-lunch dip. Kleitman is partly to blame for advancing the myth of a food-related dip in alertness, when his weighty words proclaimed the dip to be: “...related to a ‘let down’ experienced after heavy noontime meals.”

The importance of the ingested meal was examined by Blake – in one experiment he varied the timing of the meal to see if the post-lunch dip was robust in its appearance, independent of meal timings^{see 72}. On separate occasions, meals were given at 1100h, 1200h or 1400h. All tests were carried out at 1300h. The scores at the 1300h tests were almost identical in all 3 situations, indicating that the timing of the meals have little bearing upon the post-lunch dip. There has been a resurgent interest in this whole area of food and behaviour, often generating unwarranted claims and headlines in the media and popular science journals. Most of the work implicating the role of food comes from British workers at the “MRC Perceptual and Cognitive Performance Unit” at Sussex University. Their work, together with the findings from other laboratories advocating the importance of carbohydrate-rich meals will be discussed later. These topics are central to any discussion of ‘afternoon sleepiness’ and will therefore receive substantial explication and debate in the latter half of this thesis.

1.2.8 THE AFTERNOON PRESSURE FOR SLEEP (APS)

The transient sleepiness experienced in the afternoon, is one aspect of the 'biphasic theory of sleep' or the 'circasemidian theory'. It is an important half of the 12-hour sleepiness cycle. This thesis is a report of an investigation looking into the expression of that secondary sleep cycle. Unbelievably, there exists no specific term that accurately relates this part of the sleepiness equation, to the biphasic theory. I need a descriptor, or an 'operational term', that will precisely describe this propensity towards a second sleep episode.

I could use the term, '*Post-Lunch Dip*'; it possesses a literal appeal, that stands out in the bland glossary of scientific phraseology. However, the term is not accurate enough for my purpose. It was specifically coined to describe a drop in *performance* during the afternoon, which is just one manifestation of the physiological sleepiness I am trying to recount. Moreover, the term is tagged with the word *lunch* so wrongly acknowledging the centrality of food, and thereby contributing to a scientific misnomer.

The term '*siesta*', is impractical because it is culture-bound with demographic limitations, and its literal translation and dissection, renders it archaic (see footnote on page 6). A more universal term embracing the siesta, would be '*napping*'.

Napping has been described as any period of sleep which is less than 50% duration of the nocturnal sleep – from 'microsleeps' to 4 hours³⁸. Webb, relates a humorous definition of a nap as "any rest period up to twenty minutes duration, involving unconsciousness but not pyjamas".⁴¹ The term 'napping' would have been acceptable, were it not for the fact that it has no temporal frame of reference, i.e. naps can occur at any time; morning, afternoon, early or late evening. A preponderance of 'lighter naps' found between 0800–1100h have been reported by Nakagawa⁵⁰ in addition to an afternoon cluster of naps. More importantly, morning naps are qualitatively different from afternoon naps. The sleep infrastructure of morning naps contain more REM sleep compared to the increased SWS found in afternoon naps. Disentrainment studies by Nakagawa also point to morning naps being longer in duration. The coterie of nap types, e.g. 'prophylactic napping' of students in preparation of a night of revelry, together with recuperative and appetitive naps, are all driven by behavioral motives, independent of circadian elicitation. The widespread distribution of naps, and its generality makes it a nebulous term: all I am interested in are the naps located centrally, within this distribution i.e. the afternoon sleep episodes.

To explain their findings from isolation studies, Weitzman et al.⁵⁷ developed a nomenclature based on 3 parallel domains ; which they termed: i). Sleep Domain ii). Nap Domain and iii). Awake Domain. This seemed a more feasible description, as

the 'Nap Domain', was phase related (180°) to the nocturnal course of temperature and sleep. However, this classification perpetuates a misconception of sleep and wakefulness as distinct 'separate domains', when in reality, "sleep and wakefulness are complimentary phases that are mutually interactive."⁷⁵

My failure to find an adequate operational descriptor has forced me to coin a new term. It is a neology I undertake reluctantly, given the already over-jargonized lexicon of 'sleep literature' — for example Freemon^{see 61} has documented 25 different terms for REM and NREM sleep. Apart from Weitzman's 'Sleep Domain' and 'Nap Domain', we are faced with terms like Lavie's grandiloquent 'Forbidden Zones of Sleep' and 'Gates of Sleep', in addition to Strogatz's^{see 60} 'Wakefulness-Maintenance Zones'.

I consider 'napping' to be a generic term, that is all-inclusive and abstract in meaning, and have found the term 'post-lunch dip', with its implied food-sleep causal nexus, to have lost its usefulness. I have decided upon a term that is simple, unambiguous, and temporally explicit. The term I propose to use, is the 'Afternoon Pressure for Sleep' which I will abbreviate, the APS.

1.2.9 CONCLUSION

Reviewing an area of sleep research that is conspicuous by its lack of research output, makes for a very difficult task. One is compelled to collect data from a wide and often disparate range of studies, before drawing accurate and germane inferences. However, I have not marshalled data in the absence of a theory. My 'intelligence-gathering', has been conducted within the framework and agenda, first articulated by Broughton in his 'biphasic sleep model'⁷. Webb, quoting Hess (1965) makes the point: "...it is an illusion to suppose that simple facts have by themselves the power to constitute a theory. It is only the inference based upon them that will advance our viewpoints"^{see 19}. To that end, the data I present is confined to that deemed the most salient, and where the inferences to be drawn, are the least ambiguous. The limitations of space has meant that other items of evidence could not be brought to bear: for instance, the potent effects of alcohol at midday, and the frequency of narcoleptic sleep attacks during the time of the APS, are examples of deliberate omission.

It has been the aim of this review to present the APS, as a secondary period of sleepiness that is best understood within the context of the biphasic theory of sleep. Its functional significance has yet to be elucidated, but tentative speculations have been presented.

The sheer diversity of biphasic rhythms becomes apparent from Aschoff's review of the animal data. Its pervasiveness extends from terrestrial, to avian, and aquatic

habitats, from carnivores to omnivores and from nocturnal to diurnal creatures. At varying points of evolutionary history, natural selection has favoured the 12-hour periodicity for its survival value. The circasemidian rhythm runs close to the mean tidal period, and is linked to the sun, at (or near) its zenith. Parallels have naturally been drawn with the geophysical environment⁸. The sleep patterns of our early ancestors may have been influenced by the physical environment. In this technological age, we have 'mastered' our physical environment, but our sleep-wake organisation remain suppliant to new cultural forces and have had to acquiesce to societal demands. Inherited rhythms are ignored or overruled.

Nevertheless, these endogenous rhythms still exist, and are given free expression in isolation studies. The fact that the APS may not appear in everyday life, is attributable to masking by social and occupational factors. Submitting to an afternoon nap during the APS, can be beneficial to both mood and performance. Campbell and Zulley have given considerable thought to the issue, and have remarked: "Do our non-napping societies impose an artificial ceiling on performance potential ?that we can get along adequately without naps should not be interpreted to mean that we would not function more efficiently with them."⁴⁵

One is tempted to poetically conclude that the biphasic pattern has never really left us: its roots lie in the depths of our subaqueous history. During infancy, it bobs on the surface, only to be weighted down by society. In old age, at the close of life, it emerges from the bottom, able to weigh anchor at last. In this seascape of sleep, it is the siesta cultures that merrily ride the waves, whilst we find ourselves trapped in an estuary of our own making.

Because of the potential masking effects by social and occupational factors, any attempt to measure putative sleepiness during the central waking period will be greatly confounded. Therefore, if any endeavour is to succeed in isolating and charting the progress of the APS, an efficient 'unmasking' procedure will be paramount. The studies to follow represent just such an attempt, in trying to unmask the APS. Essentially, subjects will be placed in situations deemed facilitative to the onset of sleep; the underlying premise being that if physiological sleepiness is present, these conditions will allow it to surface. The cognitive and physical environments will be manipulated accordingly.

In the first experiment, boredom and room temperature will be used to exacerbate any afternoon sleepiness, whilst in the second experiment, large afternoon meals in association with previous night sleep reductions, will aim to trigger the APS. Many of the challenges to be presented in these experiments (eg. big meals and warm temperatures) have been widely held as causes for the siesta and napping behaviours.

However, these common assumptions and folklore beliefs have yet to be subjected to scientific scrutiny, in the manner outlined above.

The genuine pragmatic concerns of the APS, and its sequelae – coming as it does in the middle of the waking day – means that we must work towards experimental designs that are ecologically valid, which will allow us to extrapolate our findings to the real-world. Consequently, all the experimental manipulations will strive for a realism, partly to be achieved by presenting experimental challenges designed to be as naturalistic as possible. Moreover, the administration of frequent tests will allow this transient window of afternoon sleepiness to be accurately gauged, so providing a temporal reference by which to frame this zone of sleep vulnerability.

2

Unmasking The APS

2. Unmasking the APS.

2. Introduction

2.1 Time of Day

The 'Afternoon Pressure For Sleep' (APS) is a transitory phenomenon with a narrow time band, located in the early part of the afternoon. It represents an intrusion of sleepiness half-way through the waking period, reflecting a changing physiological state, at a particular time of day. This cyclical wave of sleepiness, must be seen in the context of the entire spectrum of daily circadian cycles, from temperature, blood pressure, hormonal secretions, and so forth. Our general physiology and psychological disposition shows changes during the course of the day – how very different we appear to be from hour to hour; sullen at an 8.00 am. breakfast, aggressive during the 5.00 pm. rush hour, and tranquil at an 8.00 pm. candle-lit dinner. Concomitant with these daily dramas' are the rhythmic biochemical fluctuations to our 'interieur milieu'. A highly simplified schema showing our body in constant flux is illustrated below.

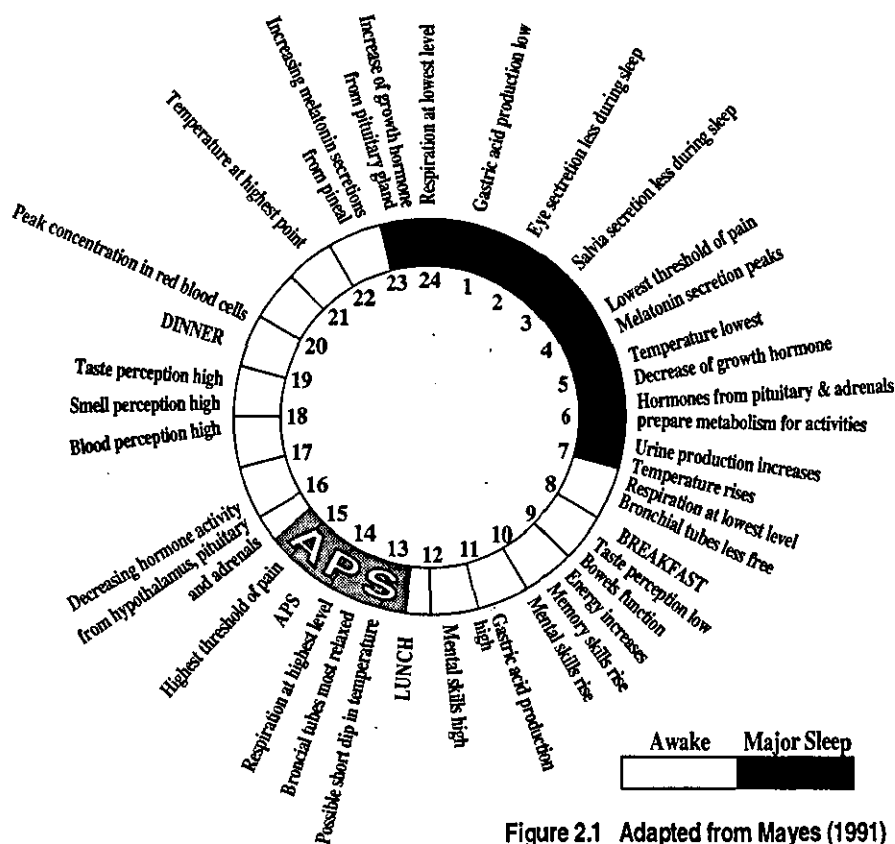


Figure 2.1 Adapted from Mayes (1991) Ref.78

The practical implications of these 'time-of-day' fluctuations are immense. A recent survey (1992) reported in the *BMJ*, finds clear-cut evidence of a diurnal variation in attacks of stroke: for instance, subarachnoid haemorrhages typically peak between 8.00–10 am., and again at 6.00–8.00 pm⁷⁹. The effects of Haloperidol will be seven times greater if administered at 4.00 pm. as compared to a 4.00 am. dosage⁴. The anti-cancer drug, Cisplatin is most effective when taken late in the afternoon, when the kidneys are at their most active; an evening dosage can prove lethal to the kidneys⁸⁰. An allergic asthmatic attack is most likely to occur around midnight than at any other period due to fluctuations in the immune system; and heart attacks are more likely at 9.00 am., and least prevalent around 9.00 pm⁸¹.

The catalogue of fluctuating behavioural and physiological parameters is vast. Embedded in this corpus of data is the transient APS: an aspect of the circadian rhythm of sleepiness, as tangible as any of the biological rhythms mentioned above.

2.2 SLEEPINESS: The 'amber light' of consciousness

It is relatively easy to sample a biochemical compound at various points of the day, and thus obtain a circadian profile of its activity. Performing a similar monitoring exercise with the state we call 'sleepiness', is problematic. Without an accepted metric, it is difficult to define and gauge levels of sleepiness. Yet, it is present at distinct periods of the day with variable intensity, and like the APS, it too is an example of biological change, with real-world consequences. To understand the APS, we must attach some meaning to its precursory manifestation – namely, the sensation we call 'sleepiness'.

Sleepiness is a transitional phenomenon, bridging the parental states of wakefulness and sleep, and thus possessing qualities of each condition⁸². As a state, sleepiness may simply reflect a 'pressure for sleep' and operationally be defined as "the probability of transition from wakefulness to sleep ($\text{Pr} [W \rightarrow S]$)."⁸⁴ This transition has been described as the "most remarkable behavioural and biological metamorphoses occurring in nature"; an example of extraordinary CNS adjustment and reorganisation between the two states⁸². Sleepiness may also represent a physiological need state; a drive akin to hunger or thirst⁸³. Attempts to locate the physiological components of sleepiness have met with varying degrees of success. In humans there are indications of increased alpha and theta activity; in animals, increased ventral hippocampal spike activity and the pharmacologic involvement of serotonin, catecholamines and acetylcholine suspected⁸².

The transition from wakefulness to sleep must be smooth: the interjecting sleepiness serving as an 'amber light', that warns and prepares the system for a change to the conscious state. The state of sleepiness – a prelude to the shift from wakefulness to

Whereas the latter authors are concerned with the *nature* of sleepiness, in terms of its qualitative aspects (Broughton) and its functional significance (Horne), Carskadon and Dement look instead to sleep tendency or *propensity* as a way of distinguishing two types of sleepiness. They have identified ‘Physiological sleepiness’, which is said to reflect an underlying sleep propensity that surfaces if subjects are placed under quiet, restful (soporific) conditions. They developed this classification, in part, to explain their instrument which measures sleepiness, the MSLT [see page 140]. On the other hand, ‘Manifest sleepiness’ is the subjective sleepiness that supervenes during boredom or which is depressed during excitement, i.e. it is mediated by the situational context. Their modus operandi was guided by the clinical need to quantify pathological cases of daytime sleepiness. Therefore, their classification is not as illuminating as the broader theoretical approach of Broughton or Horne, which ultimately seeks to address issues, central to the very ‘nature’ of sleep and sleepiness.

To date, sleepiness has been treated as a unitary concept. These newer constructs which purport that sleepiness is multidimensional, must experimentally evince the different forms of sleepiness. Broughton has attempted to correlate the different forms of sleepiness with subsequent ‘sleep onset architecture’ i.e. REM sleepiness with SOREMP’s (narcoleptic attack) or NREM sleepiness with NREM sleep. The value of Broughtons attempts using subjects with sleep pathologies (narcoleptics), has been questioned⁸². Glovinsky et al.^{see 82} induced SOREMP in ‘healthy’ subjects (via 2-night REM deprivation) and compared the nature of this REM sleepiness with NREM sleepiness. The MSLT and SSS scores failed to separate the two forms of sleepiness.

Horne’s abstraction of sleepiness is more amenable to experimental dissemination, into its component Core and Optional aspects. These characteristic forms of sleepiness can be produced by deprivation of Core/Optional sleep. Recognising that the MSLT is highly prone to behavioural influences, comparisons were made with the alternative, Repeated Test of Sustained Wakefulness (RTSW), where the capacity for wakefulness is at issue. It was suggested that the MSLT measured global sleepiness (Core plus Optional), whereas the RTSW was more sensitive to Core sleepiness⁸⁷. By examining differences between the two procedures, levels of Optional sleepiness could be ascertained.

MSLT		RTSW		
C	+	O	–	C
<i>Core</i>		<i>Optional</i>		<i>Core</i>
				=
				O
				<i>Optional</i>

Another method of partitioning Core and Optional sleepiness is by the judicious use of psychological tests. If the scores of lengthy and monotonous reaction time tests, can be improved by the offer of incentives, then the presenting sleepiness must be flexible in

kind. An instance where sleepiness can be masked and/or assauged would be indicative of Optional sleepiness. To expose Core sleepiness, sophisticated neuropsychological tests are required. Tests of divergent thinking - probing 'creative thought' - make strong demands upon cerebral prowess⁸⁸. Impairment in these tests cannot be alleviated by incentive, and must therefore be the result of a compromise to Core sleepiness⁸⁷.

Another form of sleepiness that often occasions *oversleep* (described by Globus,⁶⁹) has been dismissed as a subjective state more akin to *fatigue* than genuine sleepiness⁸⁷. Likewise, 'sleep inertia', presents yet another category in this controversial taxonomy of sleepiness^{see 82}. In summation, sleepiness is now widely regarded as a multi-faceted phenomenon. The sleepiness of the APS may not be synonomous with the sleepiness that presages nocturnal sleep. The analysis of sleepiness, in terms of its typology and hypothesised function is a first step. From this, flows the next issue of importance – our ability to 'assess' sleepiness in its various forms.

There are three broad types of tests currently used to assess sleepiness:

1. Objective measures - Performance tests, MSLT, physiological correlates.
2. Subjective measures - Introspective self-reports e.g. SSS, analogue scales.
3. Behavioural indicators - Hallucinations, memory lapses, apathy, low sociability.

The third category above, the 'behavioral indicators', are measures more germane to gauging excessive sleepiness that results from sleep deprivation, where perceptual distortions⁸⁹ and slurred or atonal speech (aprosodia) are much in evidence⁸⁷. The daytime sleepiness of the APS is not so severe, and thus requires more refined and sensitive tests. To this end, for these studies into the APS, I have turned to objective performance measures (reaction time), and the MSLT; with temperature readings and continuous heart rate monitoring as sources peripheral physiological indices. These tests are complimented by self-report subjective measures. As these tests have the potential power to 'unlock' the APS, I will briefly discuss their use in the scenario of daytime sleepiness testing. Comments on 'subjective' alertness is followed by a discussion on 'objective' performance measures.

2.3 SUBJECTIVE ALERTNESS

In the section above, I have described sleepiness as an 'amber light' of consciousness, that warns the body of an imminent change to its status. In a similar vein, Monk has described sleepiness as a 'behavioural messenger', which urges the body to prepare for sleep, by the instigation of bed-time rituals⁹⁰. It is the careful observance of these signals that constitutes good 'sleep hygiene'. These behavioural responses point to the

'subjective' quality of sleepiness – a psychological strategem that interfaces with the physiological processes which produce sleep⁹¹.

We have the capacity to introspect, and describe these subjective changes with adjectives like 'drowsiness', 'lethargy', 'vigour' and 'tiredness'. Numerous self-report measures are available to gauge sleepiness; from the 7-point Stanford Sleepiness Scale to various Analogue Scales [see pp. 59-60]. A champion of self-report measures has been Thayer¹⁵⁹, who has emphasised the correlative power of these scales with physiological test batteries [see later]. The nervous system has been conceptualised as a hierarchical system with the cortex at the top. According to Thayer, introspective self reports represent a 'cerebrocortical function' encompassing the highest level of nervous integration. If sleepiness is indeed multidimensional, then it may be the case that, subjective tests probe a form of sleepiness that is qualitatively different from the sleepiness typically uncovered by objective measures.

Nevertheless, these self-report tests have revealed circadian rhythms in subjective alertness, with distinct time-of-day fluctuations. Folkard has superimposed data from several studies which traced diurnal variations in subjective alertness^{see 91}. A clear trend emerges. Under normal sleep conditions, alertness rises sharply in the morning, to peak between 11.30 and 14.00 h.; after which there is a sharp decline. Only one study in Folkard's assemblage of reports – that of Thayer (1978) – described a post-lunch dip in alertness. The mechanisms underlying 'subjective alertness' are discussed below.

In contrast to the close links between temperature and performance [see section 2.4], no parallels were found between the circadian course of body temperature (with peaks at 20.00 h) and subjective alertness⁹¹. However, when Froberg deprived subjects of sleep (a 72-hr. vigil), the circadian rhythm of subjective alertness showed a close parallelism with body temperature⁹⁰. So, if the sleep-wake cycle is depressed (sleep deprivation), the temperature oscillator will assume control over the rhythm of subjective alertness. The respective influences of the temperature oscillator, and the sleep-wake oscillator, can be found by looking at each rhythm separately, after they have been split by an experimental procedure called 'forced desynchronisation'.

In a complex experimental design by Monk et al.^{90, 91}, subjects underwent long term temporal isolation in a bid to dissociate the period of the sleep rhythm from the temperature rhythm. They discovered that when subjective alertness was controlled by the sleep-wake cycle, maximal alertness occurred 8 to 10 hours after awakening (i.e. early afternoon). This was contrasted with the evening peak at 20.00 h., which occurred when the temperature oscillator controlled alertness (e.g. during sleep deprivation). Interestingly, one of their four subjects displayed a marked post-lunch dip in alertness.

It seems that in reality, both oscillators control subjective alertness – but the more powerful sleep-wake oscillator has a greater influence under normal nychthemeral conditions, thereby explaining the early peaks we usually find in the alertness curve. The pre-eminence of the sleep-wake oscillator, in this situation, is desirable as it is more adaptive and flexible than the rigid temperature oscillator⁹⁰. Subjective sleepiness - the signal for physiological sleep - would be thwarted if it relied on the inflexible temperature oscillator in times of phase adjustments (e.g. jet travel) or sleep alterations.

2.4 OBJECTIVE PERFORMANCE: Diurnal Variations

A sleepiness rhythm that is superimposed upon the daytime hours, is difficult to detect by *mental* introspection alone. We do however recognise the *physical* symptoms of sleepiness. Apart from the overt bodily sensations of itchy eyes, yawning, and fatigue, we realise – with little surprise – that sleepiness leads to impaired performance. Numerous laboratory studies of sleep deprivation, emphasise the detrimental effects of sleepiness upon performance abilities,^{92, 17} and data from field studies of vehicular^{93 - 95} and industrial accidents⁹⁶ (the scale of Three-Mile Island and Chernobyl) would implicate sleepiness in the causation of these disasters.

If this relationship holds true, then it should be possible to use performance decrements to gauge the presence, and magnitude, of sleepiness. Performance tests strategically placed throughout the waking day could uncover the APS; an afternoon performance nadir signalling the APS signature. The investigation to be reported in this thesis will exploit this ‘performance–sleepiness’ relationship to operationally define the APS. A degree of parsimony will be exercised in the interpretation of this performance–sleepiness correlation. Performance variations may have an inherent rhythm, quite independent of the influences of the sleep–wake continuum. Therefore, we need to retrace our understanding of the diurnal fluctuations in performance, prior to its use as a viable litmus test for the APS.

The daily fluctuations in performance measures have been put down to 3 factors:

1. Mental fatigue.
2. A close parallelism between performance and the temperature rhythm.
3. A circadian process linked to the sleep-wake and/or arousal rhythm..

Explanations based on ‘mental fatigue’ flourished at the turn of the century – persisting until the 1930’s; but these ideas on ‘fatigue’ were taken up mainly by industrial psychologists, and latterly, ergonomists, with little enthusiasm from sleep researchers. The post-war work of Nathaniel Kleitman had a greater and lasting impact. Kleitman argued that performance was causally related to the temperature rhythm— so strong did

he regarded this parallelism that he concluded: "there is probably no reaction time curve independent of temperature"^{see 73}. Apart from examples of motor tasks, he also asserted that 'mental performance' showed a causal relationship with temperature because:

"either.....a). mental processes represent chemical reactions in themselves
or, b). the speed of thinking depends upon the level of metabolic
 activity of the cells of the cerebral cortex" (an increase of
 temperature will speed up thought processes).

Kleitman confidently suggested, that time-consuming performance tests were rendered unnecessary, as one could gauge performance efficiency simply by consulting the diurnal temperature chart. This optimism was challenged by Wilkinson⁹⁷. He noted that in one study (Kleitman and Jackson [1950]), this temperature-performance association was only found in "two minor RT tests, each of 5 minutes duration", whilst the major flight simulation task (occupying two-thirds of the test battery) failed to show the predicted temperature-performance parallelism. This relationship, where present, also broke down under conditions of sleep loss; a refutation of its robustness. In a related investigation, Wilkinson and Edwards⁹⁸ studied the adjustment of ratings to naval watch systems over a course of 12 days. Temperature was recorded, and lengthy test batteries of 53 minutes each were administered (cf. Kleitman's 30 mins.). Subjects on the fixed night watch (2400-0400 h) showed a temperature adjustment by day 9; the evening temperature dip was removed or totally reversed. Despite the ensuing *rise* in evening temperature, performance on two of the three tasks, did not show the expected improvement. In a series of experiments Rutenfranz et al.^{see 99} also failed to find a link between performance and temperature and concluded that Kleitman's observations were due to the chance (independent) phasing of the temperature and performance rhythms.

The nature of the performance task must also be taken into account. Folkard et al.^{see 99} noted that tasks with a low memory load showed some correlation with temperature; those with an intermediate load showed a zero correlation, and those with a high load, a negative correlation. It appeared that this parallelism is both fragile, and may only "hold for a fairly restricted range of tasks." The association between temperature and performance generated much attention in the 1960's. Wilkinson later reflected: "If the 60's were the 'decade of confidence' in the temperature-performance relationship, the 70's have, on the whole, been years of reaction"⁹⁷.

The next major advance in thinking came from Colquhoun¹⁰⁰ in the early 70's. He invoked arousal theory to explain these time-of-day effects. While accepting that a temperature-performance parallelism is observable in some cases, Colquhoun was careful not to attribute causality to this association. He was particularly intrigued by the phenomenon of the post-lunch dip; a noon-time drop in performance widely reported

by Blake⁷⁴, and which also drew comment from Kleitman. According to Colquhoun, “This post-lunch dip is of special interest, since there is no corresponding drop in temperature level after lunch, its occurrence would appear to refute Kleitman’s thesis...” On closer examination, Colquhoun noted that those subjects exhibiting the dip, reported ‘*sleepiness*’ to be their most prominent subjective sensation above anything else. In the light of the present work on the APS, this observation may prove significant.

Colquhoun, equates the idea of sleepiness “with arousal (or rather lack of it)” and identifies the fluctuations in performance with fluctuations in arousal or ‘activation’ levels. The evident fall in sleepiness, seen during the unfolding day, is compared to the rise in levels of basal arousal. Arousal is low early in the morning, but then rise sharply to an early evening peak – a post-lunch dip may interrupt this rising arousal curve at midday. In effect, Colquhoun proposes a ‘sleepiness rhythm’, not unlike the proposition of Gates (1916), – which was based on Michelson’s (1897) views of ‘fluctuating sleepiness’ – and Howell’s (1897) ideas of cerebral recovery from the ‘anaemic’ state of sleep¹⁰¹.

However, a simple notion of rising arousal, does not satisfactorily explain the finding that some performance tasks peak in the morning (a time of low arousal), whilst others later in the day. Colquhoun, suggests that the coupling of arousal levels and performance efficiency is best understood by the application of the Yerkes-Dodson ‘inverted-U’ paradigm. Quite simply, each performance task will have its own level of optimal arousal – a simple and boring task will require a high level of arousal (and peak later in the day), whilst a difficult and complex task requires minimal arousal (and so peak early). While accommodating, this uni-dimensional theory of arousal, has been described as over-simplistic by Folkard^{102, 99}. His studies on memory processes and shift-work adjustment has led to the discovery that tasks with high and low memory loads do not adjust at the same rate – memory intensive tasks adjusts faster. There is thus the suggestion that there may be more than one oscillator controlling performance, leading Folkard to argue for the adoption of a more “sophisticated multifactor theory” of arousal.

The arousal model, and the inverted-U concept in particular, has provided a useful framework by which to interpret these diurnal changes in performance. Nevertheless, there is the acknowledged caveat that arousal is difficult to gauge, and quantify, and it remains a scientific concept that is very difficult to prove or disprove. A recent review by Neiss^{103, 104} and Anderson¹⁰⁵ examines the issues in depth. I will return to this topic.

Irrespective of the underlying mechanisms responsible for diurnal performance variations (mental fatigue, temperature or arousal), one thing is clear: performance

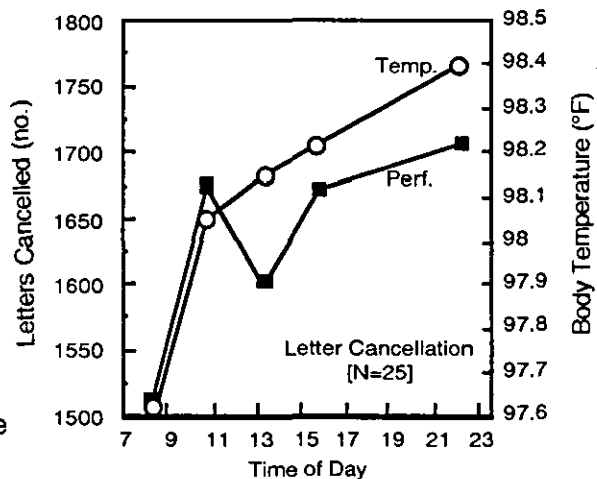
decrements remain a useful index that is sensitive to daytime sleepiness. According to Dinges¹⁰⁶, performance measures may serve as “a critical *probe* of CNS capability...” But, being a behavioural act (and thus volitional) it may not be as clear-cut an indication of sleepiness as physiological indices. Nevertheless, it is argued that performance measures are good estimates of “zones of [sleepiness] vulnerability” that can be demonstrated in the laboratory, so negating the need for costly field studies¹⁰⁶. The ease of quantifying performance scores also makes it amenable to reliable statistical evaluation. The value of performance tests are well-recognised and credible: on that basis, I shall incorporate performance measures into this study of the APS.

2.5 THE PROBLEM OF MASKING

The subjective and objective instruments used to measure sleepiness are plentiful, and they all vary in degree of sophistication – from the 10 cm. line of an analogue scale to complex psychophysiological techniques. However, the ability of these instruments to measure sleepiness during the central waking period, is greatly confounded by the process known as *masking*. This refers to the suppression of an endogenous rhythm, like the APS, by exogenous factors. The overt circadian rhythms are after all, products of both endogenous and exogenous influences. The superimposition of exogenous factors upon the endogenous APS, can *mask* or suppress its expression, so rendering attempts to measure this sleepiness, ineffective. Suppression of the afternoon sleep drive is wholly feasible given the flexibility of the sleep-wake cycle. This flexibility is illustrated by our ability to temporarily offset sleep; adapt to shift-work, and our easy entrainment to the 24-hour geophysical rhythm, when our biological periodicity is nearer 25-hours. The masking factors in the ‘real-world’ which nullify the encroaching APS, include occupational and social demands⁴⁵. It is not surprising that performance data do not always show decrements during the APS, as powerful masking factors can override the momentary lull. The resulting inconsistencies in the performance data will be discussed.

In his classic studies, Blake uncovered strong diurnal trends in a range of performance tasks¹⁰⁷. In one such task, subjects had to cross out the letter ‘e’ from sheets of english prose. This ‘letter cancellation task’ was simple, but laborious. His results, showing an afternoon dip, is plotted to the right.

Fig. 2.2 Body Temperature & Performance
[Adapted from ref:107]



Folkard contrasted Blake's results with data from 3 similar studies; (Klein et al. (1972), Fort and Mills (1976), Hughes and Folkard (1976))^{see 102}. In the comparison, only Blake's data was to show a dip in performance; a finding which Folkard found, "somewhat surprising." An equivalent study by Patrick et al.¹⁰⁸ also failed to unmask a significant drop in performance. By way of causal explanation, the authors lay stress on the fact that the duration of their test was much shorter than that used by Blake. Similarly, Folkard noted that the only real difference between Blake's study, and the 3 comparative studies which failed to reveal a dip, was in the duration of the test. Parenthetically, Blake's subjects were tested for a full 30 minutes, on this most laborious of tasks.

These discoveries allude to a relation between the 'unmasking' properties of a performance test, and the duration of that test. From his experience of sleep deprivation studies, Wilkinson found that 'sleepiness' was only detectable if the administered tests were sufficiently long¹⁰⁹. In addition, the tests had to be simple, monotonous, and uninteresting. Blake's visual search task met these criteria, and, in doing so, unmasked the APS trough.

Another research paradigm that has successfully unmasked the biphasic nature of sleep has been the isolation studies employed by Campbell and Zulley⁴⁵⁻⁴⁹. These were reviewed in section 1.2.5. Essentially, by removing exogenous factors (physical and social zeitgebers) sleepiness was offered a "course of least resistance." Naps under these conditions were not randomly distributed across the nycthemeron, but clustered around 1400h.

The unmasking of the biphasic rhythm under more naturalistic conditions was examined by Hildebrandt et al.¹¹⁰. They analysed the frequency of braking errors made by German train drivers. A monthly tally of 2,000 errors was recorded from 15,000 engine drivers. There was a bimodal distribution of these errors – a nightly peak followed by a secondary peak, 12-hours later, during the early afternoon. They noted that the post-lunch dip was more likely to occur if the work shift began early, i.e. the performance decrement was attributed to "accumulated tiredness". So, in this case, gross occupational *fatigue* was sufficient to unmask the APS.

Finally, Colquhoun has pointed out that time-of-day effects are most prominent under conditions of sleep loss¹⁰⁰. He cites as evidence, a study by Alluisi and Chiles (1976), in which diurnal rhythms in performance only became apparent after 40-44 hours sleep deprivation. Similarly, Lavie has discovered that the midafternoon peak in sleepiness (the "secondary sleep gate") is augmented by sleep deprivation which activates a putative "hypnogenic mechanism" at this time of the day^{111, 112}.

In summary, we can see from the reported data that the following conditions have been successful at unmasking the APS:

1. Lengthy (monotonous) test batteries [102, 107, 109].
2. Temporal isolation studies [45-49].
3. Excessive occupational fatigue [110].
4. Sleep deprivation studies [100, 111, 112].

On closer inspection, we see that these findings and research protocols have one thing in common: they all serve to *lower* arousal.

2.6 MANIPULATING AROUSAL

The conclusion that emerges from contrasting the above studies is that drops of arousal do occur, either as a direct function of the experimental manipulations, designed to procure such biological change; or it may simply reflect an unwanted artefact of the experimental protocol. In either case, there is a strong dearousing force, which has proved useful in unmasking circadian events; in particular, the afternoon dip in alertness and performance.

Under normal conditions, basal arousal levels may fluctuate in a rhythmic circadian fashion – indeed, our explanations of diurnal changes in performance which invoke the inverted-U function, is based upon a premise of changing arousal. Levels of arousal will be low during the time of the APS compared with the early evening (circadian peak), when it will be much higher. Experimentally induced dearousal, arising from one of the protocols described earlier (e.g. sleep deprivation), will amplify the naturally occurring arousal troughs (e.g. the APS) and can be expected to have a minimal effect during the circadian peaks of the early evening. Such external potentiation of arousal troughs, can rescind any masking influence, and the underlying sleepiness given free expression.

However, there are circumstances when the depression of arousal alone, is not a sufficient guarantee to induce sleepiness and/or unmask the APS. A case in point is the the complex '*sleep deprivation-arousal*' controversy. The dearousing properties of sleep deprivation was highlighted by Wilkinson¹⁰⁹ and Murray³, and it even provided a neat theoretical base for the Walter Reed Group to explain their Lapse Hypothesis^{see 17}. Kjellberg¹¹³ reviewed the data on the widely held *sleep deprivation* → *dearousal* relation, and brought to the discussion an important point (earlier raised by Malmö and Surwillo, 1960) concerning 'situational factors' and their interaction in this scenario. Basically, the environmental context, it is said, can intensify or moderate the dearousing effects of sleep deprivation. For instance, a sleep deprived person will not

suffer the debility of sleepiness whilst on an outing at Disneyland as opposed to a “drive through Kansas on a warm summers afternoon.”⁸⁶ Likewise, Wilkinson has shown that the reaction-time of a sleep-deprived subject can improve if ‘knowledge-of-results’ are provided, or in the case of a study by Horne et al., financial incentive¹¹⁴. Clearly, the environmental context is an important mediating factor⁸⁹.

The soporific situations which lower arousal, such as motorway driving or attending to a lengthy vigilance task, are classic examples known to induce ‘*boredom*’. A boring situation will lower arousal – a necessary pre-requisite for the unmasking of sleepiness. There is an arousal trough and an underlying sleepiness, present during the APS – a boring situation that temporally coincides with the APS, will potentiate and so uncover this sleepiness. The same situation when transposed to the early evening, at the alertness crest, will (it is hypothesised) only have a modest effect upon sleep induction.

A novel approach to be implemented in this study, will use ‘acute boredom induction’ as a means of unmasking the APS. The use of boredom induction in this guise, will have the same net result of arousal depression, as achieved by the earlier protocols of sleep deprivation, zeitgeber-free confinement, and subjection to lengthy vigilance test-batteries. The short-term boredom induction to be employed here, has methodological advantages, in that it is:

- ❑ Functionally (and economically) efficient relative to costly bunker studies which are restricted to small sample sizes.
- ❑ Preferable over difficult deprivation studies, where physiological and biochemical perturbations compound the subjective stress of sleep loss (eg. irritation and anxiety reactions)^{109, 3}.
- ❑ Less tortuous than performing multiple test batteries such as the monotonous Wilkinson Auditory Vigilance Task lasting 30-50 minutes. Furthermore, complex signal detection formulations are required to decipher these performance changes (eg motivation with time on task).

For the set requirements of unmasking daytime sleepiness, it would seem that the induction of acute boredom (when critically phased), could furnish the same dearousing effect, at a less exhaustive fiscal and human cost. More importantly, the dearousal procured here, is ‘purer’ in form, in that it does not bring with it the baggage of fatigue, disorientation, and stress, that is commonly found with sleep loss studies, caused by the accumulated hours of prior wakefulness. Short term boredom does not produce a similar stress according to studies by Thackray¹¹⁵ and Suedfeld¹²⁷.

2.6.1 BOREDOM

Boredom is said to be major contributing factor to problems of drug abuse, vandalism, classroom disruption, industrial safety and institutional living (prisons and mental hospitals). Yet, in a literature review (spanning 1926-1981) Smith was surprised to find that the published papers on this important subject averaged less than one per year¹¹⁶.

One of the first psychological theories on the concept of boredom was presented by Barmack in the late 1930's. His proposal had two components: i). boredom resulted from low physiological arousal, and, ii). boredom was due to motivational conflicts between persisting with/and withdrawing from, an unpleasant task (an approach-avoidance conflict)^{116, 117}. Barmack saw a clear association between the 'feeling' of boredom, and the descent into sleep or sleepiness, induced by a drop in arousal. The relation between boredom and low arousal is open to controversy. Hebb stressed that 'curiosity' was a basic human trait – the human nervous system actively sought novel stimulation rather than passively accept repetitive stimuli¹¹⁶. A bored person was thus driven to exploratory behaviours to seek stimulation, argued Berlyne; and consequently, boredom would indirectly *raise* arousal. This contradictory view has received experimental support from London et al.¹²⁹ – but their findings are debateable (eg. heart rate of the high boredom condition increased by about one beat per minute! and the reports of their 'bored' subjects of increased sleepiness and fatigue, is directly antithetical to their final conclusions). However, the general consensus (see review by Thackray¹¹⁵) firmly associates boredom with a state of lowered arousal.

If one hopes to induce boredom then an understanding of the antecedents of boredom is essential. The early work of Hebb and Berlyne in the 1960's suggested that boredom was caused by physically monotonous stimulation¹¹⁸. It is therefore not unusual to find laboratories inducing boredom, with tasks that are deemed physically monotonous. London et al. for example, required subjects to monitor a flashing light in a vigilance task lasting 40 minutes. In another experiment, they induced boredom by asking subjects to repeatedly write the letters "cd" over and over for 30 minutes¹²⁵. Perkins has argued that this kind of monotonous stimulation is not a vital pre-requisite for boredom to occur. He relates anecdotally that a lecture or a lively party, filled with varied stimulation, may still be perceived as boring by an individual¹¹⁸. There must be some 'meaning' to the things we do; tasks must be 'mentally' challenging to stave off boredom. A study by Morton-Williams and Finch found that 50% of school-leavers attributed their early departure from school to feelings of boredom, caused by their inability to *understand* their school subjects^{see 118}. There was also a perceived 'uselessness' towards the subjects being taught. In a survey of a working population, Guest et al. discovered that their sample traced their experiences of boredom to feelings

of: meaninglessness, constraint, lack of interest and challenge^{see 117}. A type of 'content boredom' was particularized by Baldamus – for instance, a book may be perceived as boring, despite it's varied prose, suggesting that the 'content' of the book may not have matched the reader's interest, and so boredom results^{see 117}. In an empirical study using english proverbs and assemblages of 'nonsense words' as stimuli, Landon and Suedfeld found boredom to be highest in groups presented with the meaningless stimuli^{see 118}. The picture that emerges suggests that boredom has a very strong cognitive component. Hill and Perkins have also identified two further components : an affective component (boredom is linked to frustration) and a psychophysiological component (yet to be established; though heart rate changes may prove a useful index). In reviewing a number of studies, I have found that physical monotony is still the commonest strategy employed by researchers to induce boredom (see table 2.2).

Table 2.2 Methods Used To Manipulate Arousal

Study*	Arousal Manipulation	N
Provine, R.R; Hamernick (1986)	Boring: Viewing a colour test card on a TV for 30 minutes. Interesting: Viewing a Rock Music video.	32
Perkins, R.E.; Hill, A.B. (1985)	Examining and rating of 7 pictures of motorbikes.	24
Green, C.; Bell, P.; Bayer W.N. (1983)	Attending to a 10 min. audio tape in which the word "door" and "chair" were constantly repeated.	140
Troutwine, R.; O'Neal, E.C. (1981)	Listening to a boring audio tape for 5 minutes.	40
Thackray, R.I. et al. (1977)	Simulated radar monitoring task. Duration = 1hr.	45
Grandjean, E. et al. (1977)	Boring: Counting nails [at a fixed pace] for 3 hrs. Interesting: Psychomotor tests & music.	25
Bailey, J.P. et al. (1976)	Boring: Simulated radar task with infrequent signals Interesting: Slides of advertisements. Duration: 2hrs.	32
Suedfeld, P. (1975)†	Sensory Deprivation.	
London, H.; Monello, L. (1974)	Writing stories based on TAT cards (Thematic Apperception Test).	32
London, H. et al. (1972)	Boring: Monitoring light flashes for 40 minutes. Interesting: Writing stories to match TAT cards. Boring: Write the letters "cd" repeatedly for 30 mins. Interesting: Write stories in response to pictures from a magazine.	20 44
Mc.Bain, W.N. (1970)	A paced key-punching task. Duration = 42 mins.	25
Geiwitz, P.J. (1964)	Posthypnotic suggestion to induce boredom.	4

* See reference list 119-131 for bibliographic details.

† A review paper incorporating many studies.

Asking subjects to stare at a TV test card for half an hour, or subjecting them to a constant drone from a tape with the word “door” repeated every 2 seconds, or asking them to write the letters “cd” incessantly on a blank sheet for 30 minutes is physically monotonous (sensory and motoric), fundamentally unnatural, and a probable source of much irritation. Experimental attempts to raise arousal and interest levels have suffered from the same inelegance. Writing stories in responses to TAT cards (pictures) has been used to generate ‘interest’, but its inherent artificiality ranks with the other bland strategies employing white noise as an arouser (stressor?) – see Baker et al.¹³², Cohen¹³³ and Davies et al.¹²².

I intend to take a more naturalistic approach to the induction of boredom. Subjects will be presented with real-world stimuli, requiring minimal situational demands. In essence, boredom will be treated as a *cognitive* condition, which arises when a subject “perceives stimulation as relatively meaningless or irrelevant to himself.”¹¹⁸ A scenario more akin to classroom boredom or the ‘dentists waiting-room’ will be cultivated. The prevailing *mental underload* will give rise to a condition simulating ‘content boredom’.

2.6.2 BOREDOM AND GENDER

Early industrial research has sought to identify factors which make certain individuals (workers) more susceptible to boredom. The individual traits which have been identified¹¹⁸ as predictors of boredom susceptibility include:

- | | |
|------------------|------------------------|
| a). Intelligence | b). Extraversion |
| c). Age | d). Emotional lability |

From this, the stereotypical individual most prone to boredom, would be intelligent, young, extroverted and possess a ‘nervous’ disposition. There is however, no general agreement on the role of intelligence and extroversion, as factors predisposing one to boredom.^{117, 134} Conflicting views also prevail on the question of boredom susceptibility and age.^{134, 135}

Less controversial, is the mounting body of research findings, suggesting that gender plays an important part in determining boredom proneness. In a recent study, Sundberg et al.¹³⁶ reported on boredom susceptibility of young college students, from Western and Eastern countries (America and Australia versus Lebanon and Hong Kong). In this cross-cultural study, the males from all four countries showed the greatest levels of boredom. The differences was more marked in Western countries. The measuring instrument used, was the Boredom Proneness Scale – a 28 item self-report scale with true-false statements, e.g. “Time always seem to be passing slowly.” As an interesting aside, the original research interest was not concerned with sex differences, but the

gender effect was so strong, that the authors were forced to analyse their data on boredom proneness, separately for each group.

Perhaps, more pertinent for this thesis, is the data presented by Zuckerman et al.¹³⁵ which used students from England. In a comparative study, they examined ‘sensation seeking’ amongst American and English subjects. Boredom susceptibility was also a factor that featured in their study. Overall, males from both countries were more prone to boredom, but gender difference only reached statistical significance in the English sample. American males were less prone to boredom, than their English counterparts. Unlike the male subjects, female subjects on both sides of the Atlantic, were more stable in their scores of boredom proneness, with very similar mean scores. Other studies highlighting the predisposition of male subjects towards boredom are listed below; table 2.3.

TABLE 2.3 Boredom Susceptibility and Gender: Some recent studies.

Study *	Gender Most Prone To Boredom
Sundberg, N.D. et al. (1991)	Males
Tolor, A. (1989)	Males
Farmer, R. et al. (1986)	Males
Green, C. et al. (1983) [†]	No sex difference
Zuckerman, M. et al. (1978)	Males
Zuckerman, M. (1971)	Males

* See reference list 134-138 for bibliographic details.

[†] Ref. 121

All these studies purporting to show a male proneness to boredom, are based on questionnaire responses. Empirical data that comes from controlled and rigorous laboratory experimentation, are lacking in this respect. By way of explanation, it has been suggested that males are more likely to ‘inflate their abilities’ and consequently see their work as not sufficiently challenging, and thus inclined label it as ‘boring’¹³⁶. Despite our inadequate theories, it is nevertheless clear, that such gender effects are strong, and do warrant careful forethought in the planning and design of future experiments.

A strategy to be used in this experiment will be to lower arousal (via boredom induction) in an effort to provide a soporific situation capable of unmasking sleepiness. This will be carried out in the early afternoon (circadian trough) and early evening

(circadian peak). On the face of it, the supposed sensitivity and low boredom threshold of males, would ideally place them as experimental subjects. There is however, a problem with using male subjects. If males are so easily overwhelmed by boredom, then the circadian mechanisms controlling alertness might be rendered ineffective, in the face of such powerful benumbing effects of boredom. This sledgehammer effect in males, would not allow for the discrimination between the subtle sleepiness of the afternoon and the alert disposition of the early evening, which is likely to be quashed by oppressive boredom effects.

Our ability to make distinctions between circadian sleepiness, may be more profitable if we use female subjects, where the effects of boredom, while still apparent, are tempered and measured in scale. The effects of boredom will be manifest at points of circadian trough (eg. APS), yet not powerful enough to subjugate alertness during circadian peaks (eg. early evening). There are other grounds supporting the use of female subjects. It appears that much of our accumulated data on time-of-day effects has been gathered from male subjects. This can be traced to the pioneering work of the Applied Psychology Unit at Cambridge where naval ratings formed the subject pool (eg. Blake, 1967 and Colquhoun, 1971^{refs. 100, 107}) and persist even today (eg. Hill et al., 1991¹³⁹). The dearth of data on female subjects has also been alluded to by Baker et al..¹³² Thus, there exist good grounds for using female subjects, in the context of the experiment to be reported here.

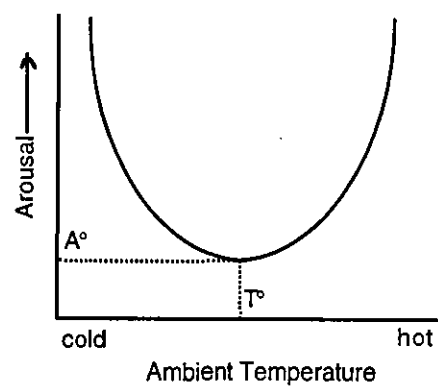
2.6.3 AMBIENT TEMPERATURE

Afternoon sleepiness, especially its cultural form, the siesta, is almost intuitively associated with sweltering heat. Under the oppressive tropical sun, all life becomes too languid for any exertion at midday – farmers desert their fields, and even chimpanzees retreat to their shaded daynests [see previous chapter]. But, it is not only agrarian societies that find heat soporific – dull committee meetings when held in warm rooms, requires almost Herculean effort to prevent members from nodding off⁷⁵. The boredom and the warmth may not *cause* the sleepiness, but, it is hypothesised, are central to the unmasking of any underlying sleepiness. From this, it follows that warm temperatures (like boredom, discussed earlier), must be able to lower arousal.

Environments which are too hot or too cold, can in fact elevate arousal, simply by the physiological stress that follows thermoregulation. Essentially, it is only mild warmth which is thought to be dearousing, and thus linked to sleepiness¹⁴⁰. For the purpose of unmasking the APS, the sleep inducing property of mild heat can be usefully exploited in experimental protocols, designed to lower arousal. This relationship between arousal and temperature is illustrated overleaf; figure 2.3.

Figure 2.3 Arousal versus Temperature

The point of thermoneutrality (ambience) should coincide with the point of lowest arousal as "sensory input to the nervous system should be at a minimum."
 In practice, arousal is at its lowest (A°) when the point T° , is fractionally in the direction of warmth. [see ref: 142]



However, the initial task for the researcher, is to gain an understanding of the thermal conditions deemed soporific. The industrial definition of thermal comfort (ASHRAE Comfort Standard 55-81) is outlined as, “that state of mind which expresses satisfaction with the thermal environment.” In practical terms, it refers to an environment perceived as neither cool nor warm (sensory thermoneutrality)¹⁴¹. Attempts to pin-point this prescriptive zone reflecting thermal comfort are numerous; but the most recent effort (Grivel and Candas, 1991)¹⁴¹ suggest the average comfortable temperature to be around 26.6°C.

Table 2.4 Ambient Temperature & Comfort [ref:141]

Study	Mean Comfort Temp. (°C)
Grivel & Candas (1991)	26.6
Grivel (1984)	25.3
Rohles (1974)	26.2
Rohles & Nevis (1971)	25.8
Fanger (1970)	25.6
Nevins et al (1966)	25.0

Determining an upper comfort zone that is warm (dearousing) and yet free from thermoregulatory stress (eg. sweating and evaporative heat loss) is difficult. We need to consider whether subjects are active or sedentary, the nature of task demands, clothing worn and the physiological characteristics of the subject¹⁴³. It is not unusual to find researchers using an upper comfort temperature of around 30°C — Holmberg and Wyon: (30°C); Mackworth: (30°C); Grether: (29.4°C); Reddy and Ramsey: (30°C)^{see 144}. Interestingly, in their investigations of ambient temperature and sleep, Muzet et al.¹⁴⁵ found the thermoneutrality *inside* the bed to be around 30°C, whilst the preferred

bedroom temperature was found to be 19°C. A review by Mc.Intyre¹⁴² concluded that mental and physical performance deteriorated markedly at temperatures above 33°C, whilst in the cold, manual dexterity fell when hand temperature dropped below 15°C.

It is important to stress that despite mild temperature changes in the zone of thermal neutrality, core temperature remains relatively stable. Any challenge to the core temperature (normally 37°C) can bring on physiological stress. Although, it is worth noting that core temperature does fluctuate in a diurnal fashion; lowest during midnight to early morning and highest during mid-afternoon to evening; a difference between the two extremes can reach 1°C.¹⁴⁴

Clearly, any study on afternoon sleepiness, must address the issue of environmental temperature, for reasons of scientific interest, as well as the unravelling of folklore beliefs connecting torrid heat with the siesta. Warm temperature appears to be a dearouser and therefore may be usefully employed to unmask circadian sleepiness. I intend to introduce temperature as an independent variable: with subjects placed in a warm room (29°C), and a cool room (18°C), at two focal points during the waking circadian phase (afternoon versus early evening). Body temperature will be constantly monitored; its stability will confirm that no physiological stress results from the ensuing thermal exposures, planned around the extremes of comfort.

2.6.4 Methodological Issues and Experimental Aims

Conducting 'sleep' studies during the normal 'waking' day is fraught with difficulties, in terms of measuring the very subtle changes to alertness. Vigilance and performance efficiencies are commonly used as indices of fluctuating alertness; and one of the most popular of these performance measures – in sleep research and in clinical drug studies – is reaction time tests (RT). Choosing the appropriate RT test and the frequency of testing requires careful forethought.

Type of Task

Performance tests, like RT measures, are designed to give us an indication of CNS functioning. Dinges has recently reported that simple RT tasks requiring sustained attention are far more sensitive to sleepiness than complex tasks where possible 'interest' may ameliorate any deficit¹⁰⁶. Wilkinson^{109, 315} drew attention to this important fact much earlier. Also, if the tests are too short in duration, then the subjects ability to muster sufficient zeal will vitiate the test's efficiency. Consequently, lengthy tests – like the Wilkinsons Auditory Vigilance Test (WAVT) – which may last 50-60 minutes, have acquired much popularity. However, the use such tests in studies of *diurnal* variation is questionable. Two principle shortfalls are outlined below:

1. Diurnal fluctuations – especially in the afternoon – are dynamic with frequent (sometimes, ultradian-like) oscillations. A lengthy monotonous test, lasting almost an hour, cannot be given frequently, and moreover, require an interim period of ‘recovery’ between tests. When tasks like the WAVT have been used in studies of diurnal variation, signal detection (d') was found to be relatively unaffected (Craig et al.¹⁹¹) – though there were changes to response criteria β .
2. During a lengthy test, boredom develops and psychological conflicts become apparent. The response criteria to stimuli become lax as motivation falls. A real-world example was provided by Millar¹⁹²: in a 20 minute task, it took 15 minutes for boredom to become overwhelming before impairment was registered [only] in the last 5 minutes of the testing session. A seemingly adequate task of sufficient duration was in effect reduced to a short 5-minute test. Similarly, Hamilton has pointed out that performance decrements are usually to be found in the latter portions of lengthy tests^{see 193}. There is thus the suggestion that boredom or some dearousing factor enters the testing scene midway. Instead of directly challenging CNS functioning, at the outset, the performance test *itself* is first used as a dearousing agent before decrements can be uncovered. Motivational conflicts and monotony, occur *during* the test. This inevitably compromises the efficacy of the test; whose purpose after all is not mood alteration, but one of psychophysics. The method of this APS experiment will be to lower arousal *outside* the testing situation, thereby maintaining the integrity of the test to directly measure the neurologic state, its sole prerogative. To summarise, lengthy tests like the WAVT are well suited to probe sustained attention, in paradigms like sleep deprivation, but are of doubtful value in diurnal studies where several tests are scattered over the course of a single day. These ‘single-day’ studies require testing at short intervals which makes tests like the WAVT impractical. For investigations during the normal waking day, a sensitive psychometric, capable of distinguishing subtle hourly oscillations is essential.

Frequency of Testing

Another contentious point concerns the frequency of testing. The transient APS has a very narrow time band, and the phenomenon can go unnoticed if the testing intervals are widely spaced in time. Blake’s five testing points were distributed at 150 and 330 minute intervals (the afternoon period contained only two tests, and the early evening, just one test). Testing intervals of 4 hours¹⁹³ and 5 hours¹⁹¹ are simply too sweeping to capture the minutæ of diurnal change. In an early review of studies into diurnal variations, Freeman and Hovland¹⁵² noted that the average testing intervals are about

120 minutes. I have decided on a 50-minute testing interval for this APS experiment as it is particularly well-suited to uncover this short-lived zone of vulnerability. In this APS experiment, subjects will also be under experimental ‘supervision’ for large parts of the diurnal day. In terms of sleepiness, the half hour between 3:00–3:30 pm. may turn out to be more soporific than the equivalent interval between 4:00–4:30 pm.; yet both periods fall into that part of the day cycle we call the ‘afternoon’.

It is quite disturbing to find studies with “Time-of-Day” in their title which administer only 2 tests (eg. Battaycharya et al.¹⁹⁴ and Folkard et al.¹⁹⁵). The full scope of diurnal change cannot be encompassed with just two widely separated tests – frequent assessment should be *de rigueur*. In choosing the frequency and temporal placement of these tests, there needs to be an underlying rationale with a clear hypothesis. One is justified in using two tests if these are designed to model specific ‘real-life events’ – eg. the effects of alcohol that is associated with lunch-time drinking versus the effects of a similar dosage during the early evening meal/recreation (see Horne et al.¹⁹⁶). These studies are directed at the effects of a specific pharmacologic agent administered at a specific time to mimic imbibition behaviours which are true to life.

Experimental Design

The merit of the between-groups design to be employed in this experiment, is that it avoids the confound of asymmetrical transfer (see Poulton¹⁴⁶ and Millar^{147, 148}). While addressing the problem of carry-over effects, this design does present the difficulty of between-subject variation. According to Millar, inter-subject variability can be easily averted if we express “performance as a change from pretreatment baseline”¹⁹⁷. It should be mentioned that there are dangers in using ‘difference scores’. In a recent homily, Webb¹⁴⁹ cautions on the use of difference scores — but, he regards their use as legitimate when testing for between-group differences through statistics such as ANOVA’s. These recommendations will be heeded in this experiment.

In sum, the aim of the first study will be to examine the easily masked afternoon propensity for sleepiness, by contrasting it with the early evening peak in alertness. The unmasking procedures designed to facilitate sleepiness will include boredom induction and warm room temperatures. To gauge the course of sleepiness, frequent measures will be taken of performance (eg. simple RT tests), subjective sleepiness, heart rate and body temperature.

2.7 EXPERIMENT

METHOD

SUBJECTS

Participants were forty-eight female volunteer subjects aged between 19-32 years. They were all in good physical health, and none of the subjects had a history of recent depression or any sleeping disorders. The subjects were undergraduates, postgraduates and librarians, drawn from the University of Loughborough. Subjects were asked to volunteer in pairs, with an acquaintance.

DESIGN

The subjects were divided into two sets:

- a). 24 S's for the afternoon study.
- b). 24 S's for the evening study.

Experimental treatment was identical for both sets. The twenty-four S's in each set were randomly assigned to one of four groups, each consisting of six subjects. The four experimental conditions were:

- 1. Warm room with low levels of interest..... [boring]
- 2. Warm room with high levels of interest..... [interesting]
- 3. Cool room with low levels of interest..... [boring]
- 4. Cool room with high levels of interest..... [interesting]

TABLE 2.5 Four experimental conditions (temperature vs. interest) at 2 points of the day

AFTERNOON			EVENING		
	Warm Room	Cool Room		Warm Room	Cool Room
Boring	6 Subjects	6 Subjects	Boring	6 Subjects	6 Subjects
Interesting	6 Subjects	6 Subjects	Interesting	6 Subjects	6 Subjects

The major independent variables included:

- (a). Room Temperature — 2 levels (Warm vs. Cool)
- (b). Subject Interest — 2 levels (Interesting vs. Boring)
- (c). Time of Day — 2 levels (Afternoon vs. Evening)

See table 2.6 overleaf.

This between-groups design was chosen to avoid the confound of asymmetrical

transfer (or carryover effects) as highlighted by Poulton¹⁴⁶ and Millar^{147, 148}.

Table 2.6 The Major Independent Variables.

ACTIVITY	Interesting	EXAMPLE: READING MATTER: Popular mags.- Cosmopolitan, Elle, Nat. Geographic, Woman, Punch, Vogue, etc... BOARD GAMES: Chess, etc. VIDEOS: Pop Videos, Films, Documentaries, etc...	
	Boring	EXAMPLE: READING MATTER: Esoteric Mags- Stamping Collector, Fly Fishing, FT Index, Fluid Mixing Technology, etc... SIMPLE GAME, VIDEOS: Open Univ. [Maths], Engineering.....	
TEMP.	Warm	29.0°C (±0.5°C)	50% Humidity
	Cool	18.0°C (±0.5°C)	50% Humidity
TIME OF DAY	Afternoon	12.00 noon to 16.00 h.	
	Evening	18.00 h to 22.00 h.	

MATERIALS

a). **Questionnaires** — All these questionnaires are reproduced in full in the Appendix.

1. Stanford Sleepiness Scale (SSS)

The SSS (developed Hoddes et al., 1973)³¹⁷ is one of the most commonly used self-rating sleepiness scales. The scale consists of seven ranked statements from:

- ☐ 1. Active, Vital, Alert, and Wide Awake
- ↓
- ☐ 7. Almost Unable To Stay Awake, Struggling To Remain Awake.

Subjects are asked to place a tick 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 15 minutes.

2. Visual Analogue Scale (VAS).

A 100 mm visual analogue scale was used to gauge sleepiness³¹⁶. Tagged to one end of the line are the words “Wide Awake”; the other side carried the words “Very Sleepy”. Subjects were required to draw a vertical line at a point along this continuum; a line towards the left indicated, alertness whilst a line towards the right reflected sleepiness.

Wide Awake—————Very Sleepy

3. Leathwood Mood Questionnaire

Developed by Leathwood and Pollet (1982)³¹⁸ and chosen here for its simplicity, heterogenous probing and brevity with respect to time needed for completion. In essence, it probed 5 aspects of subjective sensations:

- | | | |
|---------------------------|--------------------|---------------------|
| a. Sleepiness/Wakefulness | c. Lethargy/Vigour | e. Boredom/Interest |
| b. Affective State | d. Hunger/Satiety | |

4. Akerstedt Drowsiness Scale (ADS).

This scale (after Akerstedt^{see 320}) is a variant of the SSS; instead of 7 scalar points, it has 9. The descriptors at each point are simplified, and are only attached to alternate scale points, with intervening points left blank to represent an intermediate response category – this is illustrated below; with the full version reproduced in the appendix.

- ☐ 1. Extremely Alert
- ☐ 2.
- ☐ 3. Alert
- etc . . .

5. Post-Sleep Questionnaires (*Morning of the Experiment*)

- This determined:
- a. Sleep onset and wake-up times.
 - b. Sleep quality (5 point scale: 1=much better than normal, 2=better than normal, 3=normal, 4=worse than normal, 5=much worse than normal).
 - c. Difficulty in getting up. (5 point scale: 1=very easy, 2=easy, 3=moderate, 4=difficult, 5=very difficult).
 - d. Sleepiness after 30 minutes (using the SSS).

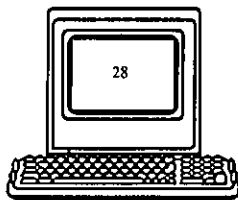
b). Apparatus

i). Simple Reaction Time

BBC microcomputer (with secondary co-processor).

Customised RT software[© NCS. Smith, Loughborough Univ.].

Description: A running centisecond digital clock close to the centre of the screen is frozen by presing the spacebar of the keyboard. Essentially, digits of approximately 12mm. in height, appear in the centre of the screen, and upon 'firing' will run incrementally from, 00—01—02....12...60...150..etc (100=1 sec.) until frozen. A response on the spacebar will freeze the running clock - the speed of responding to the running clock is the RT. The next stimulus will occur after a random time delay. The inter-stimulus presentation rate was set between 1–8 seconds. This means that stimuli (the running digits) could begin at any time between 1 second and a maximum of 8 seconds – the average stimulus would typically occur after 4 seconds. Premature triggering of the frozen clock is recorded as an error.



*Screen with running
clock in the centre*

Operational Setting

Maximum inter-stimulus delay..... 8 seconds

Mean inter-stimulus delay 4 seconds

Total Test Time 10 minutes

The choice of a 10-minute RT test duration was much influenced by the success of Lisper and Kjellberg³¹⁹. A review by Dinges and Kribbs, has pointed out that recent work by Wilkinson acknowledges the sensitivity of short duration RT tests (Tilley and Wilkinson 1984; Wilkinson and Houghton, 1982; Glenville et al., 1978)^{see 106}.

A problem with many performance tasks is the confound of learning or practice effects: — according to Dinges et al., practice effects “..occur for virtually every cognitive task from mental arithmetic to logical reasoning to low signal rate vigilance tasks—a notable exception is the simple RT tasks” (emphasis mine). The simplicity of the task requires no training and the attainment of a ‘performance asymptote’ is a probably insubstantial, given the short practice effect. Should practice effects arise, then by the very nature of the current experimental design, the effects would be constant across all the conditions.

This simple visual RT test did present performance feedback (akin to Glenville et al. 1978, Wilkinson and Houghton, 1982) but according data from Dinges, it does not affect the sensitivity of the short RT test^{see 106}. Wilkinson has been the foremost scientist to develop and give theoretical understanding to these RT tests^{315, 328}.

ii). Heart Rate Monitor

The heart rate (beats per minute) was measured throughout the experiment at 1-minute intervals using the Sports Tester™ PE 3000 (Polar Electro KY, Kempele, Finland). This is a wireless device – a belt with a transmitter is worn around the chest, and pulse data is transmitted to a receiver unit strapped around the wrist.

iii) The Grant Squirrel Data Logger and Temperature Sensitive Ear Thermistor

Aural temperature was recorded continuously with a temperature sensitive probe placed in the ear. Readings were taken as one-minute averages, and the data stored in a Grant Squirrel Meter/Logger (model SQ 32-4U; Grant Instruments, Cambridge, England).

iv). Environmental Chamber

The climatic chamber was a medium sized room (3m x 4m x 3m) capable of producing a fairly accurate and controlled range of air temperatures and humidities.

c). Material For Arousal Manipulation

- (i). Videos.
- (ii). Books.
- (iii). Popular and 'specialised' esoteric magazines.
- (iv). A selection of popular boardgames & puzzles.

— For full details of the materials used, see Appendix —

d). Other Materials

- (i). Netelast [G] – Elasticated Surgical Net.
Trunk size (O.S. Adult); [Penn International].
- (ii). Circular cotton wool pads (9cm. diameter).
- (iii). Soft circular sponge (size 2. synthetic)
[Boots, Nottingham].
- (iv). Micropore® Surgical Tape [3M Ltd.].

e). Food

The Menu

Energy Value : 650 kcal (2740 kj)

- (i). Baked Potato – (medium size : approx. 100g).
- (ii). Grated Cheddar Cheese – (50g)
- (iii). Salad – Fresh vegetables:
(tomatoes, cucumber, lettuce and radish).
- (iv). Fruit Yogurt – 1 carton.
- (v). Brown Bread – 1 slice.
- (vi). Butter – 1 pat (12g).

PROCEDURE

The exact nature of the study was concealed from all the participants, though on ethical grounds, subjects were fully informed about the apparatus (the monitoring equipment and the climatic chamber). They were also shown copies of the standardised lunch-menu, in case they were not able to consume the meal for medical, moral or any other reason. For each experimental trial, subjects were run in pairs. All subjects were encouraged to participate with an acquaintance – a strategy designed to reduce anxiety and to promote a relaxed ambience during the lengthy confinement in the climatic chamber. Those subjects accepted for the study were given written details of their role in the experiment (without compromising the covert experimental aims), and a date was set for their attendance. All subjects were required to visit the laboratory the day before the experiment in order to familiarise themselves with the RT apparatus (one full 10-minute test), and to receive further instructions. Essentially, these were a set of rules which the subject had to adhere to during the hours preceeding the experiment. These are outlined below:

-
-
1. Tea or coffee drinking was prohibited 4 hours prior to the experiment.
 2. No strenuous physical exertion or sport to be engaged in 3 hours before arriving at the laboratory.
 3. Subjects had to complete a Post-Sleep Questionnaire soon after rising, on the morning of the experimental day.
 4. They were issued with a digital thermometer and asked to record their temperature upon awakening and thereafter at half-hourly intervals. A similar half-hourly log was also kept of their physical activity, mental activity and food intake.
 5. For the experiment, subjects' outergarments were restricted to the following attire: jeans or trousers; blouse or shirt; and a light-weight sweater. The insulation derived from clothing is expressed as clo units. The clothing prescribed above was estimated as 0.9 ± 0.1 clo — for a resting subject in a ventilated room of 21°C and 50% humidity, 1.0 clo provides satisfactory insulation (ASHRAE Standard 55-81).
-

Experimental Day

<u>[12 Noon]</u>	Subject-pairs were met by the experimenter and escorted to
<u>[18.00 h]</u>	lunch/dinner at a near-by catering facility. All food was consumed.

[12.20–12.25] At the laboratory, instructions were given to the subjects on the
[18.20–18.25] attachment of the Sports Tester™ (pulse monitor). They then went into individual private cubicles and attached the device themselves.

[12.25–12.35] Subjects were fitted with a temperature sensitive ear thermistor
[18.25–18.35] embedded in an ear-mould to fit snugly. The wires leading from the probe were secured (with surgical tape) to the bony structure behind the ear-lobe. This temperature sensitive probe was insulated from the effects of the external environment. This was achieved by placing a thin circular cotton-wool pad over the ear, on to which an additional soft sponge was placed, to augment the insulation. To hold the cotton wool and sponge in position, over the ear, an elasticated gauze netting was stretched over the head, thus securing the entire ear padding. This netting was then trimmed to give a balaclava-like head dress. On the basis of earlier practise trials, it was found that such a protuberant ear padding applied to just one side of the head resulted in an unequal distribution of weight, causing mild discomfort. This problem was overcome by simply placing an identical ear-padding to the other side of the head, thus restoring equilibrium.

Baseline Session

[12.35–12.50] Subjects were given a battery of sleepiness/mood questionnaires, a
[18.35–18.50] pencil/paper checking task, and a test of reaction time. The reaction time test was conducted on a BBC computer, using a customised software package (see Apparatus). In essence, a running centisecond digital clock in the centre of the screen is frozen by pressing the spacebar. The time taken to freeze the clock gives a measure of the RT. This test was specially designed to be as simple as possible (unlike auditory tests eg. WAVT; where tonal discriminations requires much practice). The simplicity of this RT task (responding to digit movement) needed little or no practice — nevertheless a 1 minute ‘familiarisation’ session was given prior to the full 10-minute test.

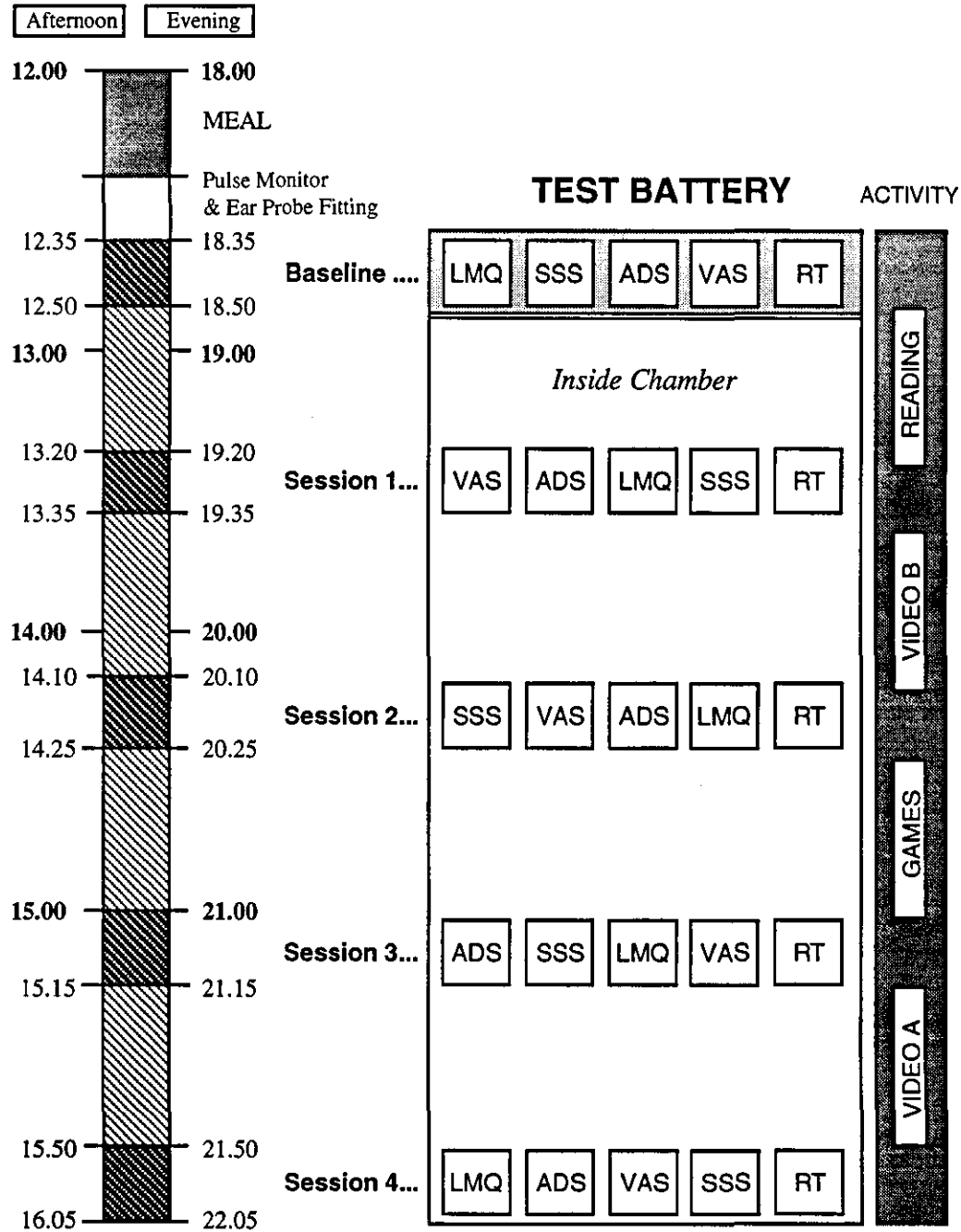
In summary, each test session comprised of the following tasks:

1. Stanford Sleepiness Scale.....[SSS]
2. Akerstedt Drowsiness Scale..... [ADS]
3. Leathwood Mood Questionnaire..... [LMQ]
4. Visual Analogue Scale - *Sleepiness*..... [VAS]
5. Reaction Time Test[RT]



These baseline tests were done in a room, outside the climatic chamber.

Fig. 2.4

EXPERIMENTAL SCHEDULE



Key

-  = Testing Period
-  = Activity Period (boring or interesting)

- RT = ReactionTime
- LMQ = Leathwood Mood Questionnaire
- ADS = Akerstedt Drowsiness Scale
- SSS = Stanford Sleepiness Scale
- VAS = Visual Analogue Scale (sleepiness)

Session One

[12.50–1.20] After completing these baseline measures, subjects were escorted into the climatic chamber and seated in easy chairs. A small table was placed between subjects upon which reading material was to be found. The connecting leads of the temperature sensitive tympanic probes were plugged into the battery operated Grant Squirrel Data Logger and the recording of aural temperature at 1-minute epochs commenced (recordings are taken every second and averaged for each minute).

[18.50–19.20]

Depending upon whether subjects were in an ‘interesting’ or ‘boring’ condition, instructions varied. Subjects in the ‘interesting’ condition were encouraged to ‘chat’ to each other throughout the afternoon. Those in the ‘boring’ condition were discouraged from speaking to each other. In all cases, napping was not permitted. The experimenter sat in a far corner engaged in ‘private work’, though fully attentive of the subjects’ actions.

Once seated, subjects were presented with one of the ‘leisure’ choices. The range of the activities are listed below – also see Appendix.

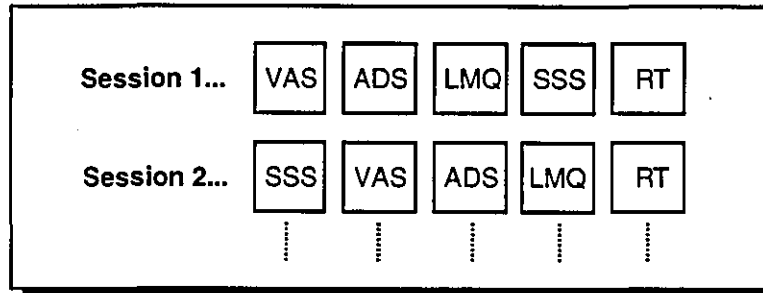
Table 2.7 Leisure items intended to influence arousal

Interesting Condition	Boring Condition
Video A [comedy]	Video A [Open Univ. Maths]
Board Games [eg. scrabble]	Reading [esoteric books & mags]
Video B [documentary]	Game [simple game]
Reading [popular mags]	Video B [technical lectures]

During the course of the experiment, the subjects engaged in all of the ‘leisure activities’ prescribed for their group. The order in which these activities were presented, was varied between experimental trials as a counterbalancing measure. So, whilst one pair might start their session viewing a video tape, another pair might begin by reading magazines.

At 13.20 h (*afternoon group*) and 19.20 h (*evening group*) subjects were given their first battery of tests *inside* the chamber - ‘Session 1’. These tests were identical to the first batch (baseline), but here again, the order in which the tests were presented was deliberately varied as a

counterbalancing strategy. At each of the ensuing test sessions, the order of test presentation was always varied (see below).



In all cases, the RT test was always presented last. This was deemed necessary as this test did elicit negative mood reactions; it was considered mentally fatiguing by some subjects. As such it would have influenced responses to the mood and sleepiness questionnaires had they been executed after the RT test.

Session Two

- [13.35–14.10] During this period subjects were offered a different ‘leisure activity’.
- [19.35–20.10] At 14.10 h.(*afternoon group*) and 20.10 h.(*evening group*) subjects were presented with their second battery of tests inside the chamber. This was Session Two.

Session Three

- [14.25–15.00] Another ‘activity’ followed by testing for Session Three at 15.00 h.
- [20.25–21.00] or 21.00 h.

Session Four

- [15.15–15.50] A final ‘activity’ followed by testing for Session four at 15.50/21.50 h.
- [21.15–21.50]

The End

- [16.05] The completion of this test battery would bring to end the experimental session. Subjects were requested to continue taking their oral temperature and to log their activity/food intake until they retire in the evening. They were also issued with another Pre-Sleep / Post-Sleep Questionnaire.
- [22.05]

Measuring Aural Temperature



Temperature sensitive ear thermistors embedded in ear-moulds to fit snugly.



Insulating sponge placed over ear probes (held in place by netting; see photo below)

Reaction Time Measures



Computerised visual reaction time test : inside the climatic chamber

2.8

Results

The results of all the tests were compared to the baseline scores (conducted outside the climatic chamber). In most cases the data was subjected to analysis of variance with;
3 Between Group Factors: a). Room Temperature b). Subject Interest c). Time of Day
1 Within-Subject Factor: The 4 Post-Baseline Test Sessions.

Reaction Time (RT).

RT Main Effects:

Time of Day Baseline RT measures taken immediately before entering the climatic chamber and experimental manipulation, showed that the 24 afternoon and 24 evening subjects to have near identical RT scores at the outset. This was confirmed by a two tail t-test which found no significant differences between the two sets; see table below.

Table 2.8. Baseline RT Scores (mean & se)

Afternoon Group Mean [N=24]	Evening Group Mean [N=24]
0.299 secs (.006)	0.302 secs (.007)
$t = -.321$ [df. 46] $p = .750$	

However, once in the chamber, and exposed to the experimental manipulations the two groups reacted differently. The afternoon group were markedly impaired, with a significantly longer RT compared to the evening group; $F = 6.72$ [1,40], $p = 0.012$.

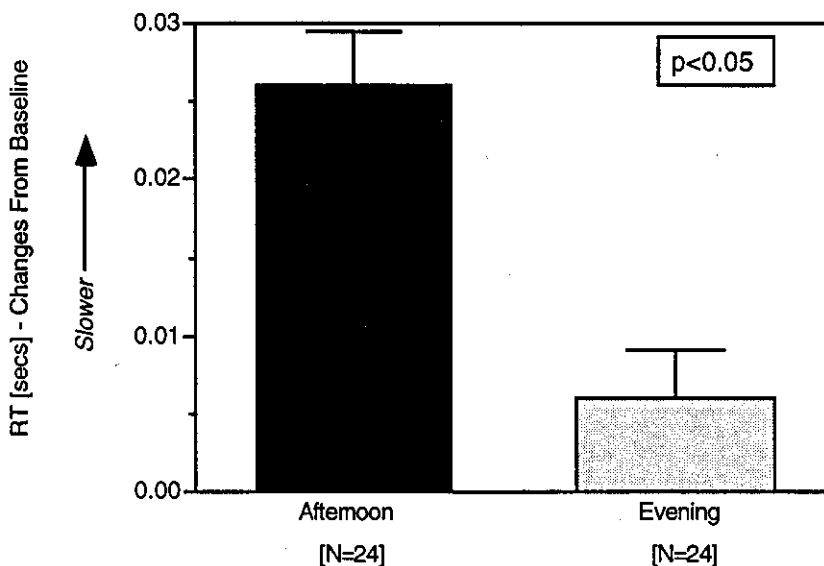


Fig. 2.5 Reaction Time and Time of Day

Subject Interest

Significant main effects were also found for 'Subject Interest' (boring vs. interesting), $F=4.35$ [1,40]; $p<0.05$. Subjects in the boring environment had a significantly longer RT compared to those in the 'interesting' condition; see figure 2.6 below.

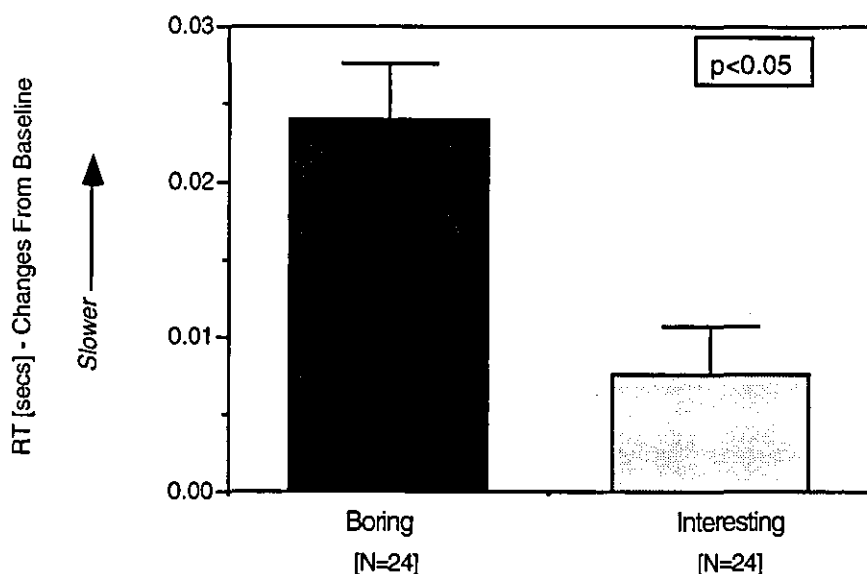


Fig. 2.6 Reaction Time and Subject Interest

Temperature

Environmental temperature produced no significant main effects or interactions. The effects of the climatic chamber (warm vs. cool) on the RT scores were thus minimal, as the table below illustrates.

Table 2.9. Effects of Temp. on RT – Changes from baseline (\bar{x} & se)

WARM [change from baseline]	COOL [change from baseline]
+0.016 secs (.004)	+0.015 secs (.003)

The significant RT scores were therefore mainly attributed to the manipulations of mental arousal, i.e. boredom versus interest, and time-of-day factors.

Within-Factor : Changes from Test Session 1-4

Each subject performed the RT test (inside the chamber) at four separate intervals; so constituting a repeated measure. This factor was significant, $F=2.92$ [3,120]; $p<0.05$., indicating that the RT scores differed significantly between sessions (see figure 2.7). As the experiment progressed, subjects RT scores worsened from session to session. However, in the last test, there was a sudden improvement .

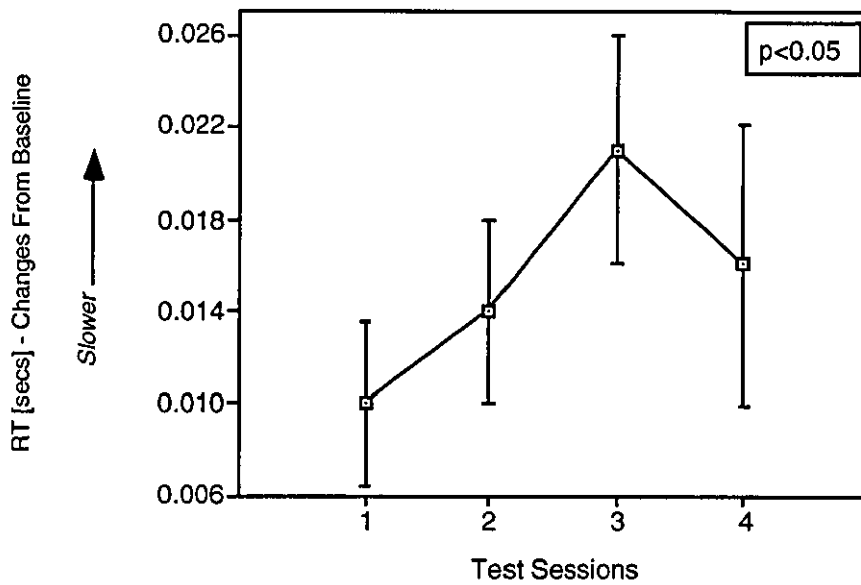


Fig. 2.7 Mean RT Changes Over Sessions (with se)

In a separate analysis, the scores from this last session were removed. No major differences were found to the resulting statistics by the exclusion of this last test session

RT Interactions

Although the interaction of 'Time of Day' x 'Test Sessions' was not significant, ($F=0.93$ [3,120] $P=0.43$) it is instructive to note that the sharp decline in RT between session 2 and 3, and the marked improvement on the last test, was particularly pronounced for the afternoon group.

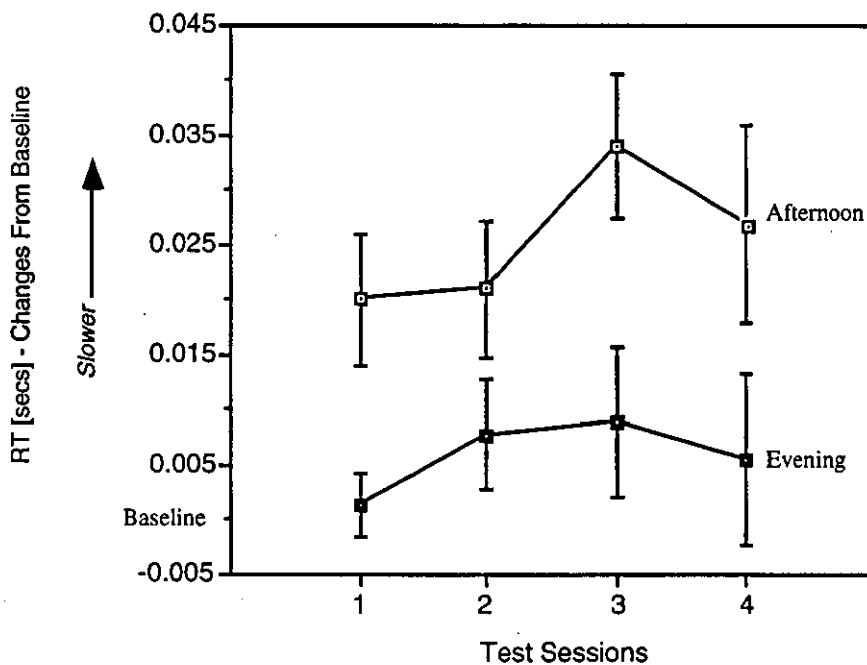


Fig. 2.8 RT - Time of Day x Test Sessions (mean & se)

BOX 1

A follow-up to the 'Time of Day' x 'Test Session' Interaction

The afternoon dip at Session 3 (1500h) can be further explored. If the transient APS does indeed lie at test session 3 (1500h) as fig. 2.8 suggests, then the two tests lying at either side of the dip (test session 2 and test session 4) can be treated as points reflective of normality. Averaging these two scores (putative baseline measures) and subtracting this score from test session 3 will give an indication of the change from normality, ie. a measure of the extent of the afternoon dip.

In order to see whether or not the dip at test session 3 is peculiar only to the afternoon, we need to do a similar analysis for the evening [control] group. A direct comparison between the changes occurring at test 3 in the afternoon and test 3 in the evening is then possible. This can be achieved by subtracting the evening scores from the afternoon scores followed by a 'one-sample' t-test on the resultant 'difference scores'. If the null hypothesis applies (ie. the size of afternoon dip at test 3 equal evening dip at test 3) then the mean of the 'difference scores' should equal zero. A one sample t-test on these 'difference scores' will establish whether or not the changes occurring at test session 3 are significantly different between the afternoon and evening groups.

$$\begin{array}{ccc}
 \text{Afternoon} & & \text{Evening} \\
 \text{Test 3 (1500h)} - \left(\frac{\text{Aft. Test 2} + \text{Aft. Test 4}}{2} \right) & - & \text{Test 3 (2100h)} - \left(\frac{\text{Eve. Test 2} + \text{Eve. Test 4}}{2} \right) = \begin{array}{c} x_1 \\ x_2 \\ \vdots \\ x_{24} \end{array} \\
 & & \uparrow \\
 & & \text{one sample t-test}
 \end{array}$$

When the procedure outlined in Box 1 above was applied to the RT interaction 'Time of Day' x 'Test Session', a 2-tail t-test failed to reveal any significant differences between the afternoon changes at test 3 and the evening changes at test 3 ($t=1.71$ [23]; $p=.101$). However, there are *a priori* reasons to suggest that 1500h represent the peak time for afternoon sleepiness (see pages 98–99), and conducting a 1-tail t-test; justifiable on the grounds of past data and theory; yields a significant result ($p=0.05$), indicative of a marked time-of-day effect with afternoon impairment maximal at test 3 (1500h).

Subject Interest x Time of Day Interaction.

The interaction between 'Subject Interest' x 'Time of Day' was significant $F=3.80$ [1,40]; $p=0.05$. This finding would indicate the negative effects of boredom are particularly striking during the afternoon as against the evening.

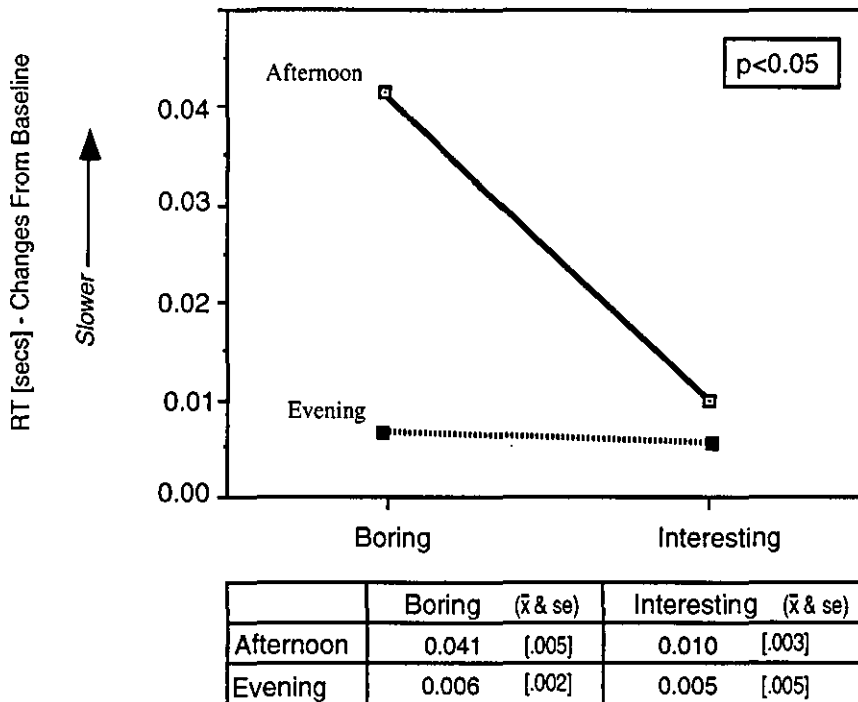


Fig. 2.9 RT – Subject Interest x Time of Day Interaction

Stanford Sleepiness Scale (SSS)

Baseline Comparisons

The sleepiness ratings of the 24 afternoon subjects, were compared with their evening counterparts, prior to experimental manipulation (i.e. baseline tests). From the table below, it is clear that both groups began the experiment with virtually the same levels of subjective alertness (in an SSS range of 1—7, they chose a score between 2 and 3).

Table 2.10 Baseline SSS Scores (mean & se)

Afternoon Group Mean [N=24]	Evening Group Mean [N=24]
2.6 (.16)	2.7 (.20)
$t = -.333$ [df. 46] $p=.741$	

SSS

2 = Functioning at a high level, not at peak but able to concentrate

3 = Relaxed, awake, not at full alertness, responsive.

SSS Main Effects

Significant main effects were found for ‘Subject Interest’, $F=11.29 [1,40]; p<0.01$. Subjects in the boredom condition reported greater levels of sleepiness.

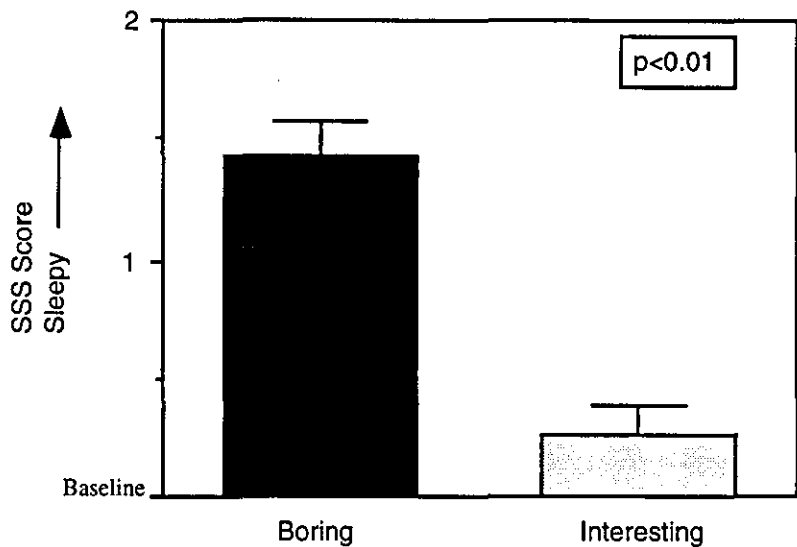


Fig. 2.10 SSS – Subject Interest: Change From Baseline (mean & se)

No significant differences were found for the main effect ‘Time of Day’ or ‘Room Temperature’.

Within-Factor : Changes From Sessions 1—4

The one repeated measure (changes between test session 1 to 4) showed significance, $F=16.84 [3,20]; p<0.001$. Sleepiness develops progressively with time in chamber.

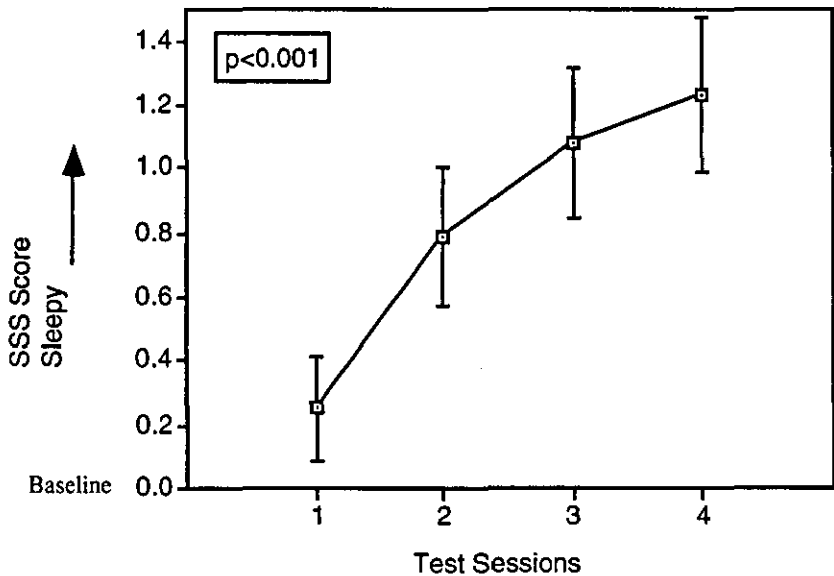


Fig. 2.11 SSS – Mean Change Between Test Sessions (with se)

SSS – Interactions

A significant interaction between ‘Subject Interest x ‘Test Sessions’ highlights the soporific effects of boring stimuli ($F=3.87$ [3,120]; $p<0.05$), which becomes potent with time, before levelling off. These effects are dampened in the ‘interesting’ group.

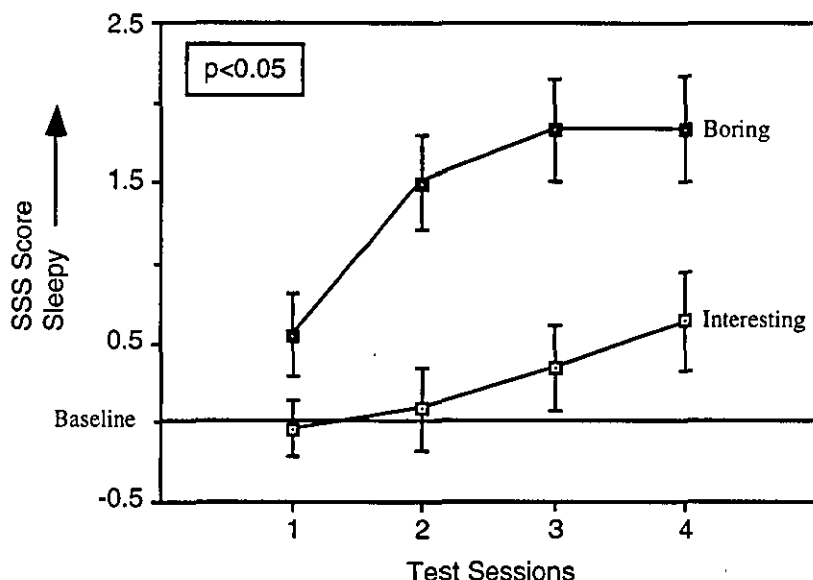


Fig. 2.12 SSS – Subject Interest x Test Sessions (with se)

An interaction between ‘Time of Day’ x ‘Test Sessions’ was significant, $F=3.35$ [3,120]; $p<0.05$. The sleepiness ratings between the afternoon and evening groups followed a similar path. However, this close alliance broke down at test session 3, where the two groups diverged markedly.

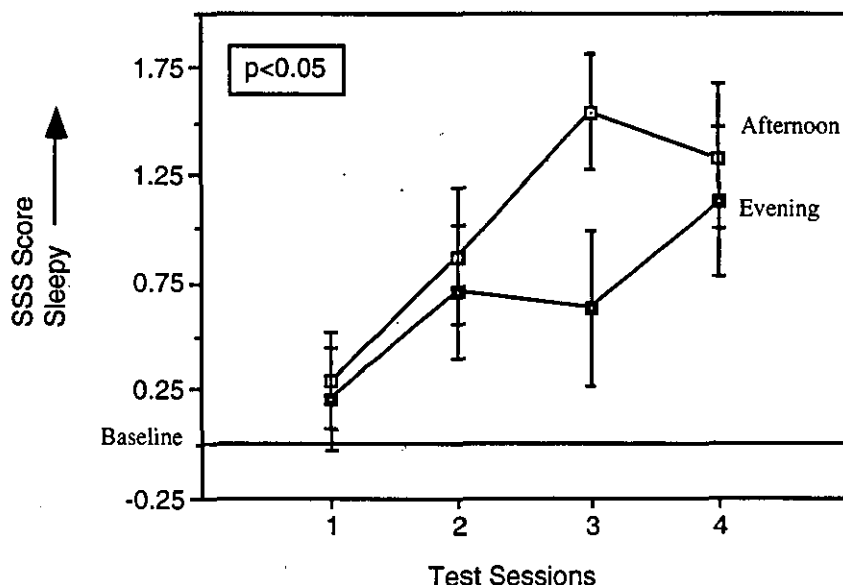


Fig. 2.13 SSS – Time of Day x Test Sessions (with se)

A follow-up to this interaction (Time of Day x Test Sessions) was carried out in the manner detailed in Box 1 (see page 72). The afternoon changes occurring at test 3 were significantly different from the evening changes at test 3 (two-tail $t=3.08$ (23); $p<.01$).

Akerstedt Drowsiness Scale (ADS)

Baseline Comparisons

The baseline sleepiness ratings of the afternoon and evening groups were very similar: on a 9-point ADS scale, both groups had a mean score of around 4 (see below).

Table 2.14. Baseline ADS Scores (mean & se)

Afternoon Group Mean [N=24]	Evening Group Mean [N=24]
4.29 (.30)	4.21 (.30)
$t = .197$ [df. 46] $p=.845$ [NS]	

ADS

4 = Intermediate between Alert (3) and Neutral (5).

ADS Main Effects

Significant main effects were found for 'Subject Interest', $F=11.75$ [1,35]; $p<0.01$. The 'boredom induced' group reported higher levels of sleepiness as shown below.

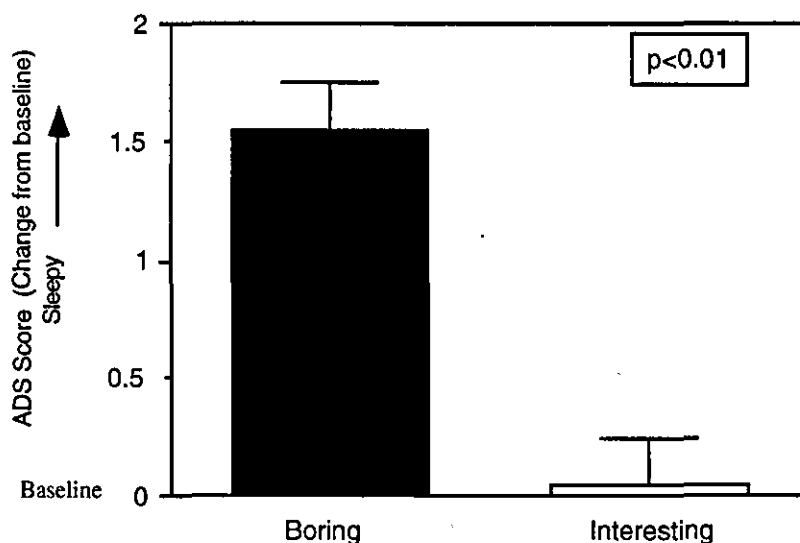


Fig. 2.14 ADS - Subject Interest: Change from baseline (mean & se)

For the main effects 'Time of Day' and 'Room Temperature', no significant effects were found.

Within-Factor : Sessions 1–4

The sleepiness ratings of subjects, varied significantly between test sessions; subjects became more sleepy with time; $F=6.35$ [3,105]; $p<0.01$.

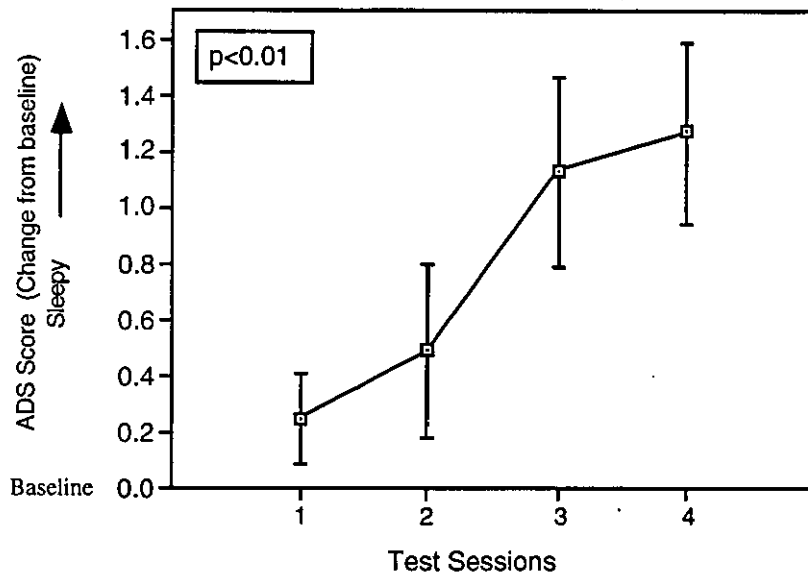


Fig. 2.15 ADS – Mean Change Between Test Sessions (with se)

ADS – Interactions

Using the ADS questionnaire, no significant interactions were found. However, 'Subject Interest' x Test Sessions approached significance; $F=2.51$ [3,105]; $p=0.06$. For the purposes of gauging trends, a plot of this interaction provides useful insight (figure 2.16). Likewise, the interaction between 'Time of Day' x Test Sessions ($F=2.37$ [3,105]; $p=0.070$) is also be plotted for illustrative purposes; see overleaf.

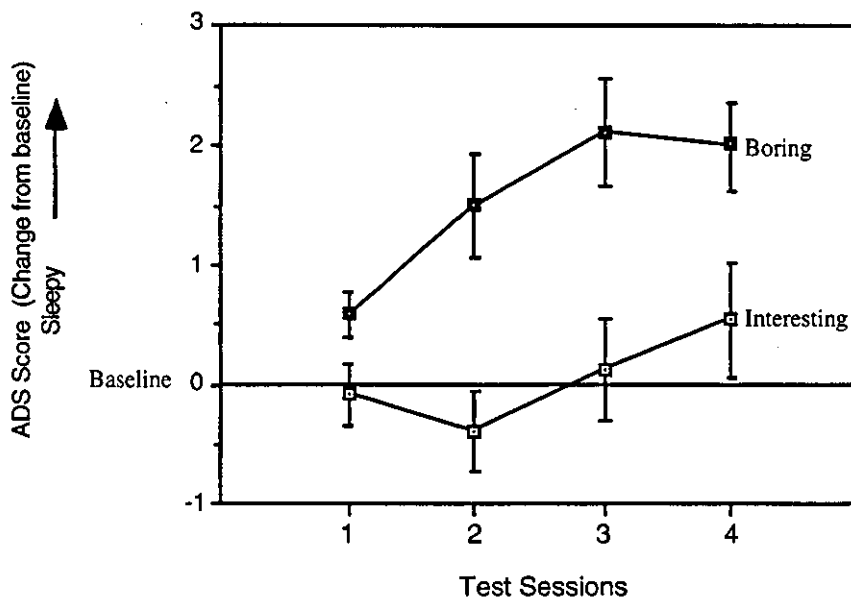


Fig. 2.16 ADS – Subject Interest x Test Sessions (with se)

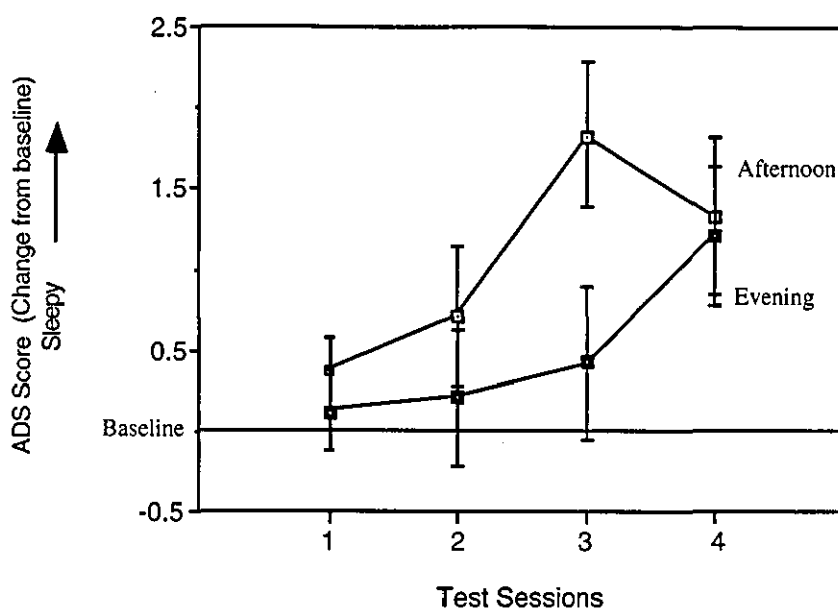


Fig. 2.17 ADS - Time of Day x Test Sessions (with se)

A post-hoc analysis of this interaction (Time of Day x Test Sessions), based on the procedures outlined in Box 1 (see page 72), revealed that the afternoon changes at test 3 (ie. 1500h) were significantly different to any changes occurring during the evening, at the equivalent test 3 juncture (two-tailed t-test: $t=3.67$ (23); $p<0.01$).

Visual Analogue Scale (VAS)

Baseline Comparisons

Initial sleepiness ratings (baseline) on a 100 mm visual analogue scale produced no significant differences between the two sets of afternoon/evening subjects.

Table 2.12. Baseline VAS Scores (mean & se)

Afternoon Group Mean [N=24]	Evening Group Mean [N=24]
38.52 (4.6)	33.42 (4.2)
$t = .816$ [df. 45] $p=.419$ [NS]	

VAS Main Effects

Subjects experiencing the 'boring' condition reported significantly more sleepiness than their counterparts in the 'interesting' condition; $F=11.14$ [1,34]; $p<0.01$ (see fig 2.18 overleaf). The main effects of 'Room Temperature' and 'Time of Day' did not produce statistically significant results.

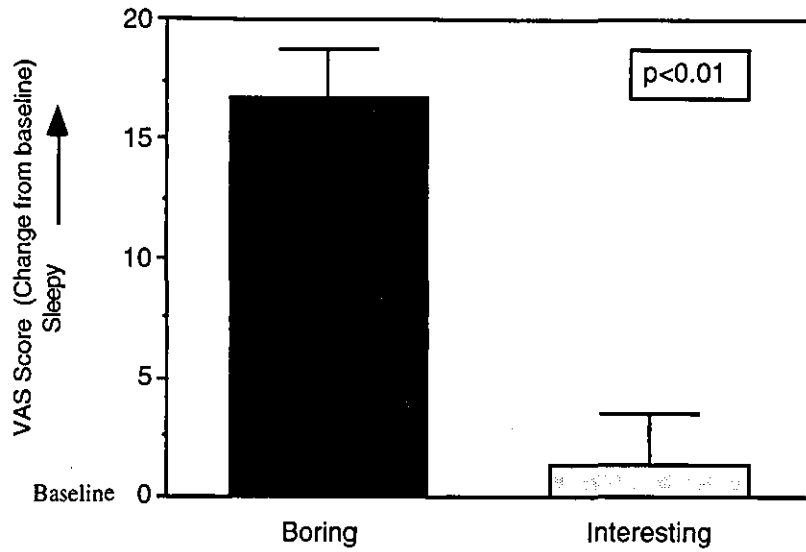


Fig. 2.18 VAS – Subject Interest : Change from Baseline (mean & se)

Within-Factor : Sessions 1—4

Significant differences were found between the test sessions; $F=9.57 [3,102]$; $p<0.001$

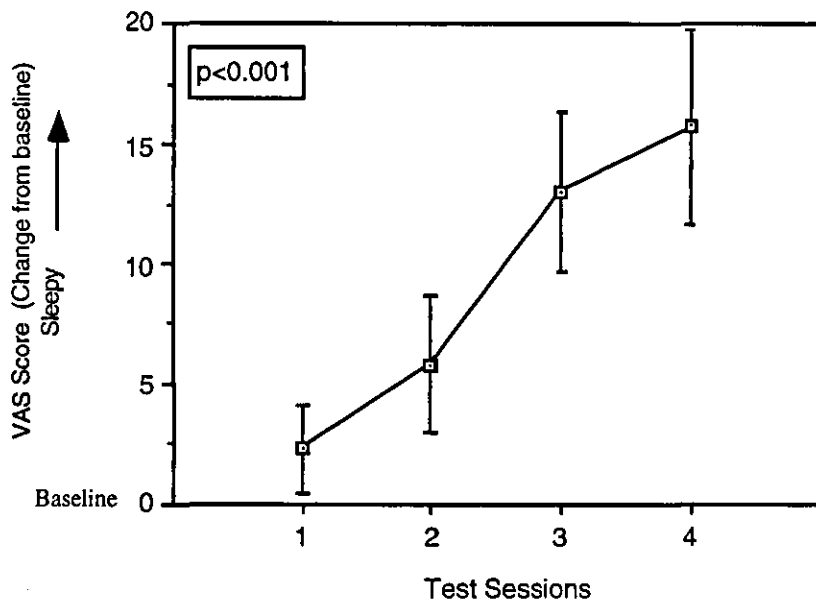


Fig. 2.19 VAS – Mean Change Between Test Sessions (with se)

VAS – Interactions

A significant between ‘Subject Interest’ x ‘Time of Day’ x ‘Test Sessions’ was found, $F=3.182$ [3,102]; $p<0.05$ (see below).

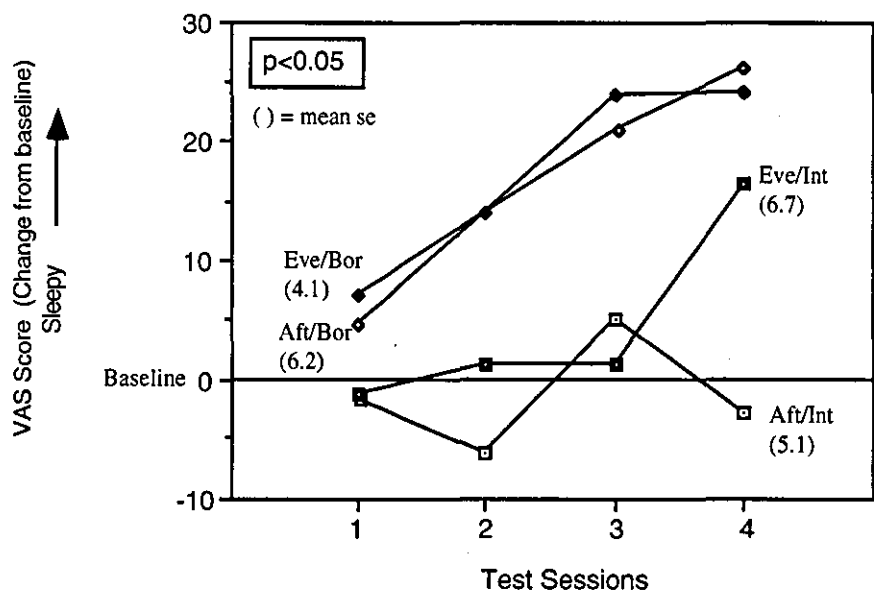


Fig. 2.20 VAS – Subject Interest x Time of Day x Test Sessions

This complex 3-way interaction indicates that subjects in the boredom group show a steady increase in sleepiness from session to session – this was true for both the afternoon and evening groups. Subjects in the ‘interesting’ condition were more variable in their ratings of sleepiness. In particular, the afternoon group showed a marked rise in sleepiness on the third session, with a reversal towards alertness, on the last session, whilst the evening group by contrast, diverged in the opposite direction on the last test session.

Leathwood Mood Questionnaire (LMQ)

The Leathwood Mood Questionnaire highlighted significant changes occurring in subjective ‘mood states’.

LMQ Main Effects

For the main effect of ‘Room Temperature’, subjects in the warm condition reported feelings of ‘lethargy’ ($F=5.79$ [1,40]; $p<0.05$), and ‘tiredness’ ($F=4.35$ [1,40]; $p<0.05$). Relative to baseline, cool conditions made subjects feel more ‘energetic’.

The main effect of 'Subject Interest' produced significant changes. Generally, under conditions of boredom, subjects reported feeling:

- a. Sleepy.....F=9.43 [1,40] p<0.01
- b. Lethargic.....F=7.97 [1,40] p<0.05
- c. Bored.....F=49.58 [1,40] p<0.0001
- d. Tired.....F=4.79 [1,40] p<0.05
- e. Unimaginative.....F=7.16 [1,40] p<0.05
- f. Inefficient.....F=4.73 [1,40] p<0.05

The only significant finding for the main effect Time of Day concerned the sensation of 'hunger': evening subjects reported greater feelings of hunger; F=5.09 [1,40], p<0.05.

Within Factor: Sessions 1—4

For the repeated measure (changes between test sessions 1 to 4), mood was found to be significantly altered in a negative direction as the experiment progressed. For example, as time continued, subjects felt increasingly:

- a. Lethargic.....F=5.96 [3,120]p<0.01
- b. Tired.....F=10.63 [3,120]p<0.001
- c. Mentally MuddledF=6.02 [3,117]p<0.01
- d. Inefficient.....F=3.42 [3,120]p<0.05
- e. Sleepy.....F=9.09 [3,120]p<0.001

It is interesting to note that these negative mood states usually peaked on the third (penultimate) test session, with little change on the fourth and final testing.

LMQ Interactions

Significant interactions were found for 'Time of Day' x 'Test Sessions'. In general, at successive test sessions, subjects felt increasingly:

- a).Tired b).Mentally Muddled c). Inefficient

On these three mood dimensions, the ratings for the afternoon group and evening group were very similar during baseline testing (outside the chamber), and corresponded well at subsequent tests, inside the experimental chamber. However, the two groups diverged markedly on the third (penultimate) test session. The afternoon group displayed a sharp deterioration at session 3 (clock time: 3.00 pm) and by session 4, the trend was reversed towards a recovery. The evening group did not show a similar degree of fluctuation. These trends are graphically plotted – see figures 2.21–2.23.

LMQ-Time of Day x Test Sessions Interaction For 3 Mood States

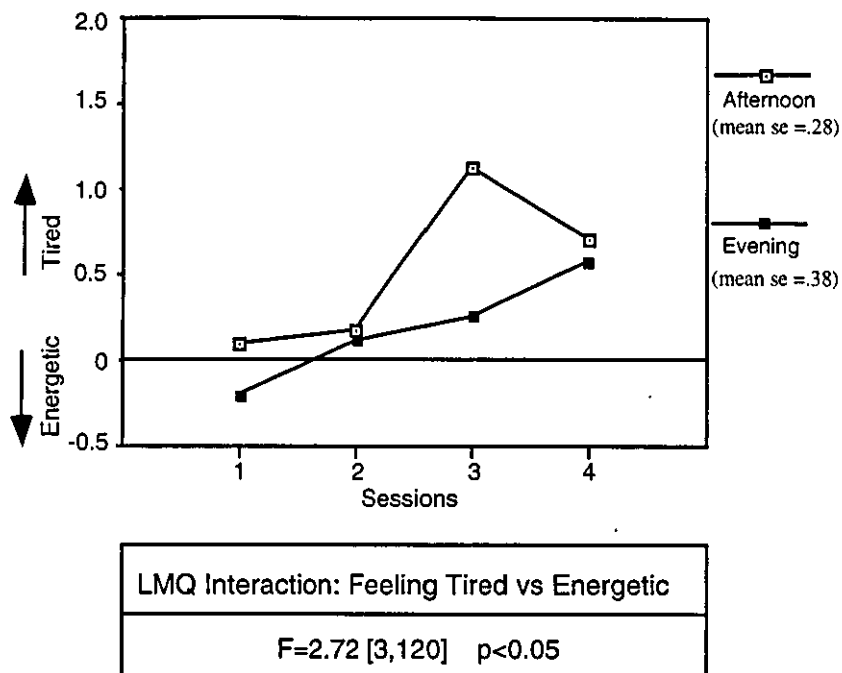


Fig. 2.21 LMQ Interaction: Time of Day x Test Session For Tiredness

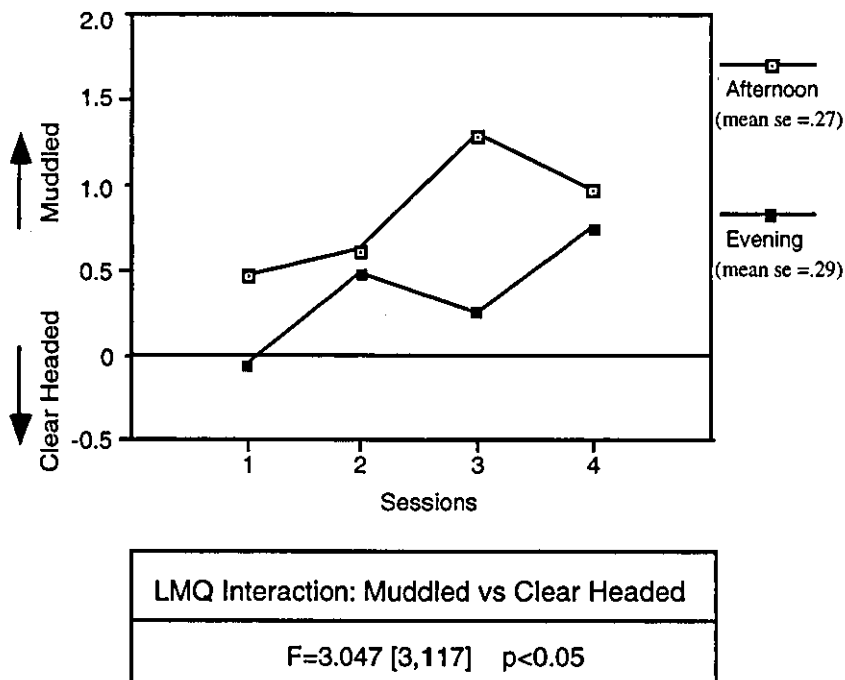


Fig. 2.22 LMQ Interaction: Time of Day x Test Session For Clear Headedness

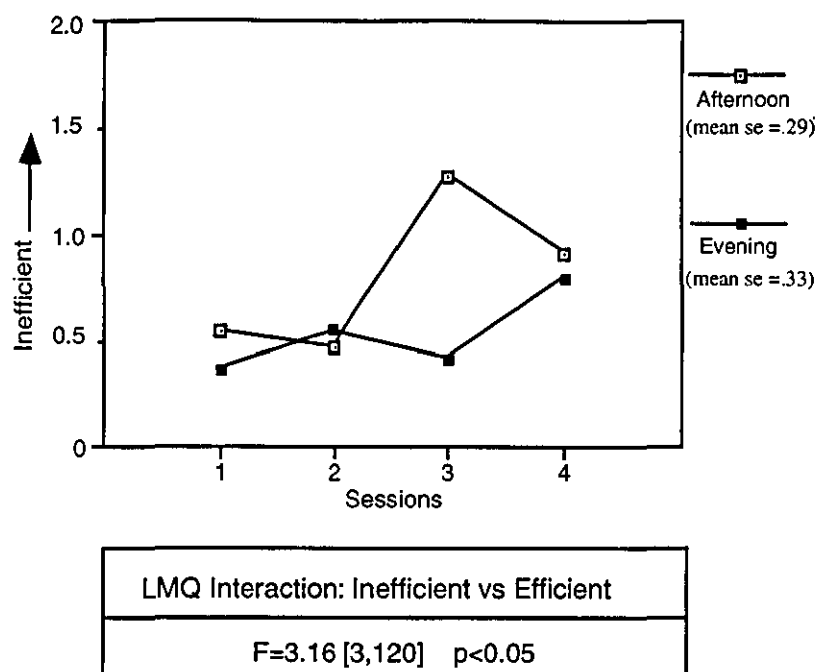


Fig. 2.23 LMQ Interaction: Time of Day x Test Session For Feeling Efficient

These three interactions plotted above (figs. 2.21 to 2.23) suggest that marked deteriorations to mood states occurs in the afternoon at test 3 (clocktime: 1500h). This visual trend can be investigated further by way of post-hoc analysis. The dip at test 3 can be examined relative to changes from the preceeding and succeeding tests (test 2 and test 4) which supposedly reflect the normal afternoon states. This can be contrasted with similar changes occuring at test 3 during the evening. A detailed account of this analytical procedure is provided in Box 1 (see page 72). Summarised below is the results of such an analysis carried out for the three mood states mentioned above which purport to show an afternoon trough at test session 3. It is clear from the results below that the negative mood states seen in the afternoon at test 3 (ie.1500h), are significantly different to any similar changes occuring in the evening at test 3.

LMQ : Mood Dimensions	Afternoon Changes at Test 3 Minus Evening Changes at Test 3 <i>Two-Tailed T-test on "Difference Scores"</i>
Tiredness	t= 3.46 (23); p<0.01
Muddled	t= 3.37 (23); p<0.01
Feelings of Inefficiency	t= 3.56 (23); p<0.01

Heart Beat Monitoring

Treatment of the Data

For each subject, the average beats per minute was continually monitored and logged. All the subjects spent 3-hours 15 mins. in the chamber – for practical purposes 3-hours was considered. A recording of 3-hours (180 mins) yielded 180 data points per subject. For statistical analysis, the 180 data points were divided into 6 equal blocks – each block containing 30 data points (i.e. 1 block = half-hour). All subjects had their mean heart beat for each half-hour block calculated, thus yielding 6 averages per subject.

Table 2.13 The Partitioning of Heart Beats Into 6 Equal Time Blocks

Afternoon	1300 - 1330	1330 - 1400	1400 - 1430	1430 - 1500	1500 - 1530	1530 - 1600
Time Blocks	Adjustment Block	Block 1	Block 2	Block 3	Block 4	Block 5
Evening	1900 - 1930	1930 - 2000	2000 - 2030	2030 - 2100	2100 - 2130	2130 - 2200

Data from Block 1 was not used for statistical analysis. The abruptness of the temperature change upon entering the chamber, and the unfamiliar environment requires a degree of acclimatization and psychophysiological adjustment. On this basis Block 1 was deemed unsuitable for inclusion in the analysis.

Macroscopic Analysis

A global overview of the cardiac data, highlights the narrow differences that exist between experimental conditions. The strongest effect appears to come from the variable, ‘Subject Interest’. However, data in this condensed form is cursory in nature, and the full effects can only be revealed by detailed statistics (analysis of variance) which follows overleaf.

Table 2.14 The Means (& SE) of Heart Rate (b/min) For The Major Variables

Time of Day		Subject Interest		Room Temp.	
Afternoon	75.68 (2.0)	Boring	73.57 (1.9)	Warm	75.55 (1.8)
Evening	77.07 (2.1)	Interesting	79.18 (2.0)	Cool	77.27 (2.2)

Overall Mean Heart Rate (b/min) For All Subjects = **76.37** (se=1.43)

Heart Rate – Main Effects

Significant main effects were found for ‘Subject Interest’, $F=4.78$ [1,38]; $p<0.05$. The boredom group showed a greater slowing in heart rate compared to the ‘interest’ group.

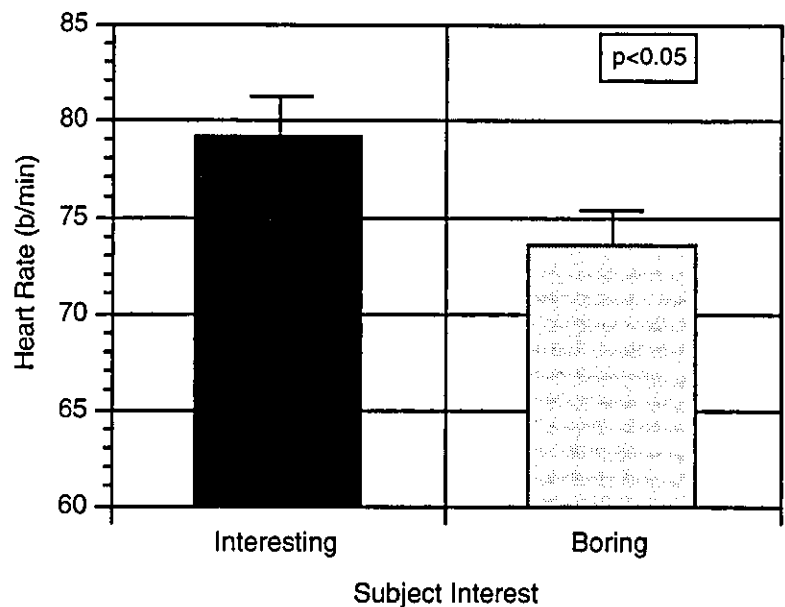
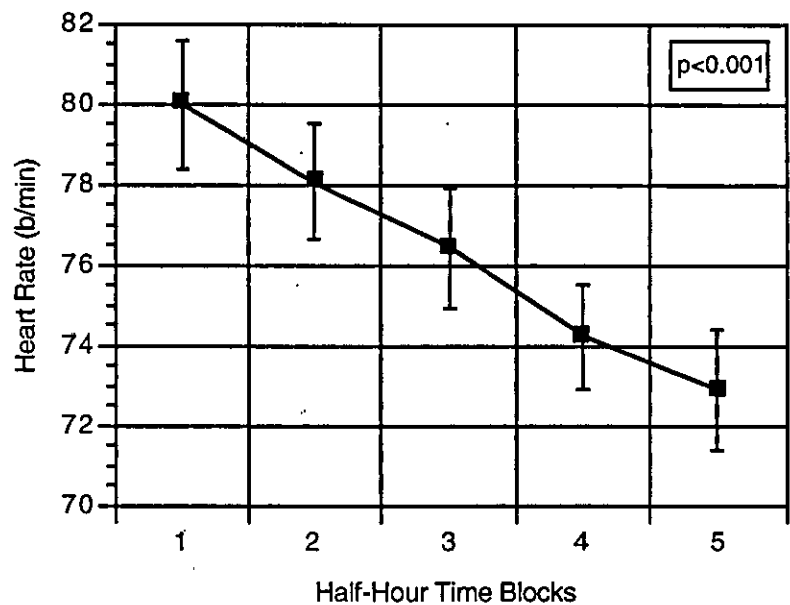


Figure 2.24. Mean Change in Heart Rate For The ‘Boring’ & ‘Interesting’ Groups

Within-Factor : Time Blocks

As subjects were seated throughout the 3-hours in the chamber, cardiac slowing was expected: this can be seen in figure 2.25, where a progressive fall in heart rate is evident at each Time Block.



Figur 2.25. The Average Beats/Min. for Each Time Block; All S's.

Heart Rate – Interactions

A three-way interaction between ‘Time of Day’ x ‘Subject Interest’ x ‘Time Blocks’ was significant, $F=2.40$ [4,152]; $p=0.05$. From the graph below, it is clear that boredom experienced in the afternoon, will cause the greatest slowing of heart rate; the evening boredom group follow a similar course, but show a smaller depression in heart rate. When subjects are aroused with interesting stimuli, the heart rate is visibly higher, but with little clear-cut differences between the afternoon and evening groups. In essence, boredom effects are potent and characterised by strong time-of-day effects.

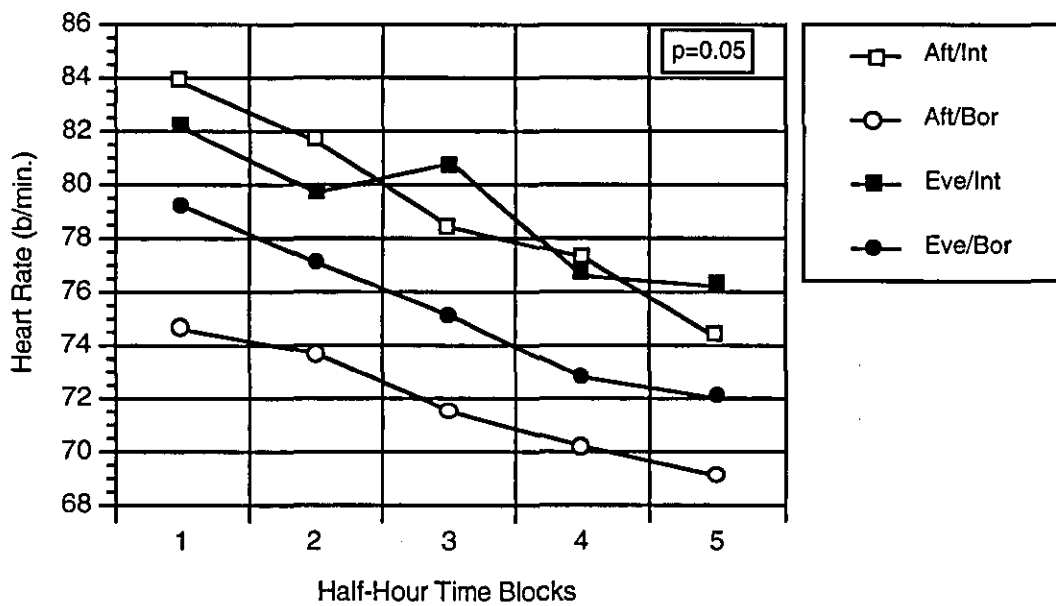


Figure 2.26 Heart Rate Interaction – Time of Day x Subject Interest x Time Blocks

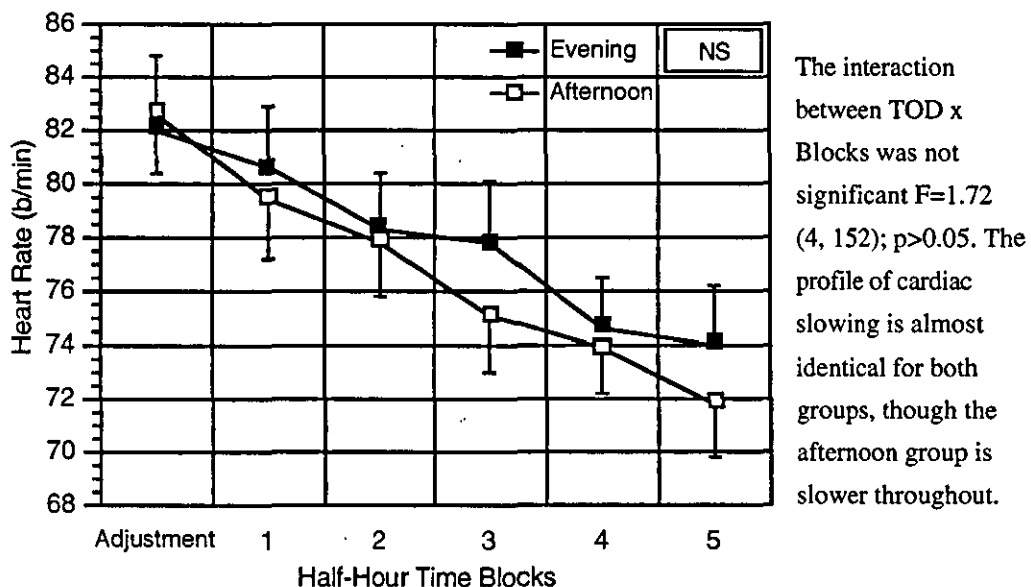


Figure 2.27. Heart Rate Interaction : Time of Day x Time Blocks

A significant interaction was also found for 'Time of Day' x 'Temperature', $F=6.05$ (1,38); $p<0.05$. In terms of cardiac reaction, temperature appears to have the opposite effect depending on whether the recordings were performed in the afternoon or early evening (see figure 2.28.). A cool chamber elevates heart rate in the evening, but depresses it in the afternoon. On the other hand, a warm room slows the heart rate in the evening, but pushes it up in the afternoon.

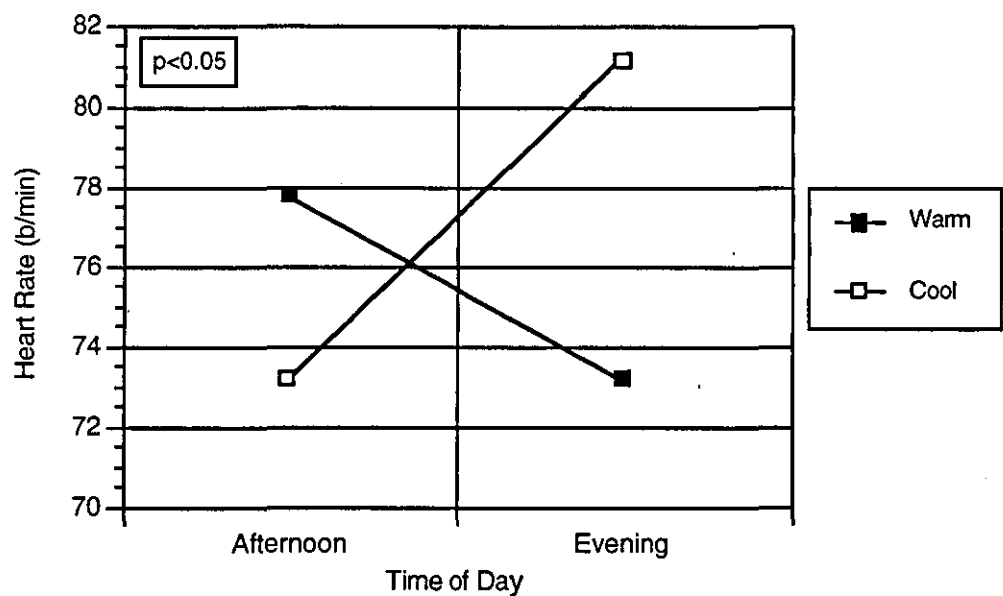


Figure 2.28 Heart Rate Interaction – Time of Day x Temperature

Aural Thermometry

Treatment of the Data

Temperature readings were obtained via the external auditory canal every second, and readings automatically averaged over one-minute epochs. The data-set was then organised in a manner identical to the treatment of the cardiac data. In essence, data from a 3-hour recording period was partitioned into 6 half-hourly blocks. The mean aural temperature for each block was calculated for all the subjects. The first two blocks represented periods of psychophysiological adjustment and were discarded, leaving 4 successive blocks for detailed analysis.

Table 2.15 The Partitioning of Aural Temperature Into Half-Hourly Blocks

Afternoon	1300 - 1330	1330 - 1400	1400 - 1430	1430 - 1500	1500 - 1530	1530 - 1600
Time Blocks	1-Hr. Psychophysiological Adjustment		Block 1	Block 2	Block 3	Block 4
Evening	1900 - 1930	1930 - 2000	2000 - 2030	2030 - 2100	2100 - 2130	2130 - 2200

All subjects entering the climatic chamber experienced an abrupt change in air temperature, away from thermoneutrality. The extended 1 hour ‘adjustment period’ was a cautionary measure to give the homostatic mechanisms of temperature regulation sufficient time to acclimatise before reacting to the experimental manipulations. Because of thermal inertia, core temperature takes time to adjust. Poulton has also noted an idiosyncratic stimulating effect which occurs upon first entering a warm room¹⁵⁰.

An analysis of variance was carried out with the actual temperature readings using:
3 Between Group Factors (Temperature; Time of Day and Subject Interest)
and 1 Repeated Measure with 4 levels (Time Blocks: 3, 4, 5 & 6).

Aural Temperature : Main Effects

The main effect of ‘Subject Interest’ produced significant changes to body temperature, $F=6.16$ [1,33]; $p<0.025$. Subjects in the ‘boring condition’ showed a greater drop in body temperature than subjects receiving ‘lively’ stimulation in the ‘interesting condition’. See figure 2.29 below.

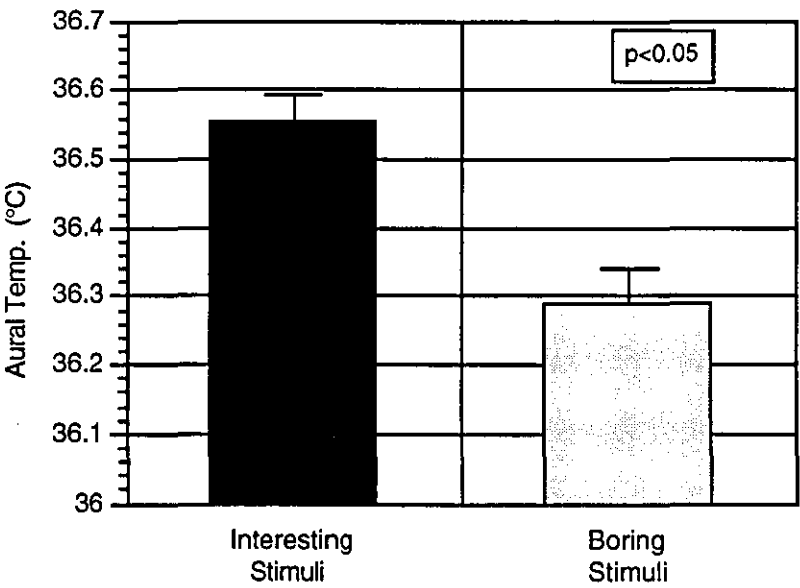


Fig 2.29. Aural Temperature : Main Effect Subject Interest

The main effect of Room Temperature did not produce significant changes to body temperature; $F= 3.37$ [1,33]; $p<0.076$. Nevertheless, an apparent trend pointed to a greater lowering of body temperature in the cool chamber. This is graphically illustrated in figure 2.30 (overleaf).

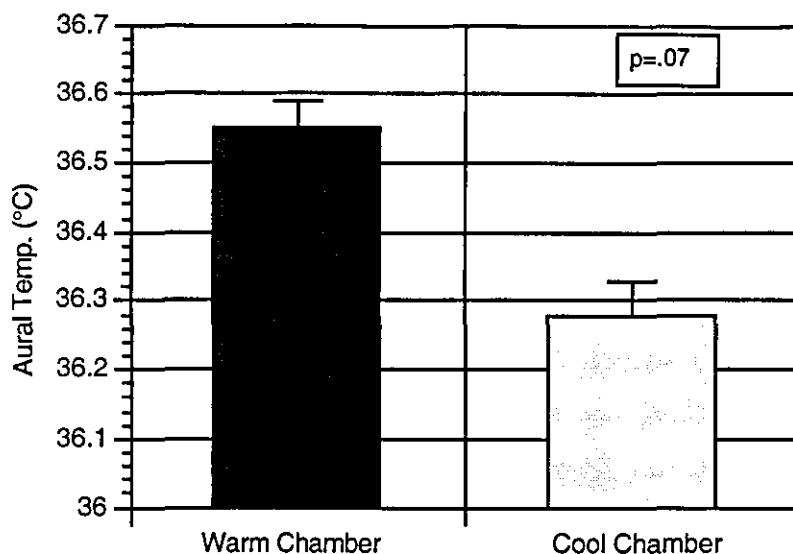
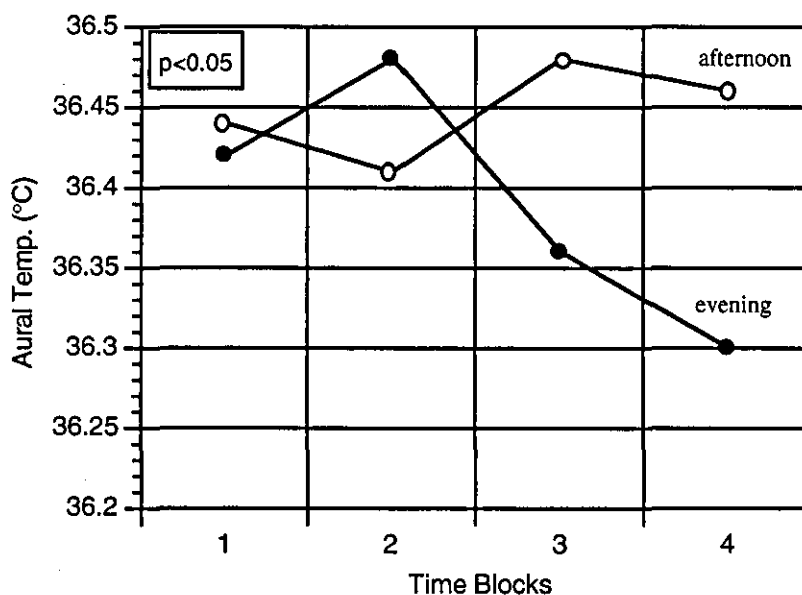


Figure 2.30 Aural Temperature: Main Effect Room Temperature

Aural Temperature: Interactions

A significant interaction was found between Time of Day x Time Blocks; $F=3.22$ [3,99]; $p<0.05$. The afternoon group, on average peaked in temperature around 1500-1530 h. The evening subjects similarly peaked in temperature between 2030-2100 h. This was followed by a sharp fall in temperature as the evening hours drew forth.



Time	1400 - 1430	1430 - 1500	1500 - 1530	1530 - 1600
Afternoon N=24	36.44 (.08)	36.41 (.09)	36.48 (.08)	36.46 (.10)

Time	2000 - 2030	2030 - 2100	2100 - 2130	2130 - 2200
Evening N=24	36.42 (.12)	36.48 (.09)	36.36 (.10)	36.30 (.12)

Figure 2.31 Aural Temperature Interaction: Time of Day x Time Blocks

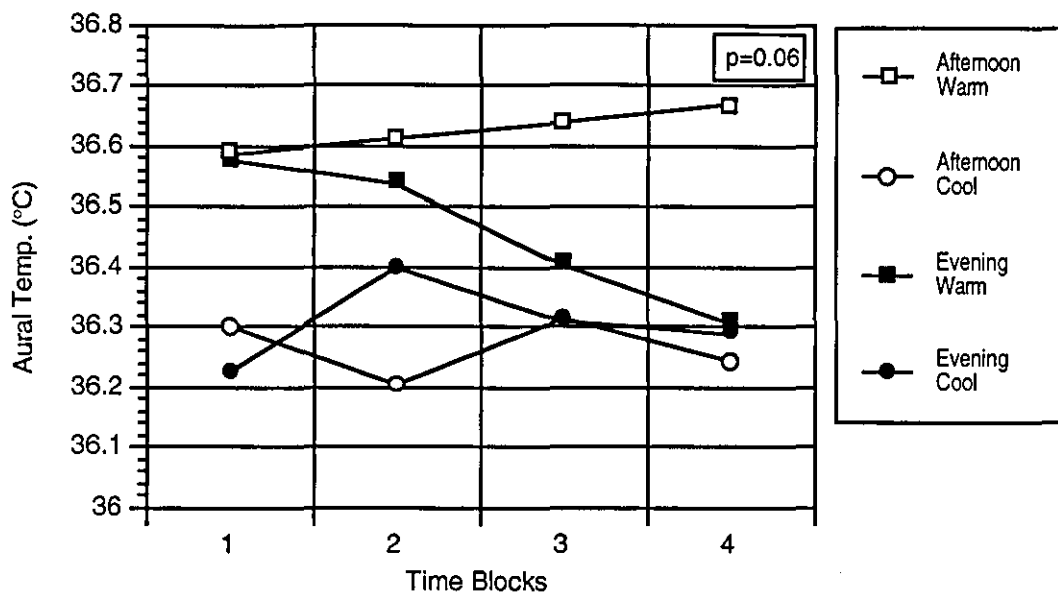


Figure 2.32. Aural Temperature Interaction: Time of Day x Room Temperature

A three-way interaction between 'Time of Day' x Temperature x Time Blocks approached significance, $F=2.57$ (3,99); $p=0.059$. Room temperature exerted a greater influence on body temperature in the afternoon: the cool chamber depressed body temperature markedly. For the evening group, the divergence in body temperature that result from a warm or cool room was less pronounced, especially after time block 2.

Sleep Profile: *Previous Night Sleep/Daytime Sleepiness.*

As a general commentary on the make up of the sample, all 48 subjects were profiled with sleep questionnaires. Essentially, interest lay in finding out how closely matched the 24 afternoon subjects were to the 24 evening subjects in habits of sleep. There were two specific areas of interest:

- The sleep obtained on the evening prior to the study.
- General experience of daytime sleepiness & napping.

1. Previous Night Sleep (evening prior to experiment)

On the morning of the experiment, all subjects completed a questionnaire probing their sleep and wake times. Tabulated below [table 2.16] are averages (+se) for both groups.

Time of Day	Fell Asleep	Woke Up	TST	Lie-In After Awakening
Afternoon	00:38 h (.23)	7:45 h (.15)	7:07 hrs (.25)	00:22 mins (.07)
Evening	00:11 h (.29)	7:41 h (.15)	7:30 hrs (.25)	00:15 mins (.07)
T-Test	NS $t=1.25$ (46) $p=.22$	NS $t=.27$ (46) $p=.79$	NS $t=-1.1$ (46) $p=.30$	NS $t=.74$ (46) $p=.46$

Considering ‘time in bed’, the evening group averaged 16 minutes more than the afternoon group. The mean TST for evening group was inflated by 3 scores (10hrs sleep) : partialling out these outliers brought the evening mean to 7:08 (hr:min), almost identical to the 7:07 mean of the afternoon group. Even without this correction, the scores between the two groups were statistically very similar. Taking these factors together, it is clear that the 24 afternoon subjects and 24 evening subjects received similar amounts of sleep on the night preceeding the experiment.

Table 2.17 below presents the data from the subjective self-reports completed on the morning of the experiment. There were no significant differences found between the groups on residual sleepiness, waking difficulty or the ‘quality’ of sleep obtained.

Table 2.17 Post-Sleep Reports: completed soon after arising on the experimental day.

Time of Day	Sleepiness after 30 min. awake (SSS)	Difficulty in getting up	Quality of sleep obtained
<i>Afternoon</i>	3.0 (.19)	3.0 (.18)	2.9 (.17)
<i>Evening</i>	3.1 (.22)	3.2 (.19)	2.8 (.15)
<i>T-Test</i>	<u>NS</u> t=-.44(45) p=.67	<u>NS</u> t=-.79 (46) p=.43	<u>NS</u> t=.18 (46) p=.89
QUESTIONS & SCALES	SSS <u>7 Point Scale</u> 2 = Functioning at high level..... 3 = Relaxed, awake..... 4 = A little foggy.....	How difficult was getting up this morning? <u>5 Point Scale</u> 2 = Easy 3 = Moderate 4 = Difficult	Which is the best description of the quality (not quantity) of your sleep last night? <u>5 Point Scale</u> 2 = Better than normal 3 = Normal 4 = Worse than normal

2. General Experience of Daytime Sleepiness

In a screening questionnaire that was completed 1–2 weeks before the experiment, all subjects were probed on their experiences of daytime sleepiness. The results which are tabulated overleaf indicate that the two groups did not differ significantly in any of the parameters profiled. From table 2.18 it is quite clear that the reports of sleepiness are localised to the afternoon. On average, daytime sleepiness occurred around 14.30 h and ended about 16.15 h. Also shown is the average midpoint of afternoon sleepiness as calculated from each report of daytime sleepiness.

Table 2.18. Responses (mean with se) to questions on daytime sleepiness

QUESTION: If you suffer from daytime sleepiness, when is this sleepiness likely to start & end?			
Time of Day	Sleepiness Starts	Sleepiness Ends	Mid-Point
<i>Afternoon</i>	14:22 h (.42)	16:06 h (.45)	15:22 h (.34)
<i>Evening</i>	14:37 h (.42)	16:23 h (.41)	15:30 h (.40)
<i>T-Test</i>	<u>NS</u> t=-.43(37) p=.67	<u>NS</u> t=-.46 (37) p=.43	<u>NS</u> t=-.24 (36) p=.81

The next question was more specific in that it probed the frequency of daytime sleep intrusions [see table 2.19, below]. For both groups, episodes of daytime sleepiness appears on average between 'once a month' and 'several times a month'. The inter-group differences were not significant.

Table 2.19 Responses (mean with se) to questions on frequency of daytime sleepiness

Question:	Time of Day	Difficulty staying awake
<i>Do you ever have difficulty staying awake during the day?</i> Never..... (0) Once a month..... (1) Several times a month... (2) Most days/week..... (3)	<i>Afternoon</i>	1.5 (.19)
	<i>Evening</i>	1.8 (.15)
	<i>T-Test</i>	<u>NS</u> t=-1.5(45) p=.14

Table 2.20 Proportion of subjects engaging in napping with mean (se) nap times & length.

Question:	Time of Day	Nappers (frequency variable)	Nap Time	Length of Nap
<i>Do you take daytime naps? If yes:Then at what time & how long do these naps last?</i>	<i>Afternoon</i>	N=7 (29%)	15:45 h (.65)	43 mins (7.6)
	<i>Evening</i>	N=7 (29%)	15:25 h (.15)	42 mins (5.1)
	<i>T-Test</i>		<u>NS</u> t=.46(11) p=.65	<u>NS</u> t=-.13(11) p=.90

Finally, an attempt was made to discover what proportion of the group actually resorted to napping as a result of daytime sleepiness. The results are tabulated on the previous page [table 2.20]. In both the afternoon and evening groups, only 7 subjects reported napping — the frequency of napping was variable in both groups.

In summary, the *raison d'être* of this descriptive sleep profile was to highlight the homogeneous nature of the sample in terms of sleep habit. It is an addendum to the main results. Subjects arrived at the experimental situation having experienced a comparable 'previous night sleep' and their subjective reports, immediately upon waking, also corresponded well. Their general experiences of daytime sleepiness were equally concordant. So, in this respect, we can rule out any confound arising from individual differences. Taken together, these findings would lend a special credence and confer a reliability upon the main results.

This first experiment sought to examine the afternoon trough in alertness within the context of circadian rhythmicity. In a comparative study, the low point of the afternoon was contrasted with the circadian peak in alertness, that is typical of the early evening. Using an independent-groups design, 24 ‘afternoon subjects’ were to receive identical treatments as 24 ‘evening subjects’. Thus, any difference in performance scores or subjective activation between these two sets of subjects, must – to a large extent – be attributable to circadian phasing. Such a prediction follows from our understanding of biological rhythms and diurnal fluctuations in particular.

On the basis that exogenous factors in the social environment can/and do mask daytime sleepiness, an attempt was made to actively thwart this deceptive shroud and so allow the free expression of endogenous sleepiness. A novel approach to the unmasking of sleepiness was employed. Arousal levels were successfully manipulated by the presentation of ‘realistic’ stimuli, designed to induce either ‘cognitive’ boredom or interest. It was hypothesised that laboratory induced arousal troughs, coupled with naturally occurring circadian troughs of alertness, would be sufficiently potent to overwhelm any behavioural impositions to sleep, and so reveal the APS.

The first point of interest to emerge from this experiment concerns the powerful effects of masking. The baseline measures (taken prior to arousal manipulation) showed that the afternoon subjects and evening subjects had very similar scores on reaction time and ratings of sleepiness. As an example, the mean (baseline) reaction time score for the afternoon group was 0.299 secs., and for the evening group, 0.302 secs. T-tests were to show that these differences were not significant. Other examples are tabulated below; the last column highlights the occurrence of significant ‘time-of-day’ effects (main effects and interactions) which followed arousal manipulations.

Table 2.21 Mean Baseline Scores & Subsequent Significant Changes After Treatment.

Test	Afternoon Baseline	Evening Baseline	Difference (t-test)	Significant TOD effects found <u>after</u> arousal shift
RT	0.299 secs	0.302 secs	NS	Yes
SSS	2.6	2.7	NS	Yes
ADS	4.29	4.21	NS	Almost
VAS	38.5	33.4	NS	Yes

It is clear from the table above, that mere testing at intervals in the waking day, is not sufficient by itself to reveal diurnal changes. However, if exposed to the experimental regimens, as proposed here, the two groups (afternoon vs evening) do react differently; RT for instance, was markedly impaired in the afternoon group (see fig 2.5). This finding, which revealed the hidden circadian effects, was due largely to the success of the experimental procedures, designed to alter arousal levels. The failure of other investigations to uncover diurnal rhythms, must be seen in this light, i.e. stymied by uncontrolled alerting factors in the environment which masked endogenous sleepiness.

In his classic study on diurnal change, Blake failed to show any significant variations in reaction time tests⁷⁴. A result that was described as “surprising” in a review by Hockey and Colquhoun¹⁵¹ (in view of other significant findings). Blake tested subjects at 5 time-points: his last two tests, at 1530 h. and 2100 h. (times which coincided with this current experiment) yielded mean RT scores of 315 and 314 m/s respectively. The reviewers concluded that reaction time “may not have been sufficiently sensitive to time of day effects.” However, in an act of prescience, it was suggested that cycling in simple reaction time tests may be prone to motivational effects.

In Blake’s repeated measures design, subjects conducted each test on a different day – the five tests took five days. Subjects arrived at each test situation in a ‘fresh’ state. It can reasonably be assumed that the mood or affective state of the subject, was different on each occasion, and the motivation to perform, would likewise vary. A major aim of this experiment into the APS was to control arousal levels prior to any psychological testing. Subjects were either ‘brought down’ to a low level of arousal, or ‘perked up’ to a lively emotional state; in essence, a homogeneity of mood particularised each group.

Transient Nature of the APS

A major hypothesis of this experiment asserted that the APS was a transient event: an afternoon trough would be followed at some point by an ‘upturn’ or recovery. To what extent was this prediction correct ?

After administering the baseline tests, further measures were taken inside the experimental chamber at four distinct periods (intervals of 50 minutes). Analysis of variance highlighted significant deteriorations to mood and performance from test 1 to test 4. For example, RT became generally slower at each testing and subjects also reported increased subjective sleepiness as measured by the SSS (fig. 2.11), ADS (fig. 2.15) and the VAS (fig. 2.19). Similarly, the measures of mood as gauged by the LMQ indicated progressive deterioration in ‘vigour’, ‘mental clarity’ and rising ‘fatigue’ at successive test sessions. These significant findings were derived from the repeated measure, ‘Test Sessions’, which represents the ‘collapsed’ scores of all 48

subjects. A generalised trend is plotted below (figure 2.33), which for illustrative purposes, paths the average course taken by many of the above variables.

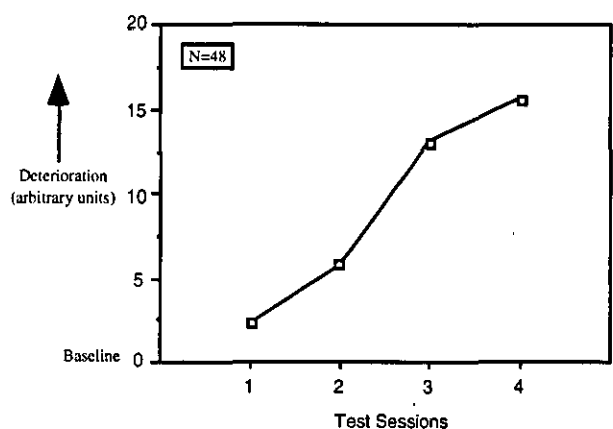


Figure 2.33
A generalised trend for the
'within factor' Test Session

The above plot draws attention to the significant changes between successive tests: it is interesting, containing the collapsed scores of all 48 subjects, but, however, it fails to reveal important between-group changes. When we examine the interaction between 'Test Sessions' x 'Time of Day', an interesting finding emerges. While both the afternoon and evening group exhibited a decline in mood and activation, it was usually the afternoon group that were worse affected. But more crucial is the finding which pin-points this afternoon impairment (with its peak in sleepiness) to occur maximally at Session 3 (clock time: 1500 h). Moreover, by the next and final testing, session 4, there was a reversal towards recovery. By contrast, the evening group continued to show a decline in an almost linear fashion. Figure 2.34, is a representative graph which illustrates the point. Real examples of this actual trend can be clearly seen for RT (fig. 2.8) and the ADS (fig. 2.17), with statistical significance in the case of the SSS (fig. 2.13) and the mood measures probing 'fatigue', 'clear-headedness', and feelings of 'efficiency' (figs. 2.21 to 2.23).

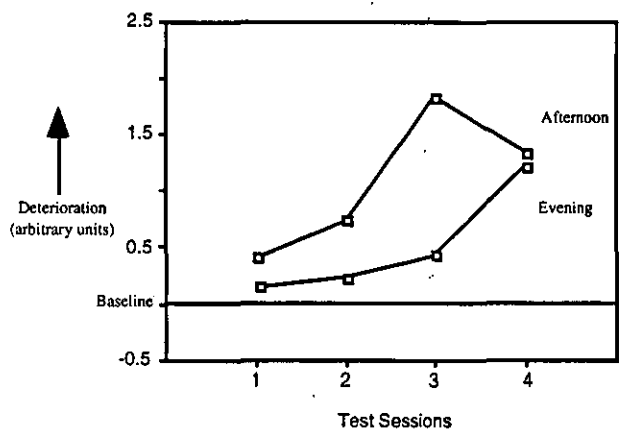


Figure 2.34
A generalised trend showing
the interaction for
'Time of Day x 'Test Session'

End of Test Effect ?

Taken together, these findings would tend to suggest that the afternoon trough, which is followed by a remarkable recovery, is evidence of the transient nature of the APS. There is however another explanation which could account for the sudden improvement on the last test. It could be a phenomenon known to many researchers as the 'end-of-day' effect – an exultant/compensatory response in anticipation of completing the day's task. Colquhoun describes a similar late peaking in Blakes seminal study⁷². Infact, both authors were cognizant of motivational factors affecting psychological tests. Studies in industry have also reported 'End-Spurt Curves' in piece-work situations in response to the 5 o'clock bell^{see 152}. Still others have invoked the Hawthorne Effect to account for such late recovery¹⁵³.

However, none of the above theories can be applied to the present findings. Indeed, there was a trough followed by a final recovery – but if this was an 'end-of-day' effect, then why was there no corresponding pattern for the evening group where conditions were replicated. If we bring to the discussion our *a priori* knowledge of the APS as a transient phenomenon, with a narrow time-window, then this final 'recovery' (peculiar only to the afternoon group) is explicable.

The Temporal Characteristics of the APS

If we propose the APS to be a transient event, occuring in the afternoon, then it is incumbent upon us to state *when* in the afternoon it is likely to occur. The real-world implications of the APS warrants such a pragmatic concern. This experiment has shown that marked impairment and greatest sleepiness occurs in the afternoon at test-session 3. The time of this test battery was 1500 h. In fact, the actual 'low-point' could have set in 15 minutes prior to the 1500 h testing and could extend to 15 minutes beyond post-testing (NB. recall that tests were given every 50 minutes). So, although the peak in sleepiness was identified to occur around 1500 h., we could conservatively place this 'low-point' in a time window between 1430 to 1530 h.— the midpoint would of course be 1500 h. Is such a time frame supported by the literature?

In general, the information on the timing of afternoon sleepiness is very scant; methodological differences, in particular the lack of frequent testing makes across-the-board comparisons very difficult. Nevertheless, the hours of 1330 – 1600 h. remains a broad enough band to cover most reports of afternoon sleepiness. Researchers investigating ultradian rhythms, are compelled to administer frequent tests, and their work has furnished invaluable data in the study of diurnal variations. A champion of this research paradigm, Peretz Lavie¹¹², has shown a peak in sleepiness to occur between 1500 – 1600 h., a period grandly referred to as the 'Secondary Midafternoon

Sleep Gate'. Other data by Lavie¹¹¹ would suggest that subjects who typically retire to bed by 2300 h. will find their midafternoon sleepiness to be greatest around 1500 h. It would appear that the timing of afternoon sleepiness is phase-locked to the onset of nocturnal sleep. Reference was made to this point in Section 1 (page 13), which indicated that the mid-point of the afternoon nap was 180 degrees out of phase with the mid-point of the primary nocturnal sleep. It follows that for a working population with early bedtimes, a 1500 h. sleepiness peak is predictable, whilst for a college populous with later sleeping habits, reports of a 1600 h. modal nap time are equally cogent²³. Tabulated below (table 2.22), is the actual primary sleep midpoint from the night prior to the experiment, for both the afternoon and evening groups – for the moment it is only the afternoon group that is of interest. A week before the experiment, all subjects were questioned on their general experiences of daytime sleepiness: also tabulated below is the midpoint of this reported sleepiness.

Table 2.22 Midpoint of previous night sleep and subjective reports of daytime sleepiness.

Time of Day	Mid-point of nocturnal sleep (night prior to experiment)	Mid-point of daytime sleepiness from self-reports prior to experiment
<i>Afternoon</i>	4:08 h (.15)	15:22 h (.34)
<i>Evening</i>	3:53 h (.19)	15:30 h (.40)
<i>T-Test</i>	<u>NS</u> t=1.07 (46) p=.29	<u>NS</u> t= -.24 (36) p=.81

The midpoint of nocturnal sleep for all subjects occurred around 04.00 h. By classic reckoning of the “180° phase relation theory”, the midpoint of the afternoon nap should be around 1600 h. If the sleep midpoint is reflective of maximal sleepiness and impairment, then the subjects of this experiment were slightly early by their 1500 h. trough. Relative to their subjective experiences of daytime sleepiness (midpoint = 15:22 h.), they were only fractionally out of phase. These disparities are slight, but they do suggest that the 180° phase relation to be less than rigid. For example, this phase relation has been found to be subject to distortion in studies of extended sleep, where, instead of the 12 hr. re-appearance of SWS, we get a secondary appearance 12.5 – 13.0 hrs later^{53, 54}. A margin of flexibility; 1-hr. either way, appears likely. A sleep rhythm that is able to cope and yield to the vagaries of the social and physical environment can only be adaptive.

The 1500 h. trough as revealed by this experiment, and the work of Lavie are essentially laboratory derived examples. For studies with greater ecological validity, the field data collected on the hourly distribution of motor vehicular accidents provide valuable insight. Zomer and Lavie¹⁵⁴ gathered data from police records concerning all sleep related vehicular accidents in Israel during the period 1984 – 1989. They found a major peak in accidents occurring during the early morning (3-4 am.) and a secondary peak occurring around 1500 h. In contradistinction, the pattern of ‘total number of accidents’ did not show a similar bimodal course as sleep related accidents; see below.

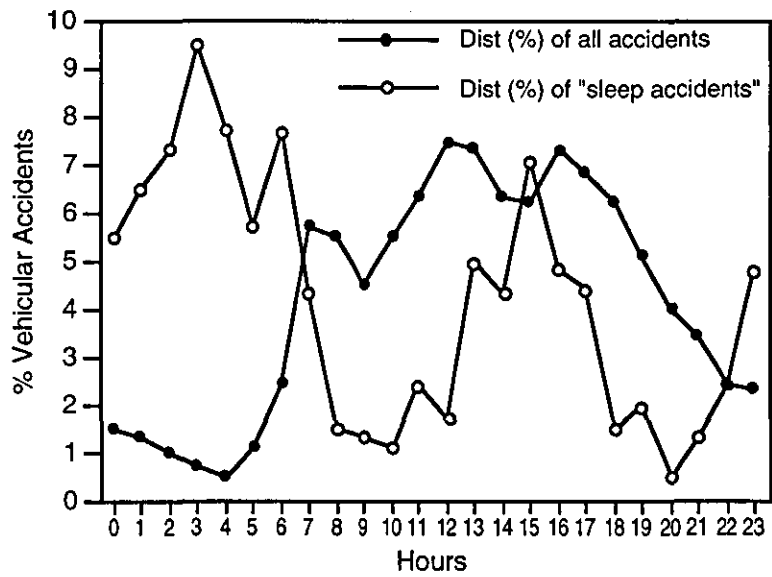


Figure 2.35 The hourly distribution of accidents in Israel during 1984–1989. [ref:154]

It is interesting to note that 75% of sleep related accidents occurred outside urban areas – where (*I assume*) environmental stimulation tends to be less varied, leading to the onset of boredom. In a similar vein, motorway drivers are victims to a surfeit of boredom, and data from Horne¹⁵⁵ show an afternoon cluster of accidents around 1500h. Bjerner et al. examined errors in meter reading in over 175,000 ledger entries^{see 156}. A large peak in errors occurred at 03.00 h. in the morning, with a secondary peak at 15.00 h. in the afternoon. Finally, in a classic study of 15,000 german engine drivers, Hildebrandt et al. examined performance efficiency in braking, and the perception of danger signals¹¹⁰. Here again, a peak in errors was reported to occur at 15.00 h.

To summarise; this experiment has located the critical zone of APS sleepiness at or around 1500 h. Compared to most studies, the time-frame of afternoon sleepiness presented here, has added accuracy due to the frequent afternoon testing intervals. Data from ultradian research paradigms and field studies, generally confirm these findings. It would also appear that the timing of the APS may be linked to the timing of the major nocturnal sleep. An extreme example of this 3 o'clock malaise is described in a recent

issue of the medical journal *JAMA* (Jan. 1992)¹⁵⁷. In a paper by Thomas Wehr, a patient suffering from pathological depression eloquently relates his experience of mood changes during the period of the APS [note also the recovery towards early evening as he enters the circadian upswing, or Lavie's 'Forbidden Zone of Sleep']:

"Afternoons were.....the worst, beginning at about three o'clock, when I'd feel the horror, like some poisonous fogbank, roll in upon my mind, forcing me to bed. There I would lie...waiting for that moment of the evening when, mysteriously, the crucifixion would ease up....The evening's relief for me—an incomplete but noticeable letup, like the change from a torrential downpour to a steady shower—came in the hours after dinnertime." [Ref: 157]

A classic symptom of depression is subjective fatigue, absence of energy and the general impression is that depressives are "in a state of very low arousal"¹⁵⁷. Note then the serendipitous observation in this clinical example which shows the power of a diminution in subjective arousal to unmask the APS. According to Wehr, 60% of depressive patients report a diurnal variation in mood.

Arousal

The above example brings to the discussion the concept of "arousal" as an explanatory construct to account for the observed time-of-day effects. So far, I have used the term rather loosely; but the operational usage of the term can be explicit; depending upon which of the conflicting schools one subscribe to. It is a debate outside the focus of this thesis, but a brief comment is appropriate.

Much has been written about the inadequacies of the *unidimensional* theory of arousal (see Folkard¹⁰²). To counter this, Lacey discussed the existence of three subsystems which correlate well and which he believed reflected levels of arousal. He characterised three kinds of arousal: Behavioural, Cortical and Autonomic. These have been conventionally reduced to i. Physiological Arousal and, ii. Behavioural Arousal. The latter is psychologically based, and influenced by factors such as cognitive boredom, incentives and knowledge of results¹⁵⁸.

There are great dangers in relying on purely physiological correlates of arousal. For example, the states of anger, excitement, sexuality and fear share a number of common physiological components: all these states affect arousal in the same direction¹⁰³. Instruments to measure physiological indices of heart rate or skin conductance would register change in the same direction, with little differentiation between the states of anger or excitement. This physiological approach to arousal patently fails to take into account the psychological context. To say that this APS experiment manipulated arousal *per se* would be unduly nebulous and sweeping. The use of 'boring' and 'interesting'

stimuli was directed at the cognitive or mental state of the subject in a bid to lower 'behavioral arousal' at a point in the diurnal phase when it was likely to be low, due to the coincident secondary sleepiness cycle. The room temperature variable was but one component in a larger scheme of change. Indeed, the temperature manipulation (physiological) and psychological 'interest' manipulation (cognition) probably affect different bodily systems. The importance of the psychological variable has been understated, leading Neiss to stress: "As an intervening variable artificially isolated from its psychological context, arousal can never be shown to cause changes in performance."¹⁰³ Recently Thayer has attempted to highlight the functional interaction between physiological and psychological arousal in producing a generalised arousal which he refers to as "energetic arousal"¹⁵⁹. According to this scheme the sleep-wake cycle is related to the changes in energetic arousal.

In attempting to relate diurnal variation in performance with the concept of arousal, the Yerkes-Dodson law has been invoked on many occasions. The inverted-U tells us that the relationship between performance and arousal is curvilinear – but very little else! Although attempts have been made to explain the 'mechanisms' of the Yerkes-Dodson law with Easterbrooks Cue Utilisation Hypothesis ^{see 160}, there still remains the untenable fact that the inverted-U function is "immune to falsification"¹⁰³.

The current zeitgeist in sleep research is to speak of circadian rhythms in "alertness" or "sleepiness". We may be held back in our researches by resorting to traditional conceptualisations of a global arousal theory. In many cases, it is a semantic jungle. Thayer sees "alertness" as bound up in his concept of "energetic arousal". The resultant tautology only adds to the incongruencies surrounding this fierce polemic.

Boredom

The APS operates in the 'real world', and as such, cannot always be expressed due to occupational and other demands. It was however unmasked by careful experiment. To understand the research paradigm that was employed, we must accept the precept that alertness is not a unitary or homogenous state; by that I mean the disposition of alertness must be viewed as an amalgam of interacting physiological and psychological events, which, depending upon their relative strengths, influence the *level* of alertness. For instance, the temperature rhythm, catecholamine rhythm, sleep-wake cycling, environmental zeitgebers, illness, affective mood, etcetra, all contribute to swaying the underlying state of alertness at any given point in time. Essentially, it can be reduced to an organismic battle between endogenous and exogenous influences. This experiment has used acute boredom as an agent to precipitate out the basal physiological sleepiness that is normally suppressed by exogenous factors.

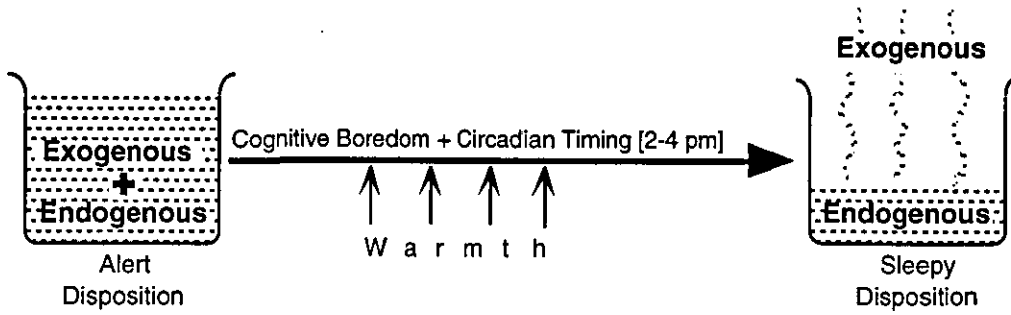


Fig. 2.36 The precipitation of basal sleepiness by dampening environmental constraints to sleep.

The objective reaction time test (RT) and cardiac measures clearly show that afternoon boredom caused greater impairment and cardiac slowing than identical evening induced boredom. This has direct bearing upon a key question recently articulated by Dinges:

“Do these behaviours or contexts (*boredom, heat, eating a large meal..*) actually cause sleepinessor do they serve to expose sleepiness (due to overall sleep need and/or circadian phase) that is not obvious in other contexts?” (emphasis mine) ref: 89.

If we accept that CNS function is impaired during sleepiness^{see 106} then the distinctly poor RT performance evident during the afternoon, is a palpable sign of this secondary sleepiness. If boredom ‘caused’ sleepiness, then its deleterious effects seen during the afternoon, should re-appear with similar intensity in the evening. This did not happen: the evening group did not suffer the same degree of impairment. We can therefore conclude that boredom presented in the afternoon, somehow ‘exposed’ an intrinsic sleepiness, at a point in the day when the sleep threshold was exceptionally low.

Field studies tend to corroborate this afternoon vulnerability to boredom effects. Industrial research conducted early this century by Wyatt, examined the falling work output at various points in the day for tasks deemed ‘boring’^{see 117}. He noted that the effects of boredom were particularly evident at certain times of the day — in the middle of the morning and again soon after the beginning of the afternoon shift. The latter drop in efficiency is amenable to interpretation under the current APS hypothesis.

An early explanation to account for the performance decrement and sleepiness resulting from boredom, was proffered by Barmack^{see 115}. In his search for physiological correlates, he made the link between these negative states with falling oxygen consumption and blood pressure levels. A more sophisticated approach burgeoned in the 1970’s when researchers tried to associate neuroendocrine activity with boredom. It was found that viewing a boring film (Levi, 1972), or reading boring magazines (Patkai, 1971), both resulted in falling adrenalin values when compared to more

engaging and interesting pursuits^{see 115}. The RT decrement produced under conditions of boredom has also been interpreted as an example of habituation of cortical arousal by Mackworth^{see 117}. While neural habituation may account for the vigilance (RT) task, it does not however explain the psychical or emotional state of boredom as revealed by self-report sleep and mood scales^{see 161}. Nonetheless, habituation may “be the psychophysiological beginning of boredom”¹⁶¹.

This experiment has also underlined the generalised effects of boredom *per se*. In every measure — from self-report sleep scales, mood scales, performance tests, to the physiological indices of heart rate and body temperature — subjects in the boredom condition were significantly affected. Plainly, they became sleepy, lethargic, performed badly, and their heart rate and body temperature fell significantly.

Whilst the subjective reports of SSS and ADS highlighted the deleterious effects of boredom, they failed to show any interaction with ‘time of day’. It is plausible that the procedure used to induce boredom was “too successful”, and its effects overwhelmed any ‘time of day’ or ‘temperature’ influences. As a side issue, it is instructive to note that the negative effects of boredom as measured by the SSS and ADS, peaked at session 3 (afternoon clock-time = 1500 h.), before levelling off. This would coincide with the results of other tests, which suggest that 1500 h. is the peak time for APS dysfunction.

There is an almost intuitive association between yawning and drowsiness: yawning has been recognised as a “paralinguistic signal for boredom”¹¹⁹. Recently, Greco et al.¹⁶² suggested that yawning served to raise arousal in situations of boredom and declining arousal. The mere physical act of yawning — opening of the mouth and deep breathing — was sufficient in itself to raise arousal; notwithstanding the various respiratory theories of yawning. It is a strategy we have to cope with boredom and sleepiness. Yawning was not a variable examined in this experiment, but one can speculate that the afternoon subjects were probably more prone to yawning, in the face of the encroaching APS.

This work on sleep facilitation (via boredom induction), allied to the circadian phase, has gained particular relevance recently as it has direct bearing to a new ‘objective model of sleep’ presented by Webb¹⁶³. He has outlined a 3-factor model of sleep, which attempts to predict sleep onset and sleep termination. This APS experiment presents data which addresses two of the factors in Webb’s schema — his third factor will be examined in the next experiment; see later. According to Webb, there are 3 primary variables which determine sleep onset. These are listed overleaf.

- | | |
|----------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------|
| 1. Sleep Demand | This is a 'time variable' which indexes the length of wakefulness that precedes sleep. |
| 2. Circadian Tendencies | This indexes the likely "time of occurrence of sleep" within the 24-hour matrix; the sidereal time of sleep (eg. sleep tendency at 1500 h. vs. 2000 h.). |
| 3. Behavioral Facilitators & Inhibitors | Behavioural factors which promote or inhibit sleep (eg. effects of boring and interesting stimuli). |

Sleep behaviour is said to be a function of:

$$\text{Sleep Demand} \times \text{Circadian Tendencies} \pm \text{Behavioral Facilitators \& Inhibitors}$$

These primary variables are modulated by species differences, individual differences, age and organismic states. This experiment has thrown light on the 'circadian tendencies' of sleep propensity (afternoon trough vs. evening circadian peak), and behavioural facilitators or inhibitors (boring vs. interesting stimuli). On the basis of the three 'observable' primary variables listed above, sleeping behaviour can be predicted. Webb has used the occurrence of afternoon napping to illustrate the point:

" If there is a high sleep demand level (up the night before), and it is midafternoon (there is good evidence of a circadian effect at this time), and the lecture is not eliciting high attending behaviour (behavioural facilitation), a nap is predicted " [ref:163].

The sleep latency during the APS period will be low compared to the early evening, and this knowledge is neatly fashioned into Webb's model. However, this APS experiment has shown that the potency of a sleep facilitator, like boredom, changes throughout the day — such that boredom effects produced during the afternoon will be quantitatively different, to any boredom effects generated in the early evening. Webb's equation treats the behavioural facilitator in a strictly additive manner; either adding or subtracting the situational component as if it were a fixed value. It clearly fails to recognise that there is a strong *interaction* between the behavioural component and the circadian component: the former is not merely supplemental to the latter.

Nevertheless, the usefulness of Webb's formulation lies in its ability to integrate antecedent and concomitant variables ¹⁶³. This APS experiment lends further credence to his approach, which incidentally, is historically rooted in Borbely's two-factor model of sleep ^{77, 164}.

Mood

This experiment also uncovered significant changes to mood, which were particularly pronounced in the afternoon, around 1500 h. During the APS peak, subjective reports of 'tiredness' and feelings of being 'muddled' and 'inefficient' were most prominent.

All the subjects were sedentary and relatively inactive throughout the experiment. Therefore, the reports of tiredness must be due to central processes in the brain rather than to neuromuscular fatigue. The role of the nervous system in 'mental fatigue' has been associated with illnesses such as myalgic encephalomyelitis and the fatigue syndrome, 'neurasthenia' ¹⁶⁵. The drop in arousal that descends with the APS, (and during laboratory induced boredom) can cause fatigue. When arousal is sub-optimal, the individual is faced with two principle choices according to O'Hanlon ¹⁶¹ :-

- i. He may leave the environment and engage in more alerting or arousing behaviours.
- or, ii. If he is 'constrained' to a particular environment when sleepiness intrudes (e.g. the climatic chamber), then he must expend 'effort' in order to maintain an acceptable arousal set-point. Effort is defined as an "internally generated process that increases arousal when the available sensory stimulation is insufficient". The expenditure of mental effort in order to maintain basal arousal probably gave rise to the 'fatigue' reported during the APS.

The subjective reports of being 'muddled' and 'inefficient' can be viewed from the perspective of Barmack's ideas on boredom. He sees boredom (or low arousal) as: "...a state of conflict between the tendency to continue and the tendency to get away from the situation" (ie. revert to sleep)^{see 116}. In this 'approach-avoidance' case, the reversion to sleep is at the heart of the conflict. The constraints of the situation and the developing conflicts ultimately gives rise to frustration ^{120, 167} and mental confusion – a scenario likely to foster reports of feeling 'muddled'.

These subjective reports of being muddled, and feeling tired must be linked to the APS. If the fatigue was simply due to 'overload' — a concept that is widely held ¹⁶⁸ — then we should expect the evening group to express greater levels of fatigue, simply because they experience greater daytime cognitive and visual loads, brought about by their accumulated hours of prior wakefulness. This was not the case, as the afternoon group were primarily affected. A further reason to see these subjective mood states as adjuncts or artefacts of the APS can be gleaned from the fact that, like the APS, they too are transient in nature. Just like the constellation of other APS effects, they also disappear when the APS window draws to a close.

Temperature

A common experience in warm rooms is the onset of drowsiness. For that reason temperature was manipulated — firstly, to add substance this folklore notion and secondly, to lower arousal as part of the strategem to unmask the APS. The effects of room temperature, based around the extremes of ‘thermal comfort’, were modest and constrained. Sleepiness— as measured by the battery of sleep scales and performance tests— was not affected; even in a warm room. There were limited effects to mood, heart rate and body temperature. Summarised below are some of the findings.

Table 2.23 The combined effects of warm (29°C) and cool (18°C) ambient temperatures

1.	SLEEP SCALES:	All the self-report sleep scales, (SSS, ADS, VAS, LMQ [sleep dimension]) failed to reveal any temperature related changes.
2.	PERFORMANCE:	Reaction time was not impaired or improved as a result of temperature manipulations.
3.	MOOD:	The subjective mood scale (LMQ), revealed growing feelings of “Lethargy” and “Tiredness” under warm ambient temperatures. A cool room made subjects feel more “Energetic”.
4.	HEART RATE:	A warm room produced greater cardiac slowing in the evening, whilst a cool room gave rise to slowing in the afternoon.
5.	BODY TEMPERATURE:	Aural temperature was depressed in the cool chamber, relative to the warm chamber; though this was not significant (p=0.07). i.). The 24 subjects assigned to a ‘boring’ environment had a significant drop in body temperature relative to the 24 subjects in the ‘interesting’ condition’ [see fig. 2.29]. ii.). The 24 subjects in the afternoon group maintained a high, but steady body temperature. A similar temperature range was initially recorded for the 24 evening subjects, but after 2100 h. the body temperature fell dramatically [see fig. 2.31].

The constant monitoring of aural temperature confirmed that the thermal challenges presented in the climatic chamber, were well within the bounds of normal physiological adjustment capability. Physiological distress was not a confound in this experiment.

Despite the fact that body temperature was recorded under contrasting thermic loads, the normal circadian rhythm of body temperature was not masked. The pattern of body temperature remained true to the traditional circadian course. Essentially, body

temperature rises after awakening, and peaks during the late afternoon to early evening, before dropping steeply towards a 0400 h nadir¹⁶⁹. The temperature data collected from this experiment concurs with this classic trend. There was a broad acrophase extending between the late afternoon and early evening. There was also a dramatic downturn in temperature which occurred after 2100 h. — this is almost identical to the sharp 2100 h. temperature fall, plotted by Colquhoun for 70 naval ratings¹⁰⁰. Similarly, Masaaki Hattori et al. have also found tympanic and rectal temperatures to fall rapidly 3 hours prior to sleep onset¹⁷⁰. This fall in evening temperature is linked to a lowered sweating threshold and raised vasodilation; the converse is true for the afternoon¹⁷¹.

In a timely review article, Deborah Sewitch examined the literature on thermoregulation at sleep onset¹⁶⁹. As part of her assessment, she distilled common points of agreement which emerged from the diverse literature. These points were then summarised in tables as definitive statements, reflective of current opinion. I have drawn 3 such statements from her list which are of direct relevance to the present study. These statements from Sewitch, are reproduced below — the claims inherent in the propositions are to be juxtaposed with the results of this APS experiment.

Statement 1: A fall in body and/or brain temperature accompanies sleep onset in humans regardless of where it intersects the daily CTR (*circadian rhythm of body temperature*).

This study has found sleepiness to be particularly acute for the afternoon group, notably at 1500 h. Moreover, afternoon sleepiness was exacerbated under conditions of boredom. According to the above summary statement from Sewitch, the afternoon subjects should display a fall in body temperature as an indicant of sleepiness. Figure 2.31 does not exhibit any marked fall in afternoon body temperature as predicted above. It could be argued, that a fall in absolute value and amplitude did actually occur, but, as it also coincided with the rising arm of the temperature curve, this sleep induced thermo-downregulation was offset by the compensatory rise in circadian temperature.

Sewitch also examined the effects of low and high ambient temperatures upon sleep. This has obvious bearing to the current experiment where the interplay between ambient temperature and the APS was examined. Listed on the next page are two statements which summarises the relevant empirical facts on this issue.

Cool Room	
Statement 2:	The lower the ambient temperature at which the subject goes to sleep, the larger the drop in rectal temperature following sleep onset, and the lower the temperature level at which it will plateau.

Warm Room	
Statement 3:	Sleeping in a warm or hot environment has consistently <i>failed</i> to demonstrate any drop in rectal temperature following sleep onset. Rectal temperature remains relatively constant across the entire sleep period.

Data from this experiment would tend to confirm the validity of the above statements. Figure 2.37 below, is a plot of the afternoon subject's aural temperature (the graph is abstracted from fig. 2.32). It indicates that under the soporific conditions which prevail in the afternoon, it is the cooler chamber which causes the greatest depression in body temperature, with the low set-point maintained throughout [see statement 2]. In contradistinction, afternoon subjects in the warm chamber failed to show any drop in body temperature (in two hours of high ambient temperatures, aural temperature rose by less than 0.1°C). This finding is in step with statement 3 above. As a caveat, it must be remembered that subjects in this APS experiment were strictly forbidden to nap in the chamber; so sleep onset *per se* did not occur. Nevertheless, the subjective reports of sleepiness, cardiac slowing and RT impairment, are tangible indicators of cortical dearousal and the presence of a 'sleepy brain', close to, or on the verge of, sleep onset.

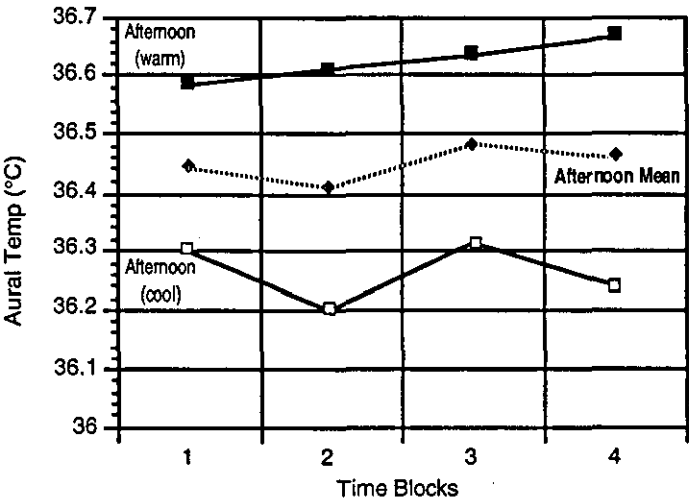


Figure. 2.37 Aural Temperature: 24 afternoon subjects in warm & cool ambient conditions

Aural thermometry as employed in this experiment, proved a sensitive measure to changes in body temperature. Using this method, it was found that the temperature of subjects in the warm chamber rose only slightly above those subjects in the cool chamber (fig. 2.30). Brain warming has been positively correlated with performance in certain tasks. In a review by Grether (1973), encompassing over 50 experiments conducted in heat, simple reaction time was found to improve with heat – a putative indication of higher neural transmission speeds during warmth^{see 173}. In this experiment, RT was not improved in the warm chamber – but then, the ambient temperature was not high enough to raise core temperature above the steady state. Work reviewed by Hancock has underscored the fact that vigilance tasks are only affected if core deep body temperature is challenged^{see 173}. An extensive review of the effects of temperature upon RT was conducted by Teichner in 1954^{see 142}. He found that RT remained unchanged in ambient temperatures spanning -45°C to +45°C. A recent contribution to the debate by Enander (1990), has acknowledged the difficulty in trying to correlate temperature with performance¹⁷³. This temperature–performance debate is beyond the remit of this current discussion – it is a discourse that is well represented in the literature related to ergonomics (eg. Mc.Intyre¹⁴², Fox¹⁷⁴, Enander¹⁷⁵). In sleep research, we are more concerned with ‘body’ temperature than ‘ambient’ temperature. Kleitman argued that “the higher the body temperature, the better was the performance”^{see 176}. This debate has acquired a certain redundancy in the light of Blakes work¹⁰⁷ and refutations by Colquhoun¹⁰⁰; the latter author prudently observed that the post-lunch dip in performance was not accompanied by a drop in body temperature. Rhythms in performance and core temperature generally appear to match each other, but it is a coincident phasing, which does not warrant extrapolations of causality¹⁷⁷. Their synchronicity should raise no surprise – most rhythms are beholden to the most powerful environmental zeitgeber, light. Recent work on photic stimulation implicates light in the circadian organisation of body temperature, performance and alertness¹⁷⁸.

A final point concerns the issue of fatigue. According to the mood questionnaire (LMQ), subjects in the warm chamber experienced increased ‘lethargy’ and ‘tiredness’. A cool room, by contrast, made subjects feel more ‘energetic’. These results which purport to show negative affect in heat, are very similar to the findings presented by Nelson et al.¹⁷⁹. They were interested in finding out how different ambient temperatures could produce fatigue, even when the task engaged in, was not physically demanding.

One hundred and forty-four subjects were given a sedentary task: they had to write stories in response to pictures from an assortment of TAT cards. This was carried out in a climatic chamber that was either warm, neutral, or cool. Their choice of 30°C for the warm condition was very close to the choice of this experiment (29°C). Fatigue was gauged with questionnaires. Their results showed that warm conditions significantly

elevated reports of tiredness and fatigue. Interestingly, their cool conditions (13°C) produced a decrease in tiredness and fatigue: a finding which corresponded well with the results of this APS experiment. The fact that non-strenuous, sedentary tasks, with minimal metabolic costs could produce such fatigue, tends to implicate psychological or “cognitive mediation”¹⁷⁹. In a study involving 1,800 air force personnel, working in a warm climate, MacPherson (1949) found a high incidence of ‘tropical fatigue’ which led to falling efficiency^{see 142}. In most cases fatigue was caused by psychological factors.

The subjective state of ‘fatigue’ that results from heat, can be adaptive. In nature, high temperatures are most likely to occur in the early afternoon – at the time of the APS. If sleep does indeed contribute to the downregulation of brain temperature and/or energy conservation, then the escalating fatigue would make further activity aversive, and so foster sleep onset. Fatigue thus serve as a “behavioural facilitator”, which promotes the withdrawal to sleep. In a similar mode, Dinges has argued that high temperatures *per se*, do not directly encourage sleep (cf. current results); infact, heat disrupts and fragments nocturnal sleep. But, in a roundabout manner heat can serve as a behavioural facilitator to sleep. In the tropics, heat brings about the cessation of work during the afternoon – the resulting inactivity and boredom presents an opportunity to sleep.

Cardiac Changes

Next to temperature, the monitoring of cardiac activity is one of the most popular physiological measures used to index psychological change. It has direct relevance to sleep research. We know that there are distinct changes to the cardiovascular system immediately before – and during – sleep. Essentially, there is a gradual slowing of the heart rate from drowsiness to deep sleep. For this experiment, the activity of the heart was gauged as the number of beats per minute (hereafter referred to as heart rate (HR)).

The first thing to notice from the data is that there is a universal slowing of the HR across all conditions, from the first half-hour to the last half-hour. This cardiac slowing could be due to three contributory factors, acting either singly or collectively :-

1. Diurnal Variation..... Part of a genuine (rhythmical) diurnal change to HR.
2. Postural Effects..... The resting or sedentary posture enhances HR slowing.
3. Experimental Treatment.. An effect of the cognitive and thermal manipulations.

I will now discuss each of these explanatory constructs, in the order presented above. The APS hypothesis would predict a transient slowing of the HR as an adjunct to the sleepiness rhythm. However, a large number of studies suggest that HR changes may be due to an independently programmed rhythm, and the diurnal slowing seen during the time of the APS, merely coincidental. The diurnal variation in HR (b/min) was

examined by Kaleque, using 7 female dayworkers¹⁸⁰. Five measures were taken between 0800 h to 1530 h. In a clear trend, HR was seen to climb from a low morning value, and peak at 1400 h., before declining sharply by 1530 h. In a comparable study, the HR of 22 air-traffic controllers, 20 factory personnel and 24 university personnel was profiled by Lille et al¹⁸¹. They found that HR declined in the morning, but rose after 1100 h. and peaked between 1300 – 1400 h. A sharp decline then began. Similarly, in an extensive review, Smolensky found HR to peak at 1300 h. before declining sharply. The emergent consensus, thus puts the peak of HR at 1300 – 1400 h. before a downturn begins (until 1800 h.). Unfortunately, most of these studies ceased monitoring at 1800 h. These results have a direct bearing upon this APS experiment. The afternoon group displayed an almost linear fall in HR, in accord with the afternoon decline reported in the above studies.

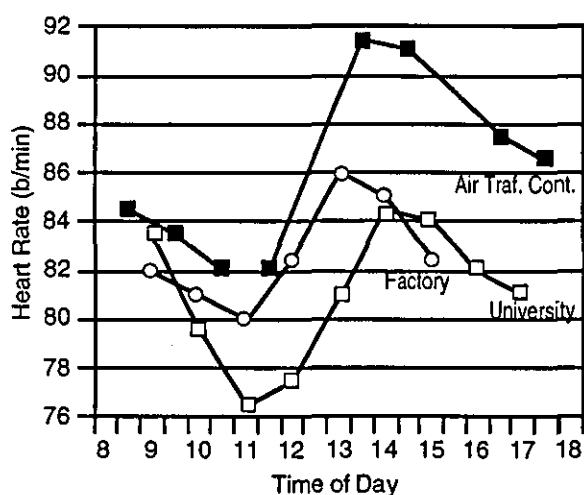


Fig. 2.38 HR Changes in various professions [181]

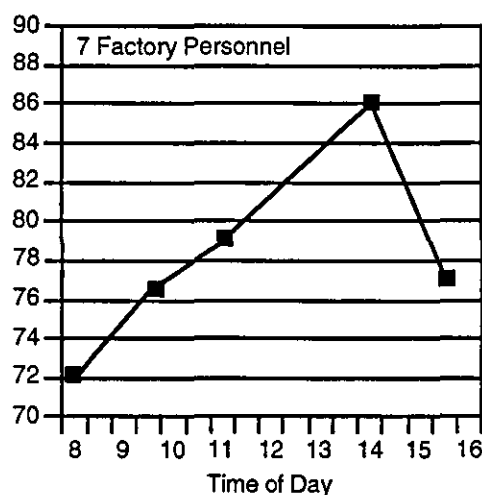


Fig. 2.39 HR in cigar factory workers [180]

If the afternoon decline continued beyond 1800 h., then the 24 evening subjects should pick up the trend. However, the HR pattern of evening group was almost identical to the afternoon group; with a high HR to commence, and followed by a steady, but parallel decline. This might indicate that a secondary peak also existed in the evening. According to a recent study by Taillard et al.¹⁸², the peak in HR depends on the chronotype of the subject ('morning' versus 'evening' types). They found that Intermediate types exhibit two peaks, one in the afternoon (~1500 h.) and one in the evening (~2000 h). This could explain the elevated evening HR peak, prior to the inevitable nightly decline. Subjects in this APS experiment were not screened for circadian typology; but it can be reasonably assumed that at least 50% of these subject were of the Intermediate type (in a random sample of 48 subjects [identical N to this experiment], Posey and Ford found an M-I-E ratio of 12-24-12)^{see 159}. This explanation of two peaks, with almost identical rates of decline in the afternoon and evening, accommodates the data, but is highly speculative and lacks credulity.

A more plausible explanation of cardiac slowing relates HR to the amount of physical expenditure. The young subjects in this experiment remained seated in the climatic chamber throughout the 3-hour experiment; they were in effect physically inactive. Can exogenous factors like the periodic activity/rest patterns affect HR? This question was addressed by Cugini et al.¹⁸³ in an experiment which monitored the 24-hour changes to HR and blood pressure in young and elderly subjects, during periods of rest and activity. In the context of this experiment, only data from the young subjects will be considered. In a repeated measures design, 13 young adults (8m/5f) had their HR and BP recorded every 15–30 minutes. During the first phase of the experiment, subjects were inactive — after awakening from a 12-hour nocturnal rest, they remained supine in bed. This condition was contrasted with the second phase in which subjects followed a normal diurnally active day (lights on 7.00 am – 11.00 pm). For the ‘resting’ subjects, the following changes to HR were evident: i. Mesor decreased. ii. Amplitude decreased and, iii. Acrophase delayed. Thus, there was clear evidence to show that physical activity was “associated with consistent changes to rhythmometric parameters.”¹⁸³

The mean HR of the sedentary subjects in this APS experiment was 76.4 (sd 9.7): this figure compares well the mean HR of 77.0 (7) reported recently by Ewing et al. (1991) in a sample of immobile orthopaedic in-patients¹⁸⁴. The influence of postural variations on cardiovascular activity cannot be underestimated. The implications for the current experiment are considerable – it could mean that the observed HR slowing did not reflect the true diurnal course of cardiac activity, but instead, was a manifestation of the sedentary condition.

One of the most important points to emerge from the cardiac data can be seen in figure 2.26 which plots the interaction between ‘Time of Day’ x ‘Subject Interest’ x ‘Time Blocks’. It tells us that the slowing of the HR — perhaps a result of circadian control and/or the seated posture — also has a strong psychological component. The graph shows that exposure to cognitive boredom during the afternoon will produce the greatest slowing of HR. If cardiac slowing is an index of sleepiness; then this group must be the sleepiest. Significantly, the other objective measure, reaction time, also shows the afternoon-bored to be the most impaired; see figure 2.9. The fact that we see a differential cardiac response to ‘cognitive’ and ‘time-of-day’ factors would mean that HR slowing is not entirely mediated by physical activity.

The main effect of ‘Subject Interest’ also showed that the bored group as a whole, experienced significant slowing of HR than subjects in the ‘interesting’ condition. The mean HR of 73.6 (sd 8.9) for the bored group closely approximates to the HR of 74.2 (7.9) which Thackray et al. reported for a group of subjects monitoring a radar display

with infrequent signals (a task deemed extremely boring by half their subjects)¹²⁴. Other laboratory studies confirm that generalised psychological arousal will elevate HR; for example, a laboratory maths task or addressing an audience¹⁸⁵. A minority of studies claim that boredom does not effect HR *per se*¹⁶⁷, or more controversially, that boredom *increases* HR. The latter claim was made by London et al¹²⁹. Their study has two principle methodological flaws. Firstly, in their experiment which lasted one hour, subjects were given boring stimuli in the first 30 minutes, and in the next 30 minutes, the *same* subjects were aroused with an interesting task. The work of Poulton¹⁴⁶ and Millar¹⁴⁷ would indicate that such a design is highly prone to asymmetric transfer effects. To go from one extreme mood state directly to a second mood extreme, would incur some carry-over effects. The testing time was too short for these effects to be extinguished. The second point of contention is more serious. The methodology used to induce boredom was questionable —subjects were required to write the letters “cd” over and over, for a full 30 minutes. The frustration and stress inherent in such an unnatural task is likely to foment much irritation, and so elevate HR instead of lowering it. The task used to generate ‘interest’ prompts similar reservations: subjects had to write stories based on pictorial TAT cards – this ‘arousing’ task depressed HR. Their findings do not conform to the accepted view that boredom depresses HR. Although cardiac measures are useful indexes of change, Roth et al. have warned on the limitations of using HR to explain psychological state, “when different emotions can produce indistinguishable physiological effects.”¹⁸⁶

The activity of the heart during the day is controlled by both the sympathetic (SNS) and parasympathetic (PNS) systems. Elevated SNS activity directly increases HR by the promotion of catecholamine release and adrenal activity¹⁸⁷. A fall in HR may be caused either by an increase in PNS activity or reduced SNS activity. Recent work by Malpass et al.¹⁸⁸ would tend to favour the latter explanation as the amount of circulating catecholamines is particularly low during deep sleep when the HR is also low. Evidence linking the SNS to boredom is mentioned by Parasuraman — a catecholamine decrease during a vigilance (boring) task is evident¹⁸⁹. Swedish researchers, Åkerstedt and Fröberg, have positively correlated the excretion of urinary catecholamine with alertness¹⁹⁰. It is interesting to note that the peak in adrenaline excretion occurred close to 1300 h.: note, this is also the time of the HR peak as discussed earlier.

To summarise, the HR slowing observed in this study may be due to the circadian aspect of cardiac activity. In other words, the timing of this experiment, may have been coincident with the falling arm of the diurnal HR curve. Again, HR slowing could be due to catecholamine modulation. The marrying of HR slowing and declining alertness during the period of the APS seems attractive. Nevertheless, there are grounds for doubt – the evening group (ostensibly improving in alertness) also show a drop in HR.

We can however account for the analogous trends between the afternoon and evening groups if we endorse the role of physical expenditure and postural influences. However fig. 2.26 provided the best evidence of a possible link between developing drowsiness (prominent in the 'afternoon-bored' group) and falling HR. The basal HR of the group in the 'interest' condition was higher throughout, as was their self-reports on alertness (SSS, VAS and ADS). Thayer notes that the best physiological measure which could match self-report measures was HR; it produced change in the expected direction (cf. self-report) with a 95% success rate¹⁵⁹.

3

Post-Prandial Action & Sleep Reduction

3

Post-Prandial Action & Sleep Reduction —Triggers of the APS?—

3.0 Introduction

3.1 Food and the APS

Most people can recount from experience a form of lethargy and sleepiness that often follows a heavy afternoon lunch. This post-prandial sleepiness has been recognised and written about since ancient times. Hippocrates (460-377 BC.) observed:

"Some who lunch, although lunch does not suit them, forthwith become heavy and sluggish in body and mind, with a prey to yawning, drowsiness and thirst" (see ref: 198).

Aristotle was more expansive, insofar as actually providing an explanation for this effect. He suggested that food in the stomach produced warm vapours which rose towards the head, so inducing "fits of drowsiness" ^{see 17}. The sleepiness described by these great sages occurs in the early afternoon. It is also a time that is very close to the afternoon break for lunch. A causal relationship between afternoon sleepiness and the ingestion of food naturally emerged, and even today, continues to influence modern scientific thought on the subject.

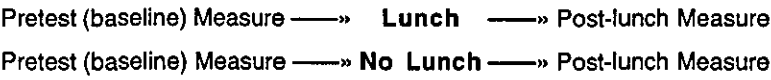
However, recent findings from sleep research and chronobiology has challenged these time-honoured assumptions which ascribe causality to ingested food. Among the current expositions, the most noteworthy derives from the theoretical models of Roger Broughton^{7,8}; EEG work by Gagnon and de Koninck^{53,54}; studies of napping behaviours in isolation chambers by Campbell and Zulley^{45,49}, and fieldwork by Hildebrandt et al¹¹⁰. Findings from these studies point to an endogenous 12-hour rhythm of sleepiness, that is independent of food intake. An extended account of their work is provided in Section 1. The evidence presented has been so impressive, that I have abandoned the term 'Post-Lunch Dip' – a misnomer that wrongly perpetuates the centrality of food. In reaction, I have adopted a simpler, and less ambiguous term, namely, the Afternoon Pressure for Sleep (APS).

Nevertheless, even if we assert that the APS is part of an endogenously programmed rhythm, we cannot preclude the influence of exogenous stimuli. Wever has written extensively on the ‘behavioural aspects’ of circadian rhythmicity, acknowledging in particular, the interaction between external and internal stimuli in the control of biological rhythms¹⁹⁹. Both are important. The daily interval for the afternoon lunch break is a social zeitgeber, but more importantly, the food consumed has a direct (exogenous) influence on the biochemistry of the ‘interieur milieu’. The afternoon lunch can greatly alter endocrine functioning (eg. elevating cortisol levels), modulate physiological parameters (eg. heart rate, core temperature, blood glucose) and neurotransmitter concentrations (eg. serotonin levels via dietary tryptophan)²⁰³. Clearly, the ‘state’ of the individual must be altered after lunch as a result of these psychophysiological perturbations. This exogenously provoked change can interact with the endogenous low point of the APS to produce quite measurable impairments.

3.2 Post-lunch Impairment: Empirical Evidence

A. Meal Dependent

The most important recent investigations into post-lunch impairments, has been carried out in Britain. The seminal studies of Blake in the 60’s, inspired a keynote paper from Christie and McBrearty in the 70’s, and this was followed by a series of papers during the 1980’s by Smith, Miles and Craig et al. based at the MRC unit at Sussex University [see refs. below]. Generally, the Sussex group employed an ‘A-B-A’ experimental design:



Examples of food-related impairments found by these workers are listed below; table 3.

Table 3.0 Studies showing meal-dependent impairments during the afternoon.

Study	Post-lunch Impairment	Pre/Post-lunch Test (hr)
Christie, M., McBrearty, E. (1979)	Nowlis Checklist (Mood)	12-1230 / 1430
Craig, A., Baer, K. et al. (1981)	Perceptual Discrimination	11-1200 / 1300-1400
Smith, A., Miles, C. (1986) ^a	Cognitive Vigilance (memory loaded)	1045-1315 / 1315-1545
Smith, A., Miles, C. (1986) ^b	5-Choice Serial Reaction Time	1045-1330 / 1315-1545
Smith, A., Miles, C. (1986) ^c	Bakan Vigilance Task (10 mins)	1150 / 1410
Smith, A., Miles, C. (1987)	Visual Search Task (memory loaded)	0900-1200 / 14.30
Craig, A., Richardson, E. (1989)	Letter Cancellation - omission errors	1115-1145 / 1315-1345
Smith, A. et al. (1991)	Search Task - increased errors	1150-1250 / 1350-1450

See reference list 200-207 for bibliographic details.

Reflecting on the decades work, Smith (in 1990) noted that post-lunch impairments and falling alertness were principally highlighted in tasks demanding sustained attention²⁰⁸. The dip reportedly occurs whether lunch is eaten early (eg. 11.00 h.)^{see 209} or late (eg. 13.15–14.30 h.)²⁰⁴. The work of the Sussex group has provided much needed ‘hard’ data. However, there are certain methodological shortcomings in their experiments:

- i. They failed to control and standardise the afternoon lunch in terms of size and calories. The meals were simply described as a standard 3-course meal available from the daily range of the university refectory. By the authors own admission: “This meant that different subjects had different meals (.. size and content)”²⁰⁴.
- ii. Surprisingly, there were no restrictions placed on smoking, even though the authors quote a study purporting to show the dampening of post lunch effects by smoking²⁰³.
- iii. Tea and coffee was also allowed after lunch — in one case, just 10 minutes before recording baseline measures²⁰⁵. This could have inflated the pre-meal alertness scores and caused the post-meal decrements to appear striking by comparison, so incurring a type I error (falsely rejecting the null hypothesis). One of the authors (Smith et al., 1990)²¹⁰ later investigated the effects of caffeine on the ‘post-lunch dip’. Caffeine improved performance both before and after lunch.
- iv. Morning baseline tests (upon which later comparisons were to be made) were not always carried out at the same time (eg. 10.45 – 12 noon)^{203, 204}. In some cases scores improved at the later baseline timing. It would seem that the pre-test baseline measures may have been confounded by having dissimilar testing times.

Unfortunately, it is very difficult to comment on the contributions breakfast makes to post-lunch impairments, due in part to the paucity of research data. During the morning, alertness rises rapidly in concert with the circadian upswing, which according to Craig²⁰⁹, serves to mask any breakfast effect. Colquhoun has noted (anecdotally) that one is typically less sleepy after breakfast compared to lunch¹⁰⁰. Research by Connors and Blouin²¹¹ examined the effects of breakfast in young school children (9 to 11 yrs). In a vigilance task, children who ate breakfast showed a marked improvement in performance (greater consistency with fewer errors), and an arithmetic task improved considerably by mid-morning — these comparisons were made with a no-breakfast condition. The value of these findings for researchers interested in the effects of afternoon lunch is debatable. In the case of breakfast, the duration of prior nocturnal fasting (eg. 12 hours in children), makes a direct comparison with the effects of afternoon lunch somewhat unrealistic²⁰⁹.

B. Meal Independent

As mentioned earlier, a number of investigators (Broughton, Gagnon and De Koninck, Campbell and Zulley) are now questioning the long-standing view that post-lunch impairments are wholly dependent on the ingestion of food. If the somniferous properties of food are so potent, then why, it must be asked, do we not see equivalent post-breakfast or post-supper dips. It is important to point out that even some of the studies listed in table 3 produced results which indicated that the afternoon decrements were not entirely dependent on the consumption of lunch. Using a cognitive vigilance task, Smith and Miles²⁰³ found a time-of-day effect that was meal independent. Again, in another report, the same authors²⁰⁴ noted that the motor component and error rates in a 5-choice RT task, showed a time-of-day effect that was not related to food intake.

In a letter cancellation task, Christie and McBrearty²⁰⁰ found no evidence of a post-lunch impairment — a finding in line with Blake's earlier work^{see 100}. Blake divided 36 subjects into 3 equal groups. Group 1 received lunch at 1100 h; Group 2 lunched at 12 noon and Group 3 lunched at 1400 h. A 30-minute letter cancellation task was carried out at 1300 h.

Table 3.1 Letter cancellation as a function of lunch-hour and lunch abstinence [ref:100]

.....	Lunch Time		
	11.00 h	12.00 h	14.00 h
No. of letters cancelled at 13.00 h.	1268 (2 hrs. Post-lunch)	1311 (1 hr. Post-lunch)	1313 (No Lunch)

From the table above, it would appear that almost identical scores were obtained by subjects who lunched at 12 noon and their counterparts who had not yet lunched by the 1300 h test. Parenthetically, the poor performance at 1100 h. was not explained.

Sophisticated studies by Stahl et al.²¹², employing polysomnographic recordings, did not find evidence to attribute post-prandial sleepiness to food intake. A more recent EEG study into the post-prandial effect was carried out by Zammit et al. (1992)²³⁴. Twelve subjects were given lunch at 1145 whilst 9 controls abstained from food. From 1214 to 1515 h all subjects were seated in a recliner chair. The majority of subjects in both groups (food and no-food) exhibited EEG-defined sleep episodes. Clearly, the high incidence of sleep in the no-food condition would indicate that a lunchtime meal is not imperative for the promotion of afternoon sleepiness (though the duration of sleep was found to be longer in the luncheon group). To see whether ingested food induced

sleepiness, Carskadon and Dement,^{235, 166} gave subjects evenly divided portions of food every hour, during a day long confinement to bed (from 0815 to 1930 h). Regular MSLT's showed that sleepiness was not an invariable consequence of ingested food. The shortest sleep latency in this 'constant (feeding) routine' occurred at 1530 h, forcing the authors to conclude that the endogenous biphasic circadian tendencies of sleep, rather than food per se, was responsible for afternoon sleepiness.

Using the well-known Wilkinson Auditory Vigilance Task, Horne³¹⁴, could not establish any meal-related decrements: paradoxically, big meals *improved* response speed (though at a cost to accuracy). Other examples which failed to find pre-lunch and post-lunch differences include Richardson and Craig, Simonson et al.^{see 209}, Kanarek and Swinney (1990)²¹³, and Millar et al. (1992)²⁴⁶.

3.3a. Meal Size : Naturalistic Studies

Any mention of 'afternoon sleepiness' or 'napping' in general conversation, often invites personal tales of '*the Sunday afternoon snooze*'. Stories of Sunday afternoon napping abound, usually related with much ebullience and humour; perhaps a measure of its social acceptability. In most instances, the anecdote will contain some passing reference to the precursory Sunday lunch, so imputing a possible post-prandial effect. Traditionally, in Western cultures, church-going families usually gathered after worship on Sunday afternoons, with extended family members for a larger than normal '*Sunday Lunch*'. If such reports of post-prandial lassitude are indeed attributable to an especially large meal, then it is important to establish the veracity of this Sunday overindulgence. Should this customary pattern of Sunday eating be true, then any inference which draws causal linkages between 'meal size' and 'sleep' may contain some substance.

Recent work by De Castro (1991, 1992) has examined the weekly rhythms of meal patterning and meal sizes^{214, 215}. In a study that boasts ecological validity, 323 paid volunteers kept strict diaries of food intake over 7 consecutive days, recording details of meal content, time of eating, whether dining alone or in company, etc. The data revealed two recognisably different patterns of food intake during (i) weekdays (Monday–Thursday) and (ii) weekends (Friday–Sunday). Friday was grouped with the weekend as it showed great similarities with both Saturday and Sunday.

During the weekend, the overall food intake was significantly higher ($p < 0.05$) than weekdays. Moreover, the average meal size (at a given sitting) was significantly higher at weekends; fig. 3.1. Interestingly, the weekend days (Friday–Sunday), did not differ significantly among themselves, though the trend was for Saturdays (and not Sundays)

to show the greatest food intake and meal sizes. The large weekend food intake was not due to increased meal frequency, but rather, to an increase in the the duration of meals.

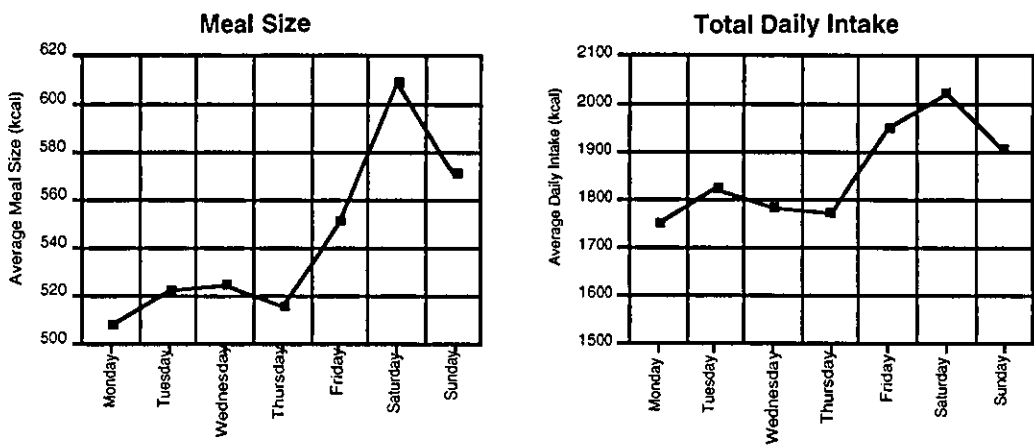


Figure 3.1 Patterns of food intake over the course of 1 week; N=323 [ref: 214].

There was also a significant increase in the presence of other diners during the Saturday and Sunday meals compared to other days. Estimates of the post-meal stomach contents indicated that a greater number of calories was present in the stomach at the end of each weekend meal (Saturday and Sunday) than at any other day. Another recent report has also highlighted this weekend acrophase in food consumption (Mejean et al, 1992²³⁶).

The large weekend food intake has been put down to 2 principal factors:

1. The duration of each meal (time at table) was longer at weekends.
 2. The number of other people present during the meals was significantly higher.
- This social facilitation extends meal duration so increasing food intake.

In summary, the work of De Castro would suggest that the pattern (and size) of food intake on Sundays was not significantly different to that of Fridays or Saturdays. In fact, there was a trend highlighting a greater consumption of food during Saturdays. Yet, we do not find equivalent lay reports of a Friday or Saturday napping propensity. Perhaps, the afternoon lunch is the largest meal of the day on Sundays, whereas the evening meal represents the largest meal on Saturdays. On the other hand, there are alternative explanations to account for Sunday afternoon sleepiness: for example, *boredom* (Sunday closing restricts leisure), *opportunity* (no occupational/social constraints) or *recuperation* (from Saturday evening excesses).

As a means of exploring the relationship between the size of meals and sleepiness, the purportedly large Sunday lunch provides a useful (naturalistic) model. The findings discussed above would suggest that we cannot attribute the Sunday afternoon lassitude to the ingestion of a large meal alone.

3.3b. Meal Size: Laboratory Studies

The field study discussed above has ecological merit, and like the epidemiological approach, it can tell us much about the incidence and distribution of a biological phenomenon in the populous. To understand the mechanisms and processes underlying a particular biological event, we need the kind of methodological control that can only come from laboratory manipulations, free from the haphazard and confounding factors present in the environment.

In a recent investigation of meal size and post-lunch changes, Smith et al. (1991)²⁰² were able to control several variables independently. Thirty five female volunteers were divided into three groups; one received a normal-sized lunch (n=12); a second group received a big lunch (n=11); and a third group a small lunch (n=12). They were interested in monitoring changes to the following parameters: mood, performance (attention), and blood pressure/pulse.

Lunch was staggered between 1230–1330 h. About an hour after consuming lunch, mood and performance tests were conducted. Subjects rated themselves as feeling feeble, bored, dreamy and generally less alert after lunch. While the negative effects of lunch were apparent, the investigators could not find any differences due to the size of the meals. In the case of performance measures (tests of attention), a large meal impaired performance accuracy but the size of lunch had no effect on the speed of response. Physiologically, pulse rate increased after lunch, but the size of the preceeding meal had no bearing on this parameter. In the main, whilst the effects of lunch were apparent on most measures, the authors concluded that meal size did not contribute to altering the effects of lunch, particularly in the case of mood, performance speed and cardiovascular function.

An earlier study by Craig and Richardson (1989)²⁰⁷ investigated the effects of lunch size on cognitive impairment. Essentially, they were interested in the interaction between meal size presented in the laboratory (large vs. small) and the size of meals normally eaten by subjects in 'daily life'. For instance, could a heavy lunch adversely affect someone who habitually eats only a small lunch. They recruited 12 subjects who habitually ate a heavy lunch, and 12 subjects who were habitual 'light lunchers'. On one occasion subjects were given a big lunch (eg. 1380 kcal) and on another occasion, a small lunch (eg. 260 kcal). The tests used to gauge the effects of these meals included subjective scales and a 30-minute letter cancellation task. Summarised below are some the major findings.

- i. Alertness ratings, as measured on a subjective bipolar scale (alert—drowsy) produced no main effect or interactions. However, there was a tendency for

- alertness to improve (and tension ratings to fall) after a big lunch. A small lunch produced an opposite trend.
- ii. The speed of letter cancellation showed no difference between the pre-meal morning baseline condition and the post-lunch testing, around 13.30 h.
 - iii. There were significant changes to the accuracy of performance. Errors of omission increased after a big lunch and dropped after a small lunch.
 - iv. The effects seen in (iii) were especially dependent upon the habitual meal size. If the experimental meal size differed from the subject's habitual meal, then the impact to performance was significant — plainly, a big meal caused greater impairment to a subject who normally ate a small meal. See graph below.

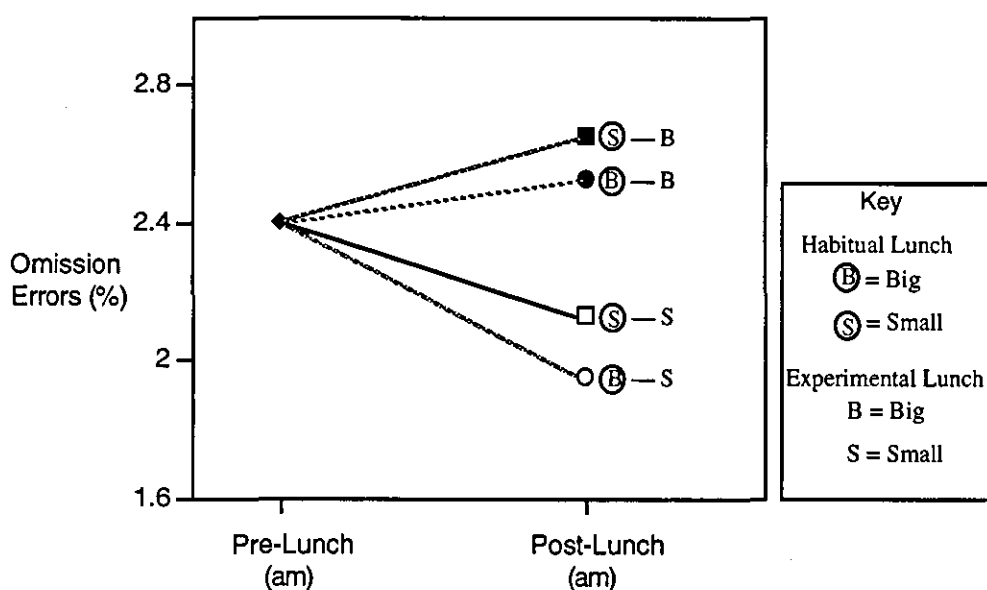


Fig. 3.2 Performance after habitual & exptl. meal sizes: relative to pre-lunch covariate [ref: 207].

The graph above appears impressive, but careful interpretation of the data is required. A look at the habitual light lunchers may illustrate the point: during the big meal challenge, they consumed over 5 times the amount of calories as compared to their small lunch condition. In spite of this huge difference, the increase in errors resulting from the big experimental lunch, was in the order of 0.6%. In reality, a 500% increase in calories: a meal close to the ceiling of normal satiety – produced a performance change of less than 1%. So, although habitual small lunchers showed a ‘statistically significant’ change after eating a big meal, it is a matter of debate whether a change of less than 1% is at all ‘clinically significant’ with any real-world impact.

The authors concluded that food has a direct influence on post-lunch impairment. However, they recognised that the habitual meal size can modulate any effects produced by the experimental meal — this would indicate that a secondary, endogenous process

was involved in post-lunch effects. Recently, Smith (1990)²⁰⁸ has reiterated that the post-lunch impairment has “both a food-related component and an endogenous one.”

There are very few studies that have addressed this issue of lunch size and post-lunch effects. One recent study by Kanarek and Swinney (1990)²¹³ has found that a late afternoon snack that is rich in calories improves performance significantly, compared to a snack that is low in calories. By contrast, Zammit et al.(1990)²¹⁶ failed to find any difference in sleep latency or architecture, following a test meal that was either high or low in calories. The whole area is controversial and requires further investigation.

3.4 Meal Constituents

There is a growing body of evidence to suggest that certain food constituents can affect behaviour through their action on brain chemistry. The nutrients present in food are directly involved in the synthesis of certain neurotransmitters such as serotonin and dopamine. As these neurotransmitters are precursor-dependent, their levels will be influenced by the nature of dietary intake. Sleep researchers are particularly interested in brain serotonin which has been linked to sleep and mood modification. Serotonin is made from the amino acid, tryptophan, which is available in dietary protein. However, a protein rich diet also contains several other large neutral amino acids (LNAA's) which all compete for the carrier molecule transporting these amino acids to the brain. Paradoxically, a meal rich in carbohydrates will (through the action of insulin) facilitate the uptake of all the amino acids – except tryptophan. Without the competitive hindrance of the other LNAA's, tryptophan gains easy access across the blood-brain barrier, thereby raising serotonin levels.

When administered in its pure form, tryptophan reportedly increases sleepiness. In a review of 43 studies examining the sleep inducing properties of tryptophan, Hartmann (1986) found the results to be ‘mixed’ in a normal population, though in sleep-disordered patients it may have a therapeutic role²¹⁹. Oral doses of tryptophan in ranges of 4–15g have been reported to cause nausea as one of its unpleasant side effects (see review by Borbely)²²⁰. In an earlier investigation, I examined the effects of an ‘acceptable’ tryptophan dosage (2g)^{221, 222}. I also attempted to amplify the effects of tryptophan by coupling it with a meal rich in carbohydrates (comparisons were drawn with a tryptophan + protein dense meal). Tryptophan significantly increased sleepiness and lethargy, and slowed down the motor component of a reaction time test. Interestingly, the carbohydrate rich meal with a lactose placebo increased sleepiness whilst a placebo/protein-rich meal improved alertness ratings. Similarly, Spring et al.^{223, 224} found a carbohydrate rich meal alone was sufficient to increase sleepiness compared to a protein meal, though there were demographic differences. Specifically,

females felt sleepier, whilst males felt calmer after a carbohydrate dense meal. Reaction time was not affected, but a performance measure (dichotic shadowing) did produce decrements after a carbohydrate lunch in the case of older subjects (≥ 40 years). Importantly, these effects which emerged at lunchtime failed to occur after an identical morning meal (breakfast). Similar work by Liebermann et al.²²⁵ looked at the effects of a carbohydrate dense meal (particularly rich in starch) versus a protein dense meal. Sleep and mood scales (SSS and POMS) revealed no significant differences between the meal types, but the trend was for the carbohydrate meal to be more soporific. Simple auditory RT was slower 1.75 hours after the carbohydrate meal, and a digit symbol substitution test was affected 3 hours after the meal. These results clearly indicate that ingested food can cause sufficient deactivation to bring on sleep, but it is an effect greatly modulated by certain macronutrients.

3.5 Methodological Issues and Research Needs

In the study by Lieberman et al.²²⁵ (see above), subjects were given a carbohydrate meal that was designed to be particularly dense in starch (120g of wheat starch presented in the form of a 'bread-like' food). The protein group were given cold turkey to eat. In their experiment, Spring et al.²²⁴ gave subjects 304g of sherbet as their carbohydrate rich meal, whilst subjects in the protein group were given 227g of trimmed turkey breast to consume. In a strong critique of these and related studies, Leathwood²²⁶ has questioned the realism of "a whole meal consisting entirely of one macronutrient"; underlining its artificiality by pointing out that "300g of turkey (without bread) is a fairly unusual meal." A significant charge, especially when one recalls that 103 subjects in the study by Spring et al. were between the ages of 40–65, a cohort likely to experience a lunch of 304g of sherbet as a gastronomic insult. Granted, these are edible diets, but they are more concerned with carbohydrate and protein 'loading', and bear no description to 'meals' in the normal sense. The physiological and postabsorptive sequelae of a meal cannot be addressed in isolation from its sensory and cognitive aspects. This point was highlighted by Westerup-Plantenga et al. (1992)²²⁷ when they showed that diet-induced thermogenesis was more likely to occur if subjects were given an 'unfamiliar' meal to eat, compared to a 'familiar' menu. Similarly, studies on taste hedonics have shown that the 'palatability' of meals can affect mood states²³³.

I intend to examine the effects of meals in relation to the APS. However, in view of the reservations outlined above, I will adopt a 'naturalistic' approach; presenting meals which are familiar and not alien to the average menu enjoyed by young adults. These meals will not be geared towards macronutrient loading, instead, I will vary the size of the meals: the reasons for which will be discussed below.

Recent studies germane to the issue of post-meal deactivation, have been reviewed and listed in table 3.0. The majority of these studies have examined the post prandial effect by comparing a 'lunch' versus a 'no-lunch' condition. This design fails to appreciate that eating a midday meal, or lunch, is an important aspect of our behavioural repertoire — in many ways, the daily regularity of lunch means that we may be 'conditioned' to expect food at certain times, and in preparation, many digestive processes may be automatically activated. By depriving subjects of lunch on one experimental day (cf. 'no-lunch condition') these conditioned responses will not be extinguished, and the automatic timer controlling gastric and endocrinological secretions will 'switch on' regardless. To confound matters further, the irksome sensation of hunger in the no-lunch condition, may prove to be a source of unwonted alertness. Early mention of the conditioning effects of lunch was made by Colquhoun over 20 years ago^{see 100}.

Much evidence to back up Colquhoun's assertion has been gathered from recent animal studies; many of which detail the entrainment of biological rhythms by scheduled meals — for reviews, see Mistlberger, 1990;²²⁸ and Boulos and Terman, 1979²²⁹. Both groups of reviewers quote the pioneering observation made by Richter who noted that his rats were stirred from inactivity and became highly active prior to the arrival of scheduled food. Such anticipatory behaviours was said to reflect the stomach's 'clock-like functioning.' Boulos and Terman list over 20 physiological parameters which are known to be synchronised by meal timing, for example, maltase, sucrase, and leucine aminopeptidase. It is worth reiterating that these changes were not directly reliant on food intake, but occurred in 'anticipation' of food. Can we extrapolate these findings across the species divide? According to Martin²³⁰ the conditioning processes which operate in animals are frequently inherited by Man as well. Like other vertebrates, humans also make anticipatory responses to expected meals²³⁸. Recent work on humans by Jansen et al. (1992)²³¹ on preparatory salivation to food, draws on the conditioning model. A more positive example is that of insulin secretion, in particular *cephalic insulin* (see Woods, 1991²³²) which is released just before the ingestion of food in response to external zeitgebers "normally associated with food." In the main, there is now sufficient evidence to suggest that scheduled meals serve as effective zeitgebers (and entrainers) for a host of biological rhythms. In view of these findings, I will not include an acute 'no-lunch' group in the the current experimental design. Only volunteers who normally have lunch will be used; and more importantly, all subjects will be given a 'familiar' lunch of some kind. However, the food dimension that I intend to manipulate will be lunch size.

The biobehavioural effects of food may be the result of food composition, gastric distension, thermic effects, calorific value, etcetera. Ultimately, food affects brain chemistry. There is thus the scope for a dose-response relationship, despite the

“putative low pharmacological activity of foods or dietary constituents.”²¹⁷ If food is indeed a sedative agent, then dose effect studies (so familiar to pharmacologists) will allow us to quantify the effect in a systematic way²¹⁸.

3.6 Amplifying The Effects of Food

The behavioural effects of whole foods are subtle in the main – unlike drugs, which have a quick and direct impact upon synaptic transmission²²⁵. The difficulties inherent in studying meal effects are well embodied in this metaphor evoked by Spring et al.:

“..whereas malnutrition researchers seek to detect the effects of a major blow to the head, many contemporary diet-behaviour researchers are looking for sequelae to a slight tickle to the scalp.”²²³ Plainly, the effects of meals taken in realistic quantities will not necessarily produce gross behavioural changes, and the subtle effects that do occur, will have to be evinced with extremely sensitive measures. Alternatively, we could potentiate or enhance the effects of food, thus improving our chances of detecting the post-prandial afternoon trough.

In keeping with the directive for a naturalistic experimental approach, we can consider two acceptable agents which may serve to augment meal-effects; namely: i). alcohol and ii). partial sleep loss. Both are facets of everyday modern living, enjoyed and endured, respectively, oftentimes with unforeseeing noon-time consequences. In this respect, the effects of post-lunch alcohol has attracted much recent interest. As a possible research strategy, we could combine meals of varying sizes with alcohol and graded sleep loss. However, such an all-inclusive design with so many powerful variables, could be construed as unduly consummate, guaranteed to safeguard an effect; or to paraphrase Millar: with enough grapeshot, a hit is surely on the cards¹⁹⁷. Therefore, to reinforce the effects of food, only one of the two possible challenges (ie. alcohol or sleep loss) will be used; and making the appropriate choice will require careful forethought.

1. **ALCOHOL** : Due to its well-known sedating properties, alcohol remains a favourite evening hypnotic amongst insomniacs. Importantly, alcohol is also drunk during the daytime for a myriad of social reasons, but notably, it also forms a congenial accompaniment at the dining table. In studies by Horne et al.,^{196, 239} the effects of alcohol during ‘lunchtime’ were contrasted with its effects during the evening. Performance measures (a vigilance task and driving simulator) showed clear afternoon decrements, highlighting the interaction between ethanol and circadian sleepiness. In a comparable placebo controlled study, Roehrs et al. found a greater sleep propensity with the MSLT following an afternoon alcohol dose, as against a similar dosage given in the early evening²⁴⁰. In sharp contrast to these studies, Rouhani et al.²⁴⁵ did not find alcohol given

in the afternoon to be a particularly good hypnotic. Following an intake of alcohol, subjects were allowed a 90-minute sleep at 14.30 h.. Polygraphic recordings after a baseline condition (day 1: no alcohol) versus an experimental condition (day 2: afternoon alcohol) showed no significant changes to sleep latency, though alcohol did in fact increase sleep disruption. Essentially, the sleep-inducing property of alcohol was found to be muted. On closer examination, we see that in the study by Horne et al., the alcohol beverage was preceded by a 'light lunch' (cheese roll), whereas in the study by Rouhani et al, lunch was more substantial (eg. steak, french fries, salad). In a recent report by Millar et al.(1992)²⁴⁶, the adverse effects of alcohol were found to be considerably dampened by the presence of recently ingested food. A large meal effectively impedes stomach emptying, and thus alcohol absorption. Moreover, food constituents can also conspire to hasten the early oxidation of alcohol. This interaction between alcohol and food – insofar as food is able to undermine and vitiate any ethanolic effect – is a great confound, and one which only adds to the complexity of data interpretation. After all, the primary interest of this study remains the post prandial effects of food, untainted by any biochemical interactions with a CNS depressant.

Whilst we may rule out the use of an alcohol–food combination, as a way of unmasking the APS, we could, as others have done, couple alcohol with sleep loss.²⁴¹⁻²⁴⁴ However, when alcohol has been combined with sleep loss, the effects on alertness has not always been consistent: reports suggests both synergistic and antagonistic effects^{242, 244}. Further concern arises from the data of two separate studies^{241, 242} which purport to show that alcohol combined with sleep loss, suppresses the time-of-day variations in MSLT scores. Presumably, the soporific effects generated by an alcohol–sleep loss coupling, is so omnipotent as to thwart any subtle diurnal changes to alertness.

As the purpose of this study is to investigate the fluctuations in basal sleepiness over the course of the afternoon, it is clear that the aforementioned variable permutations, (eg. alcohol + food, or alcohol + sleep loss) will not prove satisfactory. In this scheme to potentiate the effects food using realistic variables, we have yet to consider the coupling of food with sleep loss.

2. PARTIAL SLEEP LOSS : Lunchtime alcoholic intake is an indulgence enjoyed by a small but distinct subset of the working population. By comparison, a much larger percentage of the populous are reluctant abstainers, not of alcohol, but of recuperative nightly sleep. For example, adolescents, students, career-professionals, nursing mothers, disorientated shift-workers, moonlighters and junior doctors, all experience restrictions to their daily sleep quota. Historical data suggest that we are sleeping less than the early part of this century²⁵⁶ and incidentally, changes have also been recorded

in respect to the nature and size of contemporary meals²⁴⁷. In siesta cultures, people tend to eat their evening meal at a much later hour, and the warmer evenings encourage leisure activities to extend into the small hours. This shorter nocturnal sleep in conjunction with an afternoon meal – for example, of the rich Mediterranean sort – may be precursors to the post-prandial somnolence we call the siesta. Surprisingly, there are no empirical laboratory investigations into the effects of primary sleep length and meal size, apropos afternoon sleepiness. In a questionnaire study by Hicks et al.²⁴⁸ a simple attempt was made to relate sleep length with eating behaviours. They found that college students who were habitual short sleepers (n=31), did not adhere to the normal 3 meals per day, and were more prone to frequent snacking behaviours. Apart from this nominal survey, loosely aimed at relating REM sleep curtailment to dietary modulation, no useful data exist to link prior sleep duration with post prandial sequelae. A working hypothesis of the current experiment will argue that an ‘everyday experience’ such as sleep loss is well suited to amplify post prandial sleepiness. Support for this premise comes from Colquhoun¹⁰⁰, who, in a review, quotes data from three studies which showed diurnal effects to be most pronounced in cases of sleep loss.

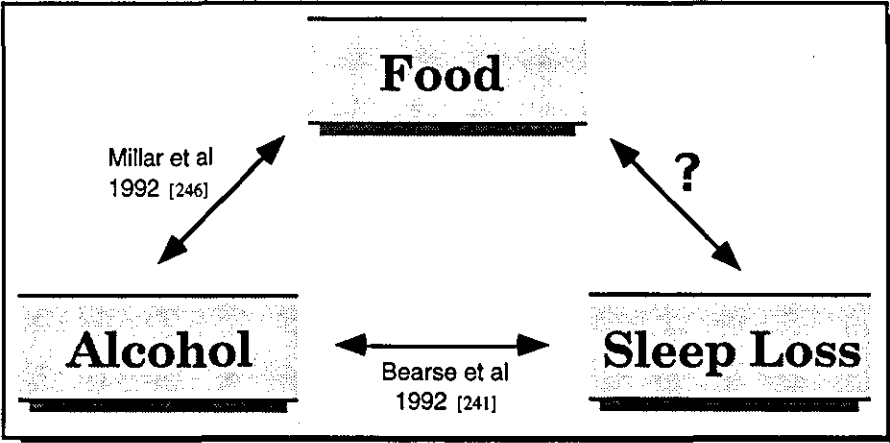


Fig. 3.3 Common exogenous variables present in the afternoon: their interactions & the APS

In summation, the figure above highlights three of the most common factors in the environment likely to exacerbate the APS. Food has been identified as a principle component in this schema, and, in light of the above review, it has been decided to amplify the effects of food with sleep loss. Remarkably, the interaction between food and sleep loss has yet to be explored in this context. Ecologically, these two variables are ever-present in daily life and common to the majority population. The widespread and chronic complaint of sleep loss in society, in conjunction with the ubiquitously eaten noon-time lunch, may represent modalities of physical triggers most likely to promote APS expression. A fuller discussion of partial sleep loss and its applicability to the present study now follows.

3.7 SLEEP RESTRICTION

3.7.1 Basal Sleep

Before discussing an 'abnormal' curtailment to sleep length, we need to clarify our understanding of what constitutes a 'normal' sleep duration. As yet, we have not identified a universal sleep quota, that is to say, an optimal sleep duration that adequately satisfies each and everyone. Indeed, the prospect of ever arriving at a wholly acceptable 'Sleep RDA' (cf. nutritional RDA's) is remote. Nevertheless, it is quite remarkable that people from diverse cultures will average between 7 to 8 hours of sleep each night.²⁵⁹ If sleep falls below 6 hours, or extends beyond 9 hours, then one is particularly disposed to ill-effects. MSLT studies by Carskadon and Dement²⁶⁰ have shown that subjects whose normal nightly sleep ranges from 7.5–8.0 h will be able to "maintain a stable [daytime] sleep tendency." Any further curtailment to this sleep quota results in a cumulative rise in sleep propensity. The authors were drawn to speculate on the existence of a homeostatic process in which the daytime sleep tendency formed part of a control mechanism which modulated the nocturnal sleep length.

Table 3.2 Examples of average sleep length per night

Study	Year	Sample	N	TST/Night
Present Study *	1992	Undergraduates	97	7.6
Koulack, D et al.	1992	Undergraduates	162	7.5–8.0
Carskadon, M et al.	1991	School Seniors	581	6.9
Acebo, C. et al.	1991	Undergraduates	98	7.0
Hicks, R.A et al.	1989	Undergraduates	734	6.8
Carskadon, M et al.	1989	Adolescents	1,268	7.6
Carskadon, M et al.	1988	Adolescents	276	7.3
Billiard et al. †	1987	Young Soldiers	35,478	7–9
Browman, C.P et al.	1977	Working Adults	365	7.1
White, R. ‡	1975	Undergraduates	89	7.4

Bibliographic details 249–255 / * Survey conducted by author / † see ref: 22 / ‡ see ref: 256

In a historical comparison, Webb and Agnew²⁵⁶ found that the sleep length of adolescents during the year, 1910, averaged 1.5 hours more than a comparable sample surveyed in 1963. More recently, Hicks et al.²⁵² examined the sleep reports of 1,489 college students collected during 1978 and compared the data with the sleep logs of 734 students collected 10 years later, in 1988. They noted that in the intervening 10 years,

the average sleep length fell by 30 minutes per night; from 7.30 h (± 0.86) to 6.82 h (± 1.04). Clearly, there has been a steady erosion to the length of our nightly sleep. Our modern lifestyle, with its exacting demands of work and school, and the advent of a 24-hour leisure industry has made inroads into the precious hours we allocate for sleep. Although we have the freedom to choose our bedtimes, we have little choice about waking up: it's a societal decision beyond our control²⁵⁶. These points are vividly made in a recent novel where the central character (a sleep researcher!), opines to an audience:

"Our society, sacrificing to the gods of efficiency [and] profitability, transforms sleep into a vulgar physiological function... sleep is no longer a pleasure, it has become a chore.. The obligation to get up at a precise time prevents our having any carefree relationship with sleep... Limiting sleep to a particular span of time is like trying to put eternity into cans. Sleep must open onto the infinite, it must not be interrupted by the shrill ringing of an alarm clock." "The Empire of Sleep" H. Blanc 1992 [ref: 261]

3.7.2 Indications of Insufficient Sleep

A. Subjective Complaints

If sleep is partially curtailed during the nocturnal hours, then sleepiness will intrude during the waking hours, in a bid to redeem the nightly shortfall. In a combined study involving 11 sleep disorders centres 5,000 case histories were pooled and examined by Roehrs et al²⁶². The most frequent complaint presented at these clinics was excessive daytime sleepiness (EDS), and one of the most common causes of EDS (after apnea and narcolepsy) was 'insufficient night sleep'. Indeed, there is now a clinical term for this: "The Insufficient Sleep Syndrome", a disorder common to all ages but widely seen in adolescents as pointed out in a recent tome on paediatric sleep medicine (Sheldon et al 1992)²⁶³. From longitudinal studies, Carskadon has mapped the developmental trends in adolescent sleep patterns²⁶⁴. During the high school teenage years, the night-time sleep length declines as bed-times become later and later. In the senior years of school, bed-times range from 10–11 pm., and by the age of 18, the average bed-time for the college freshman is around 1.20 am. With this change in sleep habit, comes the ubiquitous problem of daytime sleepiness.

In one study, Carskadon surveyed 3,000 high school students on sleep satisfaction²⁶⁵. Although most averaged 7–8 h of sleep, astonishingly, 58% still complained that they did not get sufficient sleep. Likewise, a survey of 1,000 students by Agnew^{see 256} found that under a third of subjects awoke in the morning "feeling fresh and rested", and a third of respondents experienced waking difficulties every morning. Another example comes from the data of Hicks et al.²⁵², in which the changing sleep profile was traced for the decade spanning 1978–1988. Alarming, they found a two-fold increase in the reports of sleep dissatisfaction (1978=24.4%; 1988=53.4%).

B. The Weekday–Weekend Sleep Anomaly

An indirect example of our socially imposed restrictions to physiological sleep can be gleaned from a comparison of typical sleeping habits during weekdays and weekends. Both anecdotal and survey data point to a significantly longer sleep duration at weekends. The enticing conclusion that this observation invites, is one of sleep reclamation, that is, the weekend sleep indulgence serves to recoup the sleep, lost during the working week.

Carskadon and Mancuso²⁵⁴ noted that the weekend sleep profile was characterised by a delay in bed-time and rise-time. In a further study²⁵³ they examined data from 1,268 students. This sample was particularised by having a fairly regular sleep habit, in that their sleeping times were not constrained by part-time work (apparently 59% of students engaged in part-time work after school). Presented below is the sleep duration of these and other students, during; i). weekdays and ii). weekends.

Table 3.3 Mean Sleep Duration (+sd); Weekdays vs. Weekends. Refs: 253, 250, 266

Sample	Weekday TST	Weekend TST	Difference
1 School-Girls	7.42 h (1.25)	9.15 h (1.95)	1.73 h
School-Boys	7.70 h (1.17)	8.92 h (1.98)	1.22 h
2 Senior-Girls	6.85 h (1.00)	8.64 h (1.51)	1.79 h
Senior-Boys	6.94 h (1.00)	8.78 h (1.51)	1.84 h
3 First Year US Undergraduates	6.67 h (0.85)	8.15 h (1.15)	1.48 h
4 Loughborough Undergraduates	7.60 h (0.93)	8.71 h (1.15)	1.11 h
<div><div><div>1 Adolescents (N=1,268) Ref 253</div><div>2 School Seniors (N=581) Ref 250</div></div><div>Key</div><div><div>3 Undergrads'; 1st Term (N=165) Ref 266</div><div>4 Undergrads' (N=97) Authors Survey</div></div></div>			

In the largest sample of over 1,000 subjects²⁵³, there is a clear trend for longer weekend sleeping. The table above also includes data from other surveys, and again, the findings all tell the same story. Finally, a clinical example from Roehrs et al.²⁶² provided data on

59 patients with EDS, where the diagnosis implicated insufficient sleep. It was found that during weekends patients averaged 8–8.5 h of sleep, whilst during the working weekdays, sleep was curtailed to around 6.4 h, a nightly deficit of 2 h when compared to the weekend.

C. Ad Libitum Sleep

Given the freedom to sleep without any time restrictions on waking, most subjects will readily seize the opportunity and extend their sleep. A practical demonstration of this tendency was provided by Webb and Agnew²⁵⁶. During 3 consecutive days, 16 male volunteers were entrained to sleep at 11.00 pm and arise at 7.00 am. However, on the fourth day, the subjects were not awoken and allowed to wake up at their 'own pleasure'. On this 'ad-lib sleep night' they averaged 126 minutes more sleep than on their previous controlled nights. This increased sleep length was taken by the authors as clues to the real sleep need, or at any rate, as an indictment of the sleep restrictions that is usually forced upon nocturnal slumber.

Essentially, the aim of this section (3.7.2) has been to point out the pervasive nature of sleep restriction in society. A large percentage of the caseload presented at sleep clinics involve daytime sleepiness, and often, insufficient sleep is indicated. Waking complaints, as well as the incongruous weekday–weekend sleeping pattern, point to an erosion of the desired sleep quotient. Bluntly, we would like to sleep more; but cannot, due to occupational and social demands.

In the light of obvious socio-cultural pressures, Webb and Agnew²⁵⁶ have provocatively posed the question: "Are we chronically sleep deprived?". On the basis of their experimental results, they answered in the affirmative. Although, biologically, we may only need a "core sleep" of 6 hours (cf. Horne¹⁷), it would appear that our psycho-physiological wont is disposed toward more sleep than the current '7.5 h' average.

Surprisingly, in experiments where sleep is gradually reduced over weeks (eg. Johnson et al.²⁶⁷, Friedman et al.²⁶⁸, Mullaney et al.²⁶⁹, Horne et al.²⁷⁰) subjects will on follow-up, adopt a regimen of less sleep (eg. 1–1.5 hrs less than baseline) even months after the experiment has finished — tentative indications of the role played by 'learning and adaptation' in sleep. There exist good laboratory evidence for a 6 hour 'core sleep', but there also exist compelling field data for a much greater sleep need. Perhaps, as Carskadon has suggested, "the answer lies somewhere between these two extreme positions."²⁶⁵

3.7.3 Aspects of Sleep Restriction

Now that the universality of sleep loss has been identified, there is a need to consider the practical issues related to human sleep reduction, as the various sleep curtailment strategies could, by itself, modify any deficit. For instance – in the light of the planned experiment – do we use ‘acute’ or ‘chronic’ sleep reduction, and do we institute sleep reduction by ‘delaying bedtime’ or advancing ‘wake-up time’? These are not simply methodological details, they can in fact modulate the effects of sleep restriction.

We have accumulated considerable data on ‘total sleep deprivation’ – beginning in 1894 with the work of Manaceine and peaking during 1955–1975; the so-called “golden years of sleep deprivation research” (see Jovanovic²³⁷). However, our knowledge of ‘partial sleep deprivation’ remains comparatively scant. By the time of his major review in 1965, Wilkinson could only identify 4 studies into partial sleep deprivation¹⁰⁹. Yet, partial sleep deprivation is unquestionably a commoner form of sleep loss than total sleep deprivation, and for that reason, studies into partial sleep deprivation must be considered extremely relevant; a point also echoed by Wilkinson^{109, 271} and Johnson⁹². Still, in salutation, we need to credit May Smith, who in 1916 conducted the first study into partial sleep deprivation²³⁸ — in the light of what we know today, her results were to prove highly prescient (see later).

Notwithstanding the undoubted paucity of research in this area, there do exist a growing corpus of data from which answers to important questions may be adduced. In the process of designing the current experiment, we need to articulate methodological concerns that are likely to confound any sleep reduction procedures. These have been alluded to earlier, and are summarised below :

1. Which is the appropriate mode of inducing partial sleep loss – a delayed bedtime or early rise time?
2. Is there any basis for choosing ‘acute’ sleep reduction over ‘chronic’ or gradual sleep reduction?
3. Are there distinct ‘afternoon’ changes associated with previous-night sleep loss? That is, are there any manifestations of sleep loss which are quintessentially predisposed to the afternoon period.

The design of the current experiment will emerge from a review of the issues outlined above. I will assess each of the three issues separately; beginning with point 3.

A. SLEEP RESTRICTION AND THE ‘AFTERNOON IMPACT’

Undergraduates, school seniors, and young adults in general, are notoriously sleep deprived individuals – their legendary reputation of ‘late-nights’ and sleep curtailment, are amply borne out by numerous sleep studies ^{250-255, 264-266}. In a survey of 116 apparently normal-sleeping undergraduates, Carskadon ²⁶⁵ found 87% of her sample complained of afternoon sleepiness, whilst 60% actually admitted to “having fallen asleep”. Similarly, in a survey of 430 college students, Evans et al. ³¹ also found 60% of their sample succumbed to afternoon sleep. It would seem that the problems associated with previous night sleep reduction are particularly prone to flare up in the afternoon. More specifically, the problem is most likely to manifest itself in the guise of increased afternoon napping. Unsurprisingly, undergraduates are renowned nappers ^{24, 272}. Individuals who catnap in response to a nocturnal sleep deficit have been labelled as ‘replacement nappers’ [see page 14]. Such ‘compensatory’ napping is said to typify the majority of student naps.

Using data gathered from sleep diaries, Hawkins²⁷³ categorised students into ‘frequent’ and ‘occasional’ nappers (he also recorded the average nap length). During a 14 day profile, sleep logs revealed that those subjects with a high proportion of naps (and longer nap length) also showed a significant reduction in their primary sleep length. In a similar vein, a study of 194 Nigerian undergraduates found an 80% incidence of nap taking, where crucially, the average nocturnal sleep length was noticeably low at 6.5 hours ²⁷²; though in this case, acculturation to ‘siestas’, may also play a part.

Essentially, people who habitually restrict their nightly sleep will exhibit signs of daytime sleepiness, especially during circadian low-points. Another characteristic of this group is that their nocturnal sleep efficiency (TST/TIB) is usually very high. For example, the 59 patients of Roehrs et al. ²⁶² (whose diagnosis of ‘EDS’ was attributed to insufficient sleep) all showed sleep efficiencies in excess of 90%. Plainly, they try to make the most of what little time they have in bed! Using MSLT’s, the authors also noted that the sleep-restricted patient group “showed a biphasic pattern of sleep tendency, minimal in the morning and evening, and maximal at midday.” On the basis that high sleep efficiencies are indicative of sleep restriction, Levine et al.²⁷⁴ identified college students with low sleep efficiencies ($\leq 85\%$) and those with high sleep efficiencies ($\geq 95\%$). MSLT latencies were clearly much shorter in subjects with a high sleep efficiency. Importantly, the differences were “most pronounced in the afternoon.”

<u>Sleep Efficiency</u>	<u>Mean Afternoon Latency</u>
Low (85%)	14.06 mins
High (95%)	8.90 mins

From these few examples it would seem that sleep restriction, by itself, is sufficient to unmask afternoon sleepiness, without any assistance from postprandial boosting by food. However, there are cases where acute sleep restriction on its own, cannot always guarantee an afternoon sleep propensity the following day. In many instances, depriving subjects of sleep can actually '*improve*' mood ratings and daytime alertness (see later). Recently, Soldatos et al.³⁹ contrasted the nocturnal sleep duration of 10 habitual afternoon nappers (NPRS) with 10 non-nappers (N-NPRS). The TST of the two groups did not prove to be significantly different (TST : NPRS=389.5 [\pm 11.9] vs. N-NPRS=391.0 [\pm 20.4]). This finding refutes any direct association between nocturnal sleep length and napping behaviours. Likewise, a comparison between North American and Guatemalan undergraduates by Lawrence and Shurley²⁴ showed that the American students were more disposed to napping (76% vs 52%) despite obtaining significantly more sleep than their counterparts. Finally, in his classic Mexican study, Taub²¹ found the length of the afternoon siesta to correlate positively with the length of the primary nocturnal sleep, a finding which casts great doubt on the presumed 'compensatory' role of naps.

B. ACUTE vs. CHRONIC SLEEP RESTRICTION

In laboratory studies looking into the effects of partial sleep restriction, the regimens used to procure sleep deprivation have been varied. For instance, some 'acute' studies have examined the effects of sleep loss after a single night's sleep curtailment²⁷⁵, whilst others have looked at the cumulative effect of several days of sleep restriction²⁷⁶ or several months (chronic studies)²⁶⁸. The sleep architecture, and the ensuing levels of daytime impairment, that follows each of these research paradigms is not consistent. The effects of acute and chronic sleep restriction will be contrasted below. To begin with, a brief account of two 'acute' studies will be mentioned; one looks at performance changes, whilst the other examines 'EEG' changes.

i. Acute Sleep Restriction

The effects of acute sleep restriction on performance was investigated by Wilkinson et al³¹⁵. Six male volunteers were continually tested (with short breaks) between 8.00 am and 10.30 pm. over two consecutive days. The tests consisted of an auditory vigilance task and an addition test. On the evening prior to testing, subjects were allowed 0, 1, 2, 3, 5 or 7.5 h sleep; the six conditions spread over six weeks. In the auditory vigilance task there was a slight deterioration from baseline when sleep was reduced to 5 h. However, when sleep was curtailed to 3 h and below, there was a dramatic deterioration to performance. By comparison, the addition test was more resilient; impairment occurred only when sleep fell to 2 h and below. Importantly, both tests were worse on the second day of testing, reflecting the cumulative effects of sleep loss.

In an EEG study, Carskadon and Dement²⁶⁰ gauged physiological sleepiness with the MSLT. After 3 baseline sleep nights, 12 subjects had their sleep restricted to 4 hours for two consecutive nights, followed by one recovery night. As a result of sleep restriction, daytime sleep latencies were significantly lower than baseline nights; this was particularly pronounced on the second day of sleep restriction, where two-thirds of the subjects achieved pathological MSLT scores (<5 mins).

ii. Chronic Sleep Restriction

When sleep is gradually reduced (eg. by spreading 30-minute reductions over several weeks) its impact, in terms of performance decrements, is considerably diminished. In a programme of sleep reduction which lasted several months, Friedman et al.²⁶⁸ required 4 collegiate couples to gradually curtail their habitual sleep length. Following 3 weeks of baseline measures, subjects were asked to reduce their sleep by 30 minutes every fortnight (until TST=6.5 h), after which sleep was reduced a further 30 minutes every 3 weeks (until TST=5.0 h) and finally 30 minutes was subtracted in each ensuing month. After 6-8 months of this regime involving stepwise sleep reductions, it was found that performance scores did not deteriorate until sleep was curtailed to 4.5–5.5 hours, and the main factor which limited further reductions to sleep was ‘subjective fatigue’.

In another example of chronic sleep restriction, Webb and Agnew²⁷⁷ monitored 15 male volunteers who maintained a reduced sleep length of 5.5 hrs for a total of 60 days. Despite this 2.5 h sleep loss, they found no significant changes in a battery of tests: only the WAVT produced performance decrements as sleep restriction progressed, and this was dismissed by the authors as a motivational response, rather than a consequence of sleep loss per se. Johnson and MacLeod²⁶⁷ and Horne and Wilkinson²⁷⁰ were also able to demonstrate that with a programme of gradual sleep reduction, subjects could adjust to a lower sleep budget without much harm.

From the standpoint of designing the present experiment, it would appear – from this brief review – that ‘acute’ sleep restriction is the more appropriate mode by which to supplement any post-prandial effect. The time afforded by chronic or gradual sleep reduction provides ample opportunity for adjustment to occur, and any sleepiness or performance decrement normally associated with sleep restriction, will be muted. More importantly, ‘acute’ sleep restriction has greater realism in that most people commonly experience this form of sleep loss; from the executive hastily assembling a business presentation, the weekend partygoer, a nursing mother and the oft-quoted student, revising at the eleventh hour. For these reasons, ‘acute’ sleep restriction will be incorporated into the design of this experiment, and not chronic sleep reduction.

C. SLEEP REDUCTION BY DELAYED BEDTIME OR EARLY RISING

When normal, healthy individuals are required to curtail their sleep – for whatever reason – they usually choose to do this in one of two ways (depending on the circumstances): they can either; delay bedtime (DB) or advance rise-time (AR).

Shift-workers are obliged to wake up early during their early morning shift, and on the whole, this particular shift with its AR, elicits the greatest number of complaints²⁷⁸. On the other hand, delaying bedtime appears less aversive, and by all accounts is the most common mode of sleep loss; usually resulting from an evening ‘spree on the town’ or more mundane occupational pressures. The necessity to delay bedtime first arise during the middle teenage years, when school and social pressures mount. Indeed, there is a certain attraction among this cohort to ‘late nights’; Price et al^{see 264} noted that almost two-thirds of 11th and 12th graders “enjoy staying up late.”

Clodore and coworkers^{278, 279} have studied the problem of DB versus AR. In one experiment, 12 volunteers restricted their sleep by 2 hours, over two consecutive days, using the protocols of DB and AR²⁷⁸. Polygraphic data were to show that DB produced a longer TST, higher sleep efficiencies and greater REM sleep compared to AR. Similar results were reported by Taub and Berger²⁸⁰ and Tilley and Wilkinson²⁸¹. The latter authors found that a DB produced significantly more REM and stage 4 sleep, with a decline in stage 2 sleep. In view of these changes to the sleep structure, their results on performance measures were made all the more remarkable by their failure to find any differences between performance scores after DB or AR. They found that sleep restriction *per se* impaired performance, and was not modulated by the composition of the obtained sleep or the method of sleep curtailment.

Using Thayer’s subjective scales of alertness, Clodore et al.²⁷⁸ found that subjects in the AR condition were reportedly more ‘tense’ and less alert the next day, compared to subjects in the DB condition. They also found subjects in the AR condition to show a 33% increase in urinary 17 ketosteroids – a possible marker for elevated stress (though other hormonal changes could not corroborate this trend).

The conclusion to be drawn from these studies is that most individuals are ill-disposed to early rising, which appears to be more problematic than DB. There is an important element of choice involved in DB; we can generally choose our bedtime as our energies begin to flag, but waking times are usually precise, and governed by external strictures. As the subject pool for the intended study will be drawn from a campus population; it is important that the sleep protocols to be employed here, are not entirely alien to this subject group. In a survey conducted by the author, 112 students were questioned on

this point of sleep/waking preferences: twice as many students selected a DB option, given a choice of sleep restriction schedules; see table 3.4 below. Clearly, students do favour DB over the more ‘traumatic’ AR schedule.

Table 3.4 Survey of students (N=112) on sleep scheduling preferences. Authors data.

Question	
Which would you prefer if your sleep was restricted to just 4 hours?	
	<div>Bedtime</div> <div>Wake—Up Time</div>
a.	24.00 hours.....04.00 hours
b.	03.30 hours.....07.30 hours

Delayed Bedtime	Advanced Risetime
67.0 %	33.0 %
N = 112	

Carskadon and Davis²⁶⁶ monitored the sleep profiles of 165 students during their last term at high school, and their first term at university. In the transition to university life, they found an average 2–hour phase delay in bedtime to occur. In the light of such clear-cut findings, the method to be chosen for instituting sleep loss will be DB.

3.8 EXPERIMENT

METHOD

SUBJECTS

Participants were 48 university undergraduates, comprising of both sexes (22 males, 26 females) aged between 18–25 years. All subjects were normal sleepers, routinely obtaining 7–8 hours of sleep/night, with no history of daytime sleepiness, habitual nap taking or treatment with CNS medication. They were all of apparent normal weight; accustomed to moderate eating habits at lunchtime – dieters were excluded. All agreed to adhere to the experimental conditions and were paid a fee of £10.00.

DESIGN

There were two major experimental variables under investigation; ‘lunch size’ and ‘previous night sleep length’. The separate effects of gender were also considered. Subjects were randomly divided into 3 sets:

- 1. Normal Night Sleep (N=16)
- 2. 2-hr. Sleep Reduction (N=16)
- 3. 4-hr. Sleep Reduction (N=16)

The 16 members in each set were sub-divided into two equal groups; one received a large lunch (N=8), and a second group received a small lunch (N=8). This 2x3 design yielded 6 conditions.

- 1. Normal Sleep / Small Lunch
- 2. Normal Sleep / Large Lunch
- 3. 2-hr. Sleep Reduction / Small Lunch
- 4. 2-hr. Sleep Reduction / Large Lunch
- 5. 4-hr. Sleep Reduction / Small Lunch
- 6. 4-hr. Sleep Reduction / Large Lunch

Table 3.5 Six Experimental Conditions: Lunch Size vs. Previous Night Sleep Length

	Normal Sleep	2-hr. Sleep Loss	4-hr. Sleep Loss
Large Meal	8 S's (5f/3m)	8 S's (4f/4m)	8 S's (4f/4m)
Small Meal	8 S's (5f/3m)	8 S's (4f/4m)	8 S's (4f/4m)

The major independent variables included:

- i. Prior Sleep Length — 3 Levels (Normal, -2 hrs, -4 hrs)
- ii. Lunch Size — 2 Levels (Large Lunch, Small Lunch)
- iii. Gender — 2 Levels (Males, Females)

MATERIALS / TESTS

a). Questionnaires

- i. Stanford Sleepiness Scale (SSS) — See Expt. 1; page 59
- ii. Visual Analogue Scale for Sleepiness (VAS) — See Expt. 1; page 60
- iii. Post-Sleep Questionnaire — See Expt. 1; page 60

b). Apparatus

- i. Computerised RT with BBC microcomputer — A full description of this apparatus is given in section 1 (page 61). Again, the RT was given for a full 10 minutes. However, for this experiment, the software was upgraded to allow finer analysis of the scores in the manner described by Dinges^{106, 321}. In an effort to find the most sensitive ambulatory measure of sleepiness, Dinges analysed 39 performance and mood variables. The most sensitive measure was found to be the slowest 10% of scores derived from the Wilkinson Simple VRT. These slowest scores are indicative of attention deficits and were thought to represent the “Lapse Domain”. The RT scores from the present investigation, were also filtered, to tease out the slowest 10% of scores.
- ii. Actigraph – A wrist actigraph (Gaehwiler Electronic, Hombrechtikon, Switzerland) containing a piezoelectric motion sensor was used to discriminate between periods of wakeful activity and sleep³²²⁻³²⁴. This device was worn on the evening prior to the study to ensure that subjects conformed to the strict experimental ‘sleep regimens’. This electronic tag served as a psychological deterrent to any infringement of the experimental rules.
- ii. Electroencephalograph – Model: Grass 78.

Recording Montage:

- a). Electroencephalogram (EEG) at C3, referred to A2.
- b). Eye movement lead: one electro-oculogram (EOG) placed on the outer canthus of the eye to gauge the slow, rolling eye movements indicative of drowsiness; referred to A1.

c). The Multiple Sleep Latency Test (MSLT).

The speed with which a person falls asleep (sleep latency) is a quantifiable guide to underlying sleepiness, and, it is from this basic presupposition that the MSLT has evolved. The MSLT was developed during the 1970's by the Stanford group in response to the clinical needs of sleep disorders centres⁸⁶. Today, it is one of the most popular instruments that is used by researchers and clinicians to gauge excessive daytime sleepiness, achieving the status of a 'clinical standard'²⁸¹.

Essentially, subjects are given four to six opportunities to nap at 2-hour intervals throughout the day – usually between 10.00h and 18.00h. Each nap opportunity is polysomnographically monitored and restricted to 20 minutes. Within this period, the latency to the first signs of stage 1 sleep (after lights-out) are recorded. If sleep onset occurs, then the nap is promptly terminated after 3 epochs of stage 1 sleep to prevent the accumulation of further sleep; alternatively, if no sleep is evident, then for analytical purposes, the latency is recorded as 20 minutes. Detailed guidelines for practitioners of the MSLT emerged from the standardisation committees of the governing bodies of American sleep societies — the first guidelines were authored by Carskadon, 1986,²⁸¹ with the most recent clinical update authored by Thorpy, 1992²⁸².

The MSLT is carried out in a setting likely to promote sleep, ie. a quiet and darkened room. According to the basic premise of the MSLT, this setting, devoid of arousing stimuli, is ideally placed to offer free expression to the underlying *physiological sleep tendency*. This environment does not *cause* sleepiness but *unmasks* sleepiness. An individual who is excessively sleepy will have a shorter sleep onset latency: a fact which makes the MSLT a well placed instrument to monitor the effects of partial or total sleep deprivation. Carskadon has demonstrated the sensitivity of the MSLT in studies of acute sleep restriction in children²⁸³, young adults²⁶⁰ and the elderly²⁸⁴. The MSLT has also proved sensitive enough to uncover the subtle post lunch changes to sleepiness (Richardson et al.²⁵⁷, Levine et al.²⁷⁴, Carskadon et al.²³⁵), in accord with the bicircadian model of human sleep. In healthy, asymptomatic individuals, the MSLT has been shown to be a highly reliable probe of daytime sleepiness; in a 'test-retest reliability measure' it produced consistent results over two occasions separated by 4 to 14 months²⁸⁵.

The MSLT was deemed suitable for this experiment on the basis of its sensitivity to *daytime* sleepiness (cf. Sugerman et al.²⁸⁶) under conditions adjudged as *moderately* soporific (cf. Hartse et al.²⁸⁷). The major interest remained the 'ease of falling asleep' during the afternoon trough.

d). Meals

Two contrasting lunch-time menus were provided; a large meal, high in energy (approx. 5,061 kJ) and a small meal, low in energy (approx 1,516 kJ). The tryptophan content of the large meal was 0.4g and for the small meal, 0.2g. These figures are well below the 1g threshold required to produce a soporific effect (see Hartmann^{219, 288}).

Table 3.6 Dietary Menu. Two contrasting meals and their [approx.] energy value.

LARGE MEAL		SMALL MEAL	
Beef burger (2x70g - cooked wt)	2100 kJ	One Cheese Sandwich:	
Accompanying bun (66g)	840 kJ	2 Slices of bread (67g)	571 kJ
Portion of potato chips (100g)	966 kJ	Butter(1 pat)	210 kJ
Dessert (100g)	924 kJ	Cheese (30g)	504 kJ
Glass of fruit juice (200ml)	231 kJ	Glass of fruit juice (200ml)	231 kJ
TOTAL	5,061 kJ	TOTAL	1,516 kJ

PROCEDURE

Subjects were required to maintain a regular sleep-wake schedule for 3 nights prior to the experiment (sleeptime: 23.45h–midnight; risetime: 07.30h). In the 48 hours preceding the laboratory study, all subjects were required to avoid any alcoholic intake, refrain from napping and vigorous evening exercises or sport.

On the eve of the study, sleep time was specified by the experimenter. According to the random group allocation, subjects were asked to either maintain their sleep at the pre-test regimen or to delay their sleep time by 2 or 4 hours; see table below.

Table 3.7 Sleep Regimens on Night Prior to Experiment

Group	Sleep Time	Rise Time
Normal Sleep	Midnight	07.30 h
2-hr Sleep Reduction	02.00 h	07.30 h
4-hr Sleep Reduction	04.00 h	07.30 h

To ensure strict conformity to the sleep regimens outlined above, subjects wore obligatory wrist actigraphs 4 to 5 hours before retiring – this monitoring device gauges periods of activity and quiet sleep in relation to ‘real time’. On the morning of the

experiment, subjects awoke at 07.30h, and consumed a normal breakfast, though abstaining from tea or coffee which were prohibited. For the remainder of the day, further consumption of snacks or caffeine-containing drinks were not allowed.

Subjects reported to the Sleep Laboratory at 09.30h; arriving in pairs or, sometimes, groups of three. They were given a brief tour of the adjoining conveniences and departmental facilities (eg common-room with newspapers/magazines, computer terminals and quiet study rooms). They returned to the laboratory for MSLT electrode attachment — the wiring-up montage for polysomnographic recording was described earlier. Whilst one subject underwent EEG preparation (electrode attachment), the other was given a 5-minute practice session on the RT apparatus.

At 10.25h, subjects were escorted to their respective rooms and told to remove shoes and to loosen any constricting clothes. They were then ‘hooked-up’ at the bedside and were on-line to the Grass electroencephalograph. In accordance with the standard MSLT protocol, they were then taken through a series of ‘calibration’ steps designed to test the hook-up operation and signal transmission. Following the calibration tests, subjects were given the introspective sleep measures, SSS and VAS to complete.

A very dim light was switched on and directed at the floor in one corner of the room to provide a soft and muted ‘night-light’, effectuating an ambience of semi-darkness. Subjects were then asked to adopt a comfortable position and lastly instructed:

“Relax, shut your eyes and try to fall asleep”

After this instruction, the main room lights were switched off and the door closed. At this point (time zero), the MSLT began, and the time to sleep onset latency measured.

Table 3.8 The MSLT Countdown Protocol. After Carskadon [281]

MSLT COUNTDOWN	
Time Prior To Testing	Procedure
15 Minutes	Suspend vigorous physical activity.
5 Minutes	Prepare for bed; remove shoes and loosen constricting clothes.
2 Minutes	In bed hookup. Calibration Series: <i>“Lie on back, relax. With eyes open look to the right and left and then up and down. Grit Teeth.”</i>
45 Seconds	SSS and VAS.
30 Seconds	Adopt a comfortable repose.
5 Seconds	<i>“Relax, shut your eyes and try to fall asleep”</i> [Lights Out]

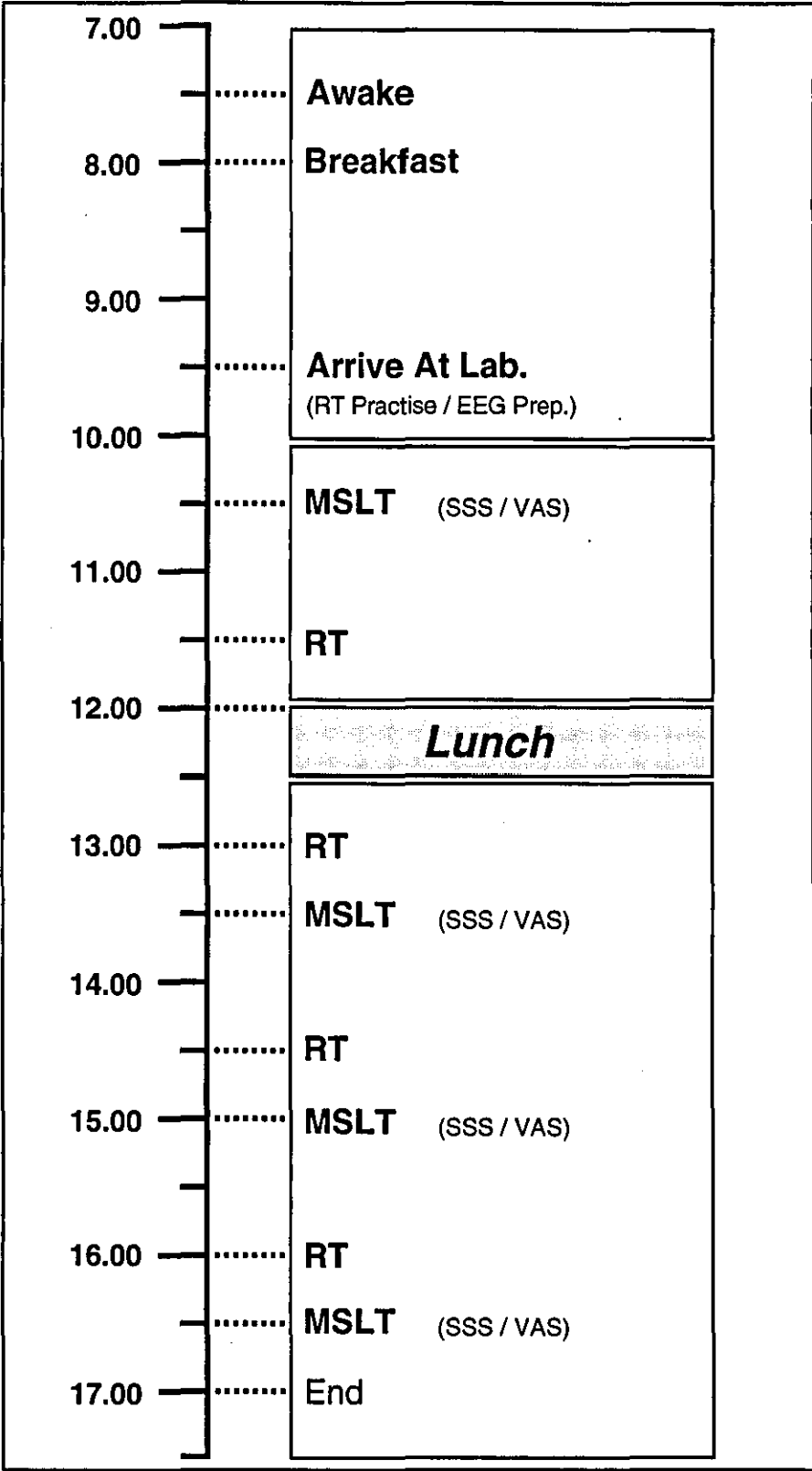
The MSLT was terminated after 20 minutes if no sleep was recorded. However, if sleep onset did occur, then 3 consecutive epochs of stage 1 sleep was allowed before MSLT termination (1 epoch=30 secs). In this case the latency to the first epoch of stage 1 was taken as the definitive sleep latency. To secure 3 consecutive epochs of stage 1 sleep it was mandatory for certain strict pre-determined criteria to be met. These conditions are summarised below.

The Three Epoch Rule

1. Three consecutive epochs of EEG trace must be classified as stage 1 — a slow trace by itself is not sufficient; there must be a virtual absence of alpha and some definite features of stage 1 sleep, eg. vertex sharp waves.
 2. For an admissible epoch, stage 1 sleep must occupy at least 75% of any 30 seconds. Isolated alpha (ie. 2 seconds in an epoch or <10%) may be scored as consistent with stage 1 and allowed under the three epoch rule.
 3. If the trace is obscured by artifact (eg. loose electrode) then score later rather than sooner, and highlight with an asterix to draw attention to possible errors.
-

After the MSLT test, subjects remained in the vicinity of the Sleep Laboratory and were allowed to pursue non-strenuous leisure activities, eg. reading, watching television and private study. At 1130h subjects returned to the laboratory for a 10-minute RT test carried out at a computer terminal. The 1030h MSLT and the 1130h RT test represented the pre-meal test battery. Following a standard noon-time lunch (large or small meal), further MSLT's and RT tests were carried out at 90 minute intervals. The experiment concluded at 17.00h.— subjects were de-briefed on the covert experimental aims, paid for their cooperation and dismissed. The experimental schedule is outlined overleaf.

Fig. 3.4 Experimental Schedule



3.9

Results

Phase One – Macroscopic Analysis of Diurnal Change

In order to gauge the changing trends in alertness and performance over the course of the afternoon, the average score was calculated for all the tests administered at each of the four testing intervals. These are tabulated below in table 3.9; and for each measure, the test result showing the greatest deterioration to alertness is highlighted in bold print.

Table 3.9 The mean scores (se) for all measures at each of the four test periods

TIME TEST	1 (1130h)	2 (1300h)	3 (1430h)	4 (1600h)
RT (secs)	.278 (.003)	.282 (.004)	.275 (.004)	.277 (.004)
RT Slowest 10% (secs)	.399 (.013)	.420 (.013)	.388 (.010)	.401 (.013)
TIME TEST	1 (1030h)	2 (1330h)	3 (1500h)	4 (1630h)
MSLT (mins)	16.740 (.683)	12.688 (.825)	15.188 (.693)	14.750 (.712)
SSS	2.875 (.135)	2.833 (.147)	2.583 (.133)	2.542 (.146)
VAS	40.125 (2.82)	37.667 (2.84)	31.021 (2.50)	28.958 (2.78)

It would appear from this broad overview of the data that the tests immediately following the afternoon lunch (at 1300–1330h) produced the worst scores in most of the measures — this was certainly true for the objective tests, and very nearly so for the subjective measures.

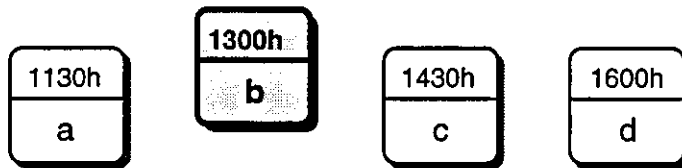
To throw light on this 1300-1330h deactivation, a further analysis was warranted — especially since it might provide additional support for the 1300h dip reported much earlier by Blake. The analytical procedures used to probe this post-lunch trough are explained in Box 2 overleaf.

BOX 2

A Strategy to Localise the Post-Lunch Trough in Alertness

The localisation of the afternoon trough to the first test following lunch (ie 1300–1330h) can be investigated further. If this immediate post-lunch test represents a time of greatest impairment, then the test scores produced during this 'dip' should differ significantly from adjacent tests. The test preceeding the dip and the final two tests of recovery following the transient dip can be viewed as 'normal' states, not characterised by major circadian fluctuations. Averaging these 3 test scores should provide a single test score that ably reflects the 'normal [baseline] state'. One could subtract this baseline score from the 1300-1330h dip in order to obtain a measure of the dip.

To exemplify; the RT test was administered at 4 time points:



(i) Normal [baseline] mean = $\frac{a + c + d}{3}$

(ii) The 1330h post-lunch change from baseline = $b - \left(\frac{a + c + d}{3} \right) =$ $\begin{matrix} \text{Difference} \\ \text{Scores} \\ x_1 \\ x_2 \\ \vdots \\ x_{48} \end{matrix}$

↑
one sample t-test

If there is no difference between the 1330h score and the 'baseline scores' then the mean of the 'difference scores' in (ii) should equal zero and the null hypothesis would prevail. A one-sample t-test on this difference score will establish whether or not the 1330h test is significantly different from the grouped average of the adjacent tests.

The procedures outlined in Box 2 to localise this afternoon trough were carried out for each of the tests. Reported below (table 3.10) are a summary of the results.

Table 3.10 . Comparisons made between the first post-lunch test and adjacent tests.

Test	Mean of First Post-Lunch Score c.1300h	Mean of the 3 Adjacent Baseline Scores	First Post-Lunch Test c.1300h Minus Mean of Baseline Scores 2-tail t-test on 'Difference Scores'
RT	.282	.277	t=2.76 [47]; p<0.01
RT (slowest)	.420	.396	t=2.43 [47]; p<0.05
MSLT	12.69	15.56	t=-4.81 [47]; p<0.001
SSS	2.83	2.67	t=1.22 [47]; NS
VAS	37.67	33.37	t=1.92 [47]; NS (p=0.06)

From the table above it can be seen that the tests that directly follows lunch (ie. tests conducted between 1300-1330h) produced levels of impairment/sleepiness that were significantly different from adjacent (baseline) scores. This was true in almost 4 of the 5 tests parameters. There are thus strong indications for a post-lunch trough localised around the 1300h mark.

The analysis carried out so far, in phase one of the results, focussed on the temporal characteristics of the afternoon dip, that is, an attempt was made to frame this window of vulnerability within a specified time-point. Beside this circadian factor, there were other independent factors also operating in this study. The size of lunch that was eaten was varied as were the levels of 'previous night sleep' and the subject demographics (gender). In order to examine the effects (and interactions) of these variables upon the numerous dependent measures, a more detailed analysis was required.

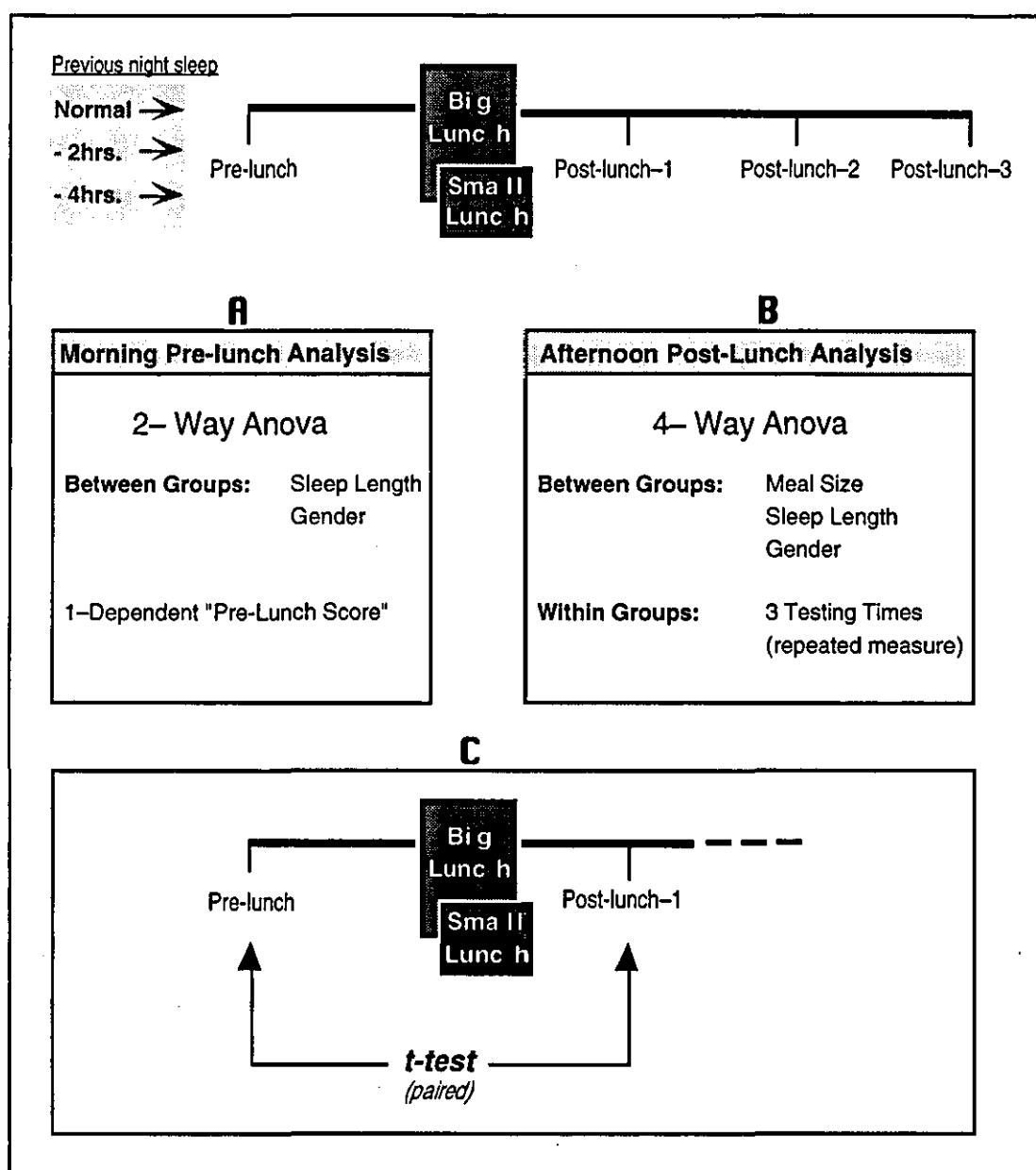
The second phase of the analysis which follows overleaf employs analysis of variance in association with post-hoc (Tukey's tests) follow-up procedures.

Phase Two – A Detailed Analysis of the Data Using ANOVA's.

Three separate analytical procedures were carried out on each test (see A, B, C below).

A. Morning Pre-lunch Analysis: Results of the morning tests (prior to lunch) were subjected to two-way anova's. At that point the only source of variance derived from differences in prior sleep length and gender. The factor for meals could not be included in the anova model as lunch was not consumed at that stage.

Figure 3.5. Model Depicting the Approach to Data Analysis: 3 Major Analytical Procedures



B. Afternoon Post-Lunch Analysis.

After lunch, tests were conducted at three time-points during the afternoon. An analysis of variance was carried out with:

- a). 3 Between-Group Factors – previous night sleep length, gender, meal size
- and b). 1 Repeated Measure – the three afternoon test points.

It was deemed appropriate to use ‘real scores’ and not to use ‘difference scores’ (ie. subtraction of afternoon scores from the pre-lunch baseline) as the subjects arriving at the morning test (baseline) were differentially fatigued due to variations in obtained sleep. For instance, subjects allowed just 4 hours sleep may be adversely affected in the morning, compared to normal sleepers who may be quite alert at the baseline test session. Should one contrast these morning scores with those during the expected post-lunch trough, then the sleep-deprived subjects may fail to show much change (having already exhibited a morning floor effect), whilst the normal sleepers may produce definite signs of post-lunch changes as their afternoon scores will be markedly different from their morning results.

C. Immediate Lunch Effects Via T-Tests.

The initial effects of lunch at test session, ‘post-lunch-1’, was directly contrasted with the pre-meal baseline state using paired t-tests. This simple comparison allows the effects of meals to be gauged.

Post-Hoc Analysis.

In cases where ANOVA’s produced significant results, post-hoc analysis were further employed where warranted. The post-hoc test chosen was the Tukey’s HSD Test which possessed the right degree of conservatism (falling between the Newman-Keuls and Scheffé procedures)

Reaction Time (RT)

The RT data was subjected to the 3-point analytical procedure outlined above:

- i). Morning Pre-Lunch Anova ii). Post-Lunch Anova and iii). Specific T-tests.

A. RT – Morning Pre-Lunch Analysis

The morning RT test was conducted at 11.30h. These data were analysed using a two-way anova incorporating the major factors of ‘previous night sleep’ and ‘gender’.

RT Main Effects (@ 11.30 h): No significant differences were found for the main effects of 'gender' and 'sleep length'. However, in the case of sleep length, there was a non-significant trend ($F=1.18 [2,42]; p>.3$) indicating that RT speed deteriorated with increasing sleep deprivation during the morning tests; see fig. 3.6 below.

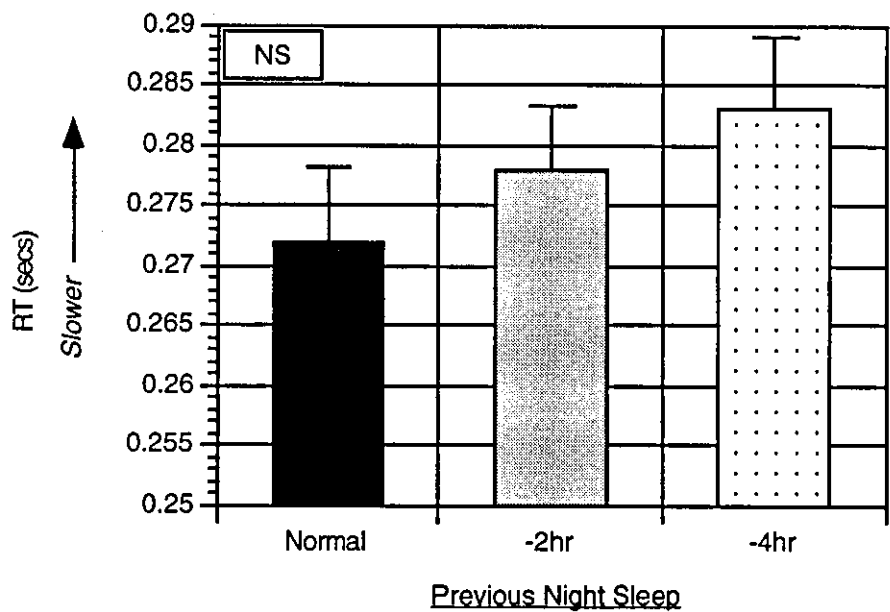


Fig 3.6 RT – Morning (Pre-Lunch) Comparisons Following 3 Sleep Regimens

RT – Interactions (@11.30 h): A significant interaction between 'sleep length' x 'gender' was found, $F=3.72 [2,42]; p<0.05$. Males and females obtained similar RT scores after a 'normal' night sleep and were similarly affected after a 4-hour sleep loss. However, in the case of a 2-hr sleep loss, females were greatly impaired, whilst male subjects showed a slight improvement; see Fig. 3.7 below.

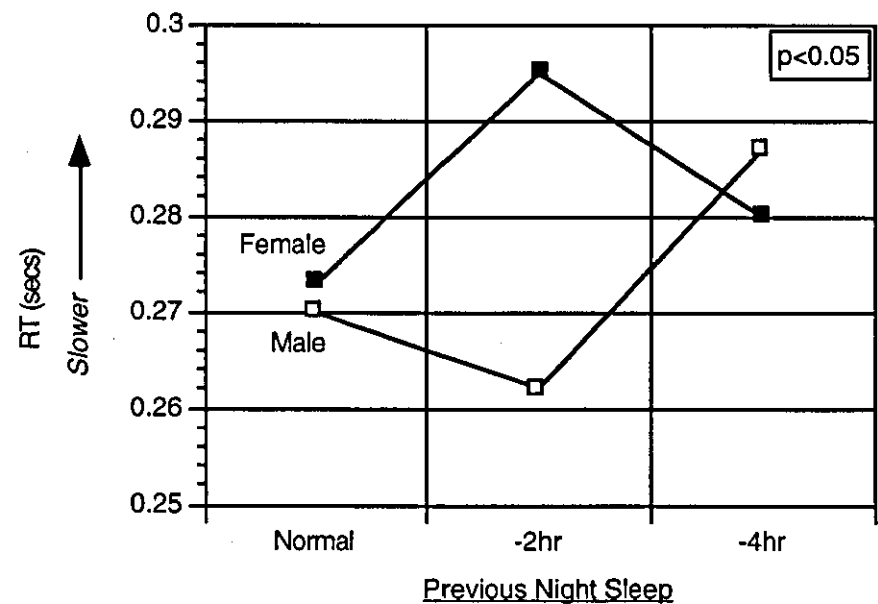


Fig. 3.7 RT – Morning (Pre-Lunch) Interactions: Sleep Length x Gender

B. RT – Afternoon Post-Lunch Analysis. This analysis (Anova) included the factors: Meals, Prior Sleep Length and Gender.

RT Main Effects: Significant main effects were found for gender in the post-meal analysis; $F=4.10 [1,36] p<0.05$. Female subjects had a significantly longer RT (note: in the pre-meal morning test, there were no such main effects for gender). For the main effects ‘sleep length’ and ‘meal size’ no significant differences were found.

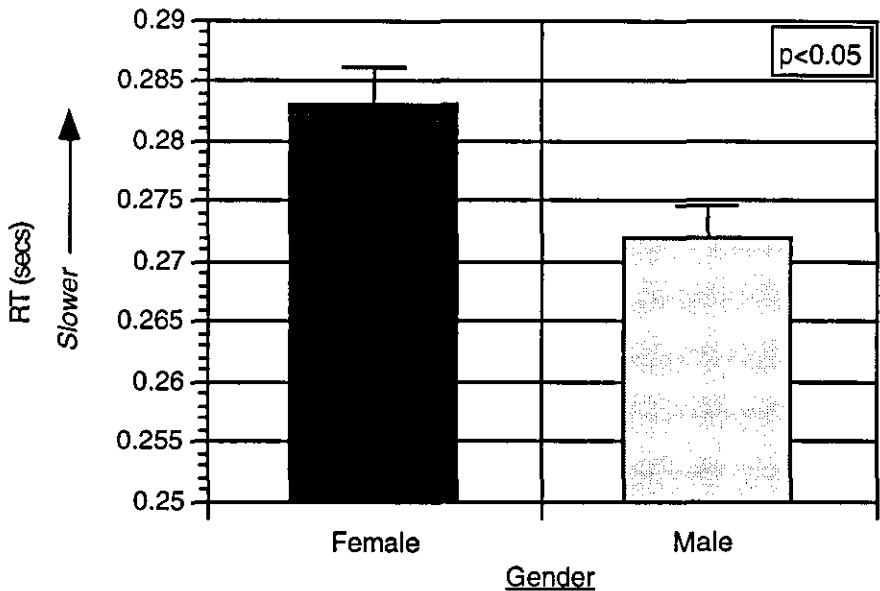


Fig. 3.8 RT: Post-Lunch Gender Effects (with se)

RT Within-Factor : Post-lunch Tests 1-3 Significant differences were found between the test sessions ($F=3.86 [2,72]; p<0.05$). The worst RT score occurred soon after lunch with an improvement in score by 14.30h. Tukeys post-hoc test revealed major differences between the 13.00h and 14.30h test.

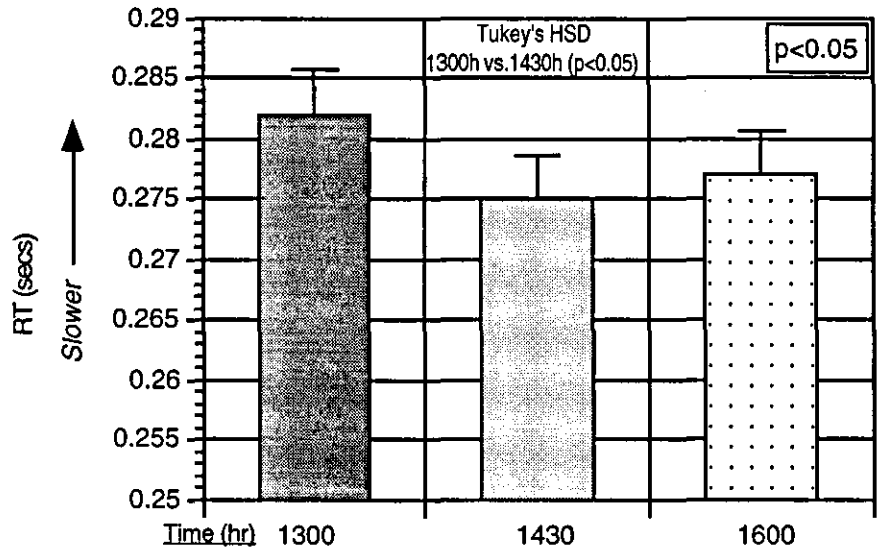


Fig. 3.9 RT: Mean Changes Between Post-Lunch Tests (with se)

RT : Interactions

The interaction between 'meal size x gender' was significant, $F=5.69 [1,36]$; $p<0.025$. After a small meal both sexes achieved near identical RT scores. Contrarily, following a large meal, performance between the sexes diverged markedly, with female subjects greatly impaired and male subjects showing distinct signs of improvement; see fig 3.10



Fig. 3.10 RT Interaction : Meal Size x Gender

Clearly, female subjects are most affected by the APS following a large meal: this result is also borne out by the results of other tests (see later). Furthermore, it is also clear from Fig. 3.9 (previous page) that the 1300h test produced the worst score of the afternoon; note too that phase one of the data analysis confirmed this point (pp.147-149). By marrying these facts, it is postulated that female subjects would be most vulnerable to any post-lunch effect, particularly at the 1300h test which follows lunch.

In a separate, but related post-hoc analysis, it was decided to test this assertion. The data pertaining only to female subjects was isolated and statistically examined to test the prediction that females consuming a large meal would show an elevated APS effect at 1300h (ie. the first test to follow lunch). The method used to carry out this analysis is explained in Box 3 overleaf.

The extent of the 1300h dip (relative to adjacent tests) was compared for subjects who had consumed a large meal against those consuming a small meal. As our prediction is a 'directed' one, based upon previous results a one-tailed t-test was employed. Results showed that the dip at 1300h was not statistically different between those subjects consuming a large meal versus a small meal ($t=1.06 (24)$; $p=.30$). The dip in RT performance would have occurred at 1300h irrespective of the size of meal eaten.

BOX 3

The Response of Female Subjects to the 2 Meal Types at the c. 1300h Test.

The effects of meal size on the APS at 1300-1330h was analysed females only. Given that the immediate post-lunch test captures the transient dip in alertness, the neighbouring tests preceeding and following the dip can be taken as indicators, representative of the normal [baseline] state. Averaging the two adjacent tests would provide a measure of this baseline score.

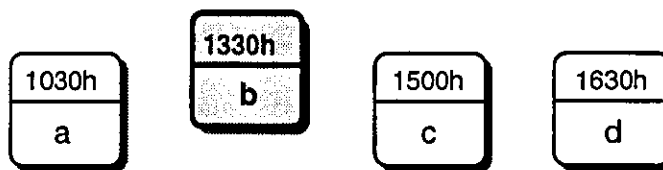
By calculating the difference between these baseline scores and the score for the 1300-1330h dip, a measure of the post-lunch trough can be ascertained.

This procedure was carried out for:

1. Female subjects consuming a large meal.
2. Female subjects consuming a small meal.

T-tests were then conducted to test the differences between the two meal sizes at 1300-1330h.

To exemplify; the MSLT was administered at 4 time points:



(i) Normal [baseline] mean = $\frac{a+c}{2}$

(ii) Change from baseline :

Large Meal

$$b - \left(\frac{a+c}{2} \right) = x_1$$

$$\vdots$$

$$x_{24}$$

Small Meal

$$b - \left(\frac{a+c}{2} \right) = y_1$$

$$\vdots$$

$$y_{24}$$

Two-Column T-test

The t-test will allow us to say whether the 1300h dip is exacerbated by the large meal and whether the difference between the meal types are significant.

There was also a significant interaction between sleep length and gender, $F=3.68$ [2,36]; $p<0.05$. The level of performance between the sexes remained essentially identical under conditions of normal sleep and 4-hr. sleep deprivation. However, under

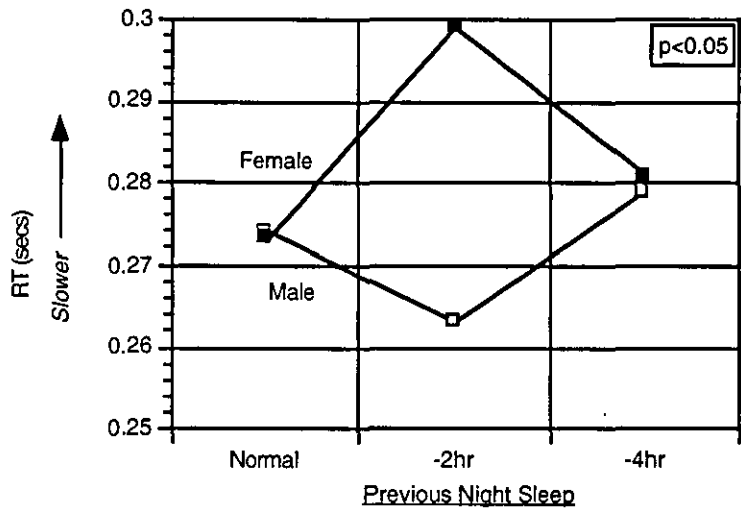


Fig. 3.11 RT Interaction : Sleep Length x Gender

the milder 2-hr sleep deprivation, female subjects were maximally impaired, whilst, by contrast, male subjects improved under this regimen. This interaction with its distinct pattern was also evident in the pre-lunch (11.30 h) test; see figure 3.7.

The 2-way interaction between ‘sleep length x time’ was not significant, though the trends produced were interesting (see below). Essentially, a normal nights sleep is very similar – in terms of variation and patterning – to the 2-hr. sleep deprived group, though the performance of the latter group is dampened throughout. Compared to the rhythmic variations seen in these two groups, the 4-hr. sleep deprived group showed no fluctuation, with an almost linear trend reflecting the marginal improvement at each test. Any notional rhythmicity is lost in this acutely sleep deprived group.

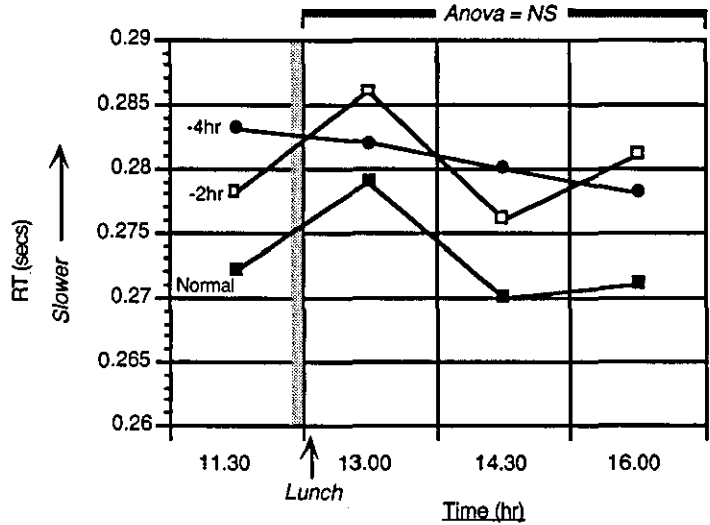


Fig. 3.12 RT Interaction : Sleep Length x Time

C. RT-Specific T-test Comparisons: *Pre-lunch (1130h) vs Post-lunch (1300h)*

The results presented above indicate that the immediate post-lunch period (ie. 13.00h) is the time of greatest performance deterioration. To probe this further, t-tests were conducted to compare the morning (no-lunch) state with the 13.00 h post-lunch state. Separate tests were performed for subjects receiving a big lunch and for subjects receiving a small lunch.

Lunch Size		Means & T - tests
Big Lunchers	[n=24]	11.30 vs 13.00 h
Small Lunchers	[n=24]	11.30 vs 13.00 h

Table 3.11 RT : Means and T-tests To Compare Morning and Post-lunch (1300h) Scores

Sleep	Big Lunch [n=24]			Sleep	Small Lunch [n=24]		
	11.30h	13.00h	Change		11.30h	13.00h	Change
Normal	.270 (.012)	.279 (.012)	+.009	Normal	.273 (.006)	.278 (.008)	+.005
-2 hr	.278 (.009)	.286 (.001)	+.008	-2 hr	.278 (.006)	.280 (.007)	+.002
-4 hr	.288 (.011)	.288 (.008)	0.00	-4 hr	.278 (.005)	.277 (.005)	-.001
Overall	.279 (.006)	.284 (.006)	+.005	Overall	.277 (.003)	.280 (.004)	+.003
t=16.77 [47] ; p<.0001				t=16.81 [47] ; p<.0001			

Overall t-tests between the 11.30 h and 13.00 h scores indicated that significant changes occurred during this time period whether subjects ate a big lunch or a small lunch. So, lunch size per se, did not appear to moderate the immediate post-lunch condition. However, when the mean score of the two lunch groups (big vs. small) was examined in the light of 'previous night sleep', interesting trends emerged. A greater change in score, towards impairment, was evident in the normal sleep group with virtually no change in the 4-hr. sleep deprived group. This clear difference can be

explained by the fact that the normal sleepers initially attained quite fast RT scores in the morning, whilst the low morning scores of the 4-hr. deprived group was maintained even after lunch, indicating that a possible ‘floor effect’ was reached in this ‘sleepy’ group. Overall, changes of this nature, were more noticeable after a big lunch was consumed.

The RT Lapse Domain: Analysis of The Slowest 10% of Scores

Sleepy subjects are particularly prone to ‘lapsing’. To evince instances of lapses in attention, the RT results were filtered, and from each test, the slowest 10% of response times were isolated, and statistically analysed. This analysis is an important addendum to the main RT results presented above.

A. Slowest RT : Morning Pre-Lunch Analysis

Slowest RT : Main Effects (@ 11.30 h)

A pre-lunch anova of the morning test yielded a significant effect for ‘previous night sleep length’, $F=3.11$ [2,42]; $p<0.05$. The deterioration in response times was positively correlated with hours of sleep loss.

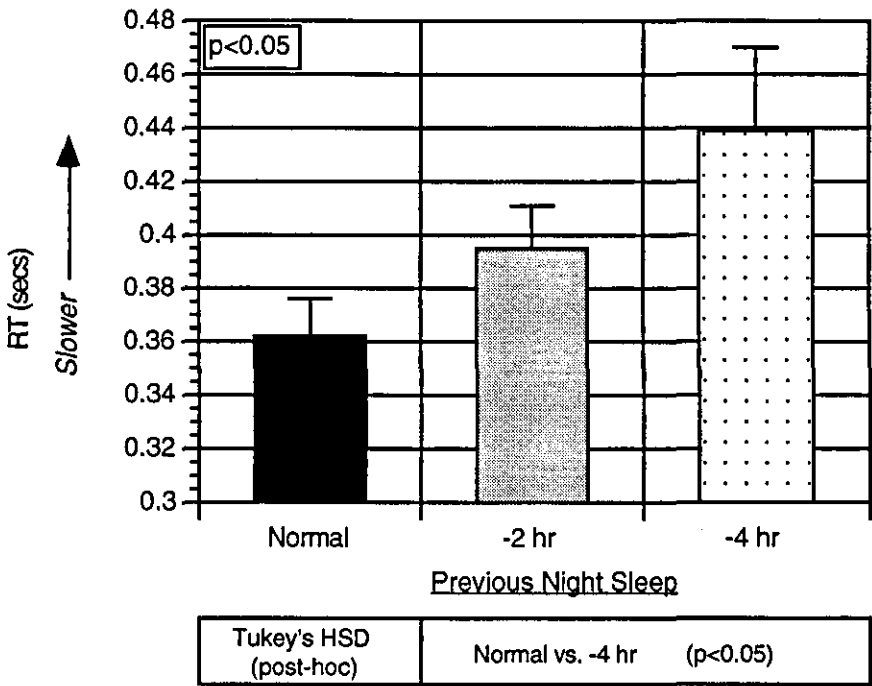


Fig. 3.13 Slowest RT – Morning Comparisons Following 3 Sleep Regimens

B. Slowest RT : Afternoon Post-Lunch Analysis

RT Main Effects: There was a significant effect for meal size, $F=4.20$ [1,36]; $p<0.05$.

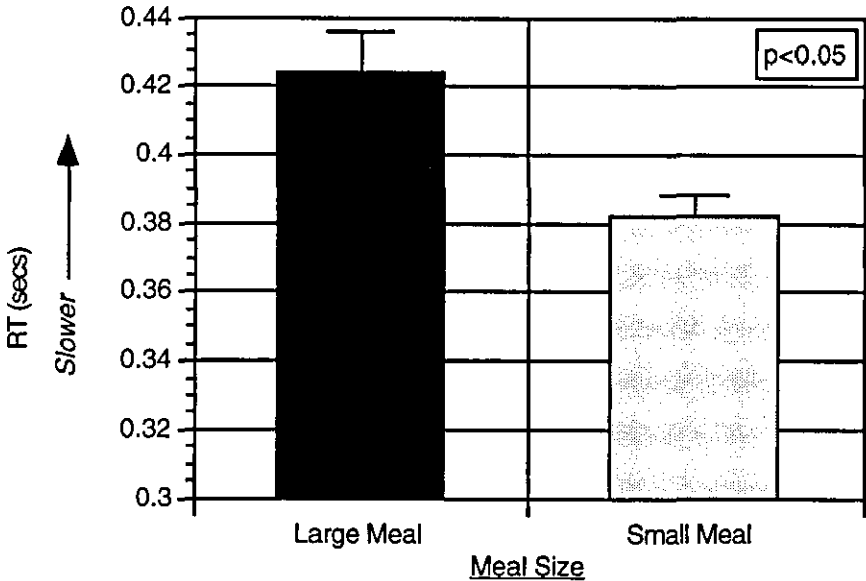


Fig. 3.14 Slowest RT: Post-Lunch Meal Effects

Slowest RT : Within-Factor

There were significant changes between successive afternoon testings, $F=3.73$ [2,72]; $p<0.05$. Post-hoc analysis revealed significant differences between the 13.00h test and the 14.30h test. This is graphically represented below; also included for illustrative purposes, is the morning (11.30 h) test result.

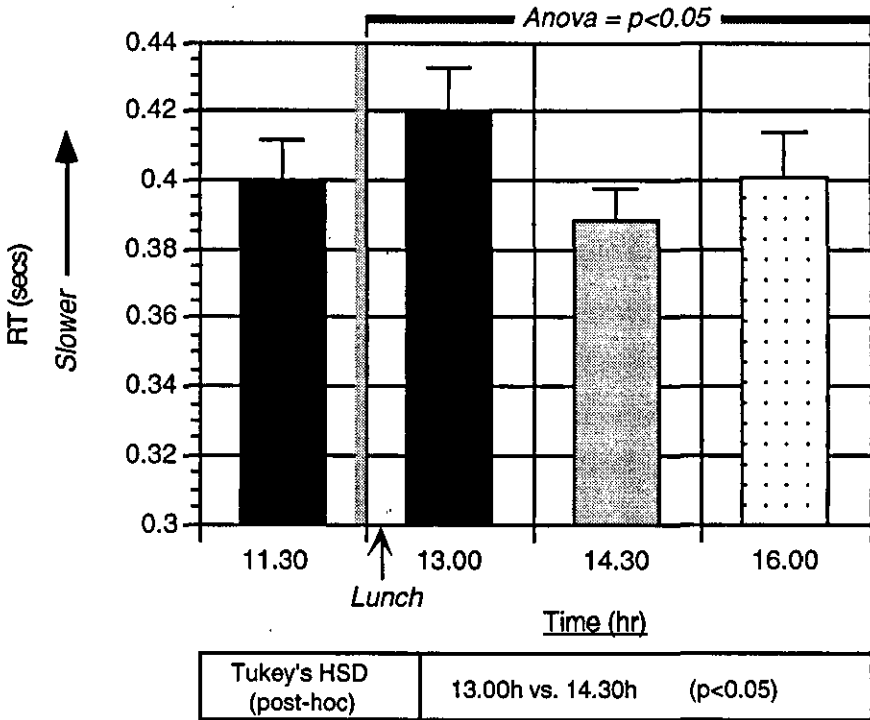


Fig. 3.15 RT: Mean Change Between Test Sessions (with se)

Slowest RT : Interactions

The interaction, ‘meal x gender x time’ ($F=3.25$ [2,72]; $p<0.05$) clearly showed female subjects to be most affected after consuming a large meal – especially after the first two tests – whilst males were less affected by meal size.

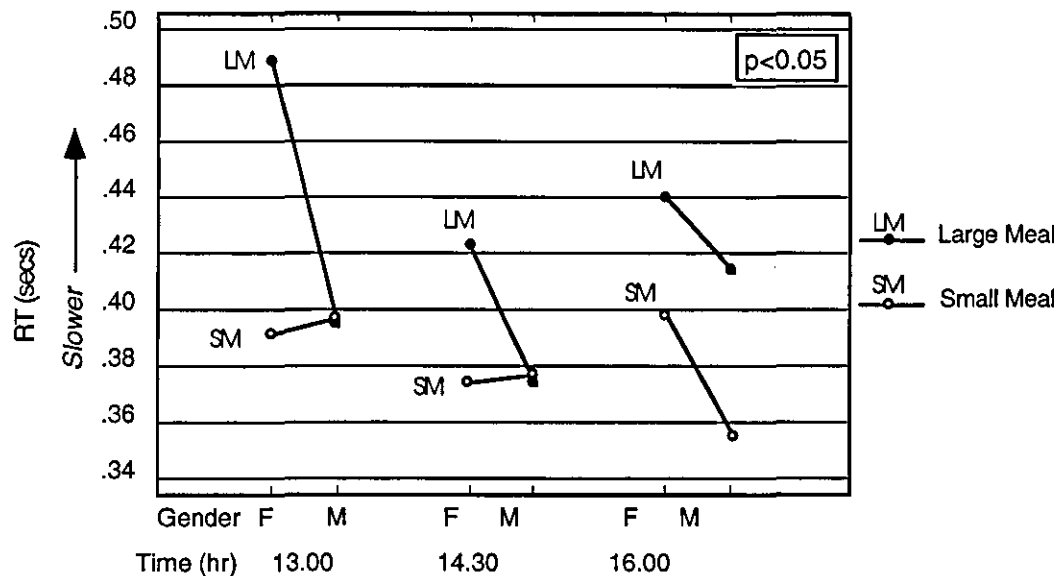


Fig. 3.16 Slowest RT Interaction: Meal Size x Gender x Time

This interaction was subjected to further analysis: specifically, the data of just the female subjects was scrutinised to see whether the 1300h dip (as shown in figs. 3.15 and 3.16) was significantly exacerbated for subjects consuming a large meal. The method for this post-hoc analysis is explained in Box 3 (see page 155).

A one-tailed t-test comparison suggest that the difference between the size of a 1300h dip after a large meal versus a small meal approaches significance ($t=1.51$ (24) $p=0.07$) for female subjects.

Multiple Sleep Latency Test (MSLT)

A. MSLT : Morning Pre-Lunch Analysis

MSLT: Main Effects (@ 10.30 h)

There were no significant main effects for either 'previous night sleep' or 'gender'.

MSLT: Interactions

There was a significant interaction between 'sleep length' x 'gender', $F=5.05 [2,42]$; $p<0.05$. Male subjects were more sleepy than female subjects under both normal sleep regimens and 2-hr sleep restrictions. However, male subjects exhibited a marked rise in alertness under the more severe sleep reduction of 4 hours. Female subjects presented the classic trend – a declining sleep latency in response to sleep deprivation.

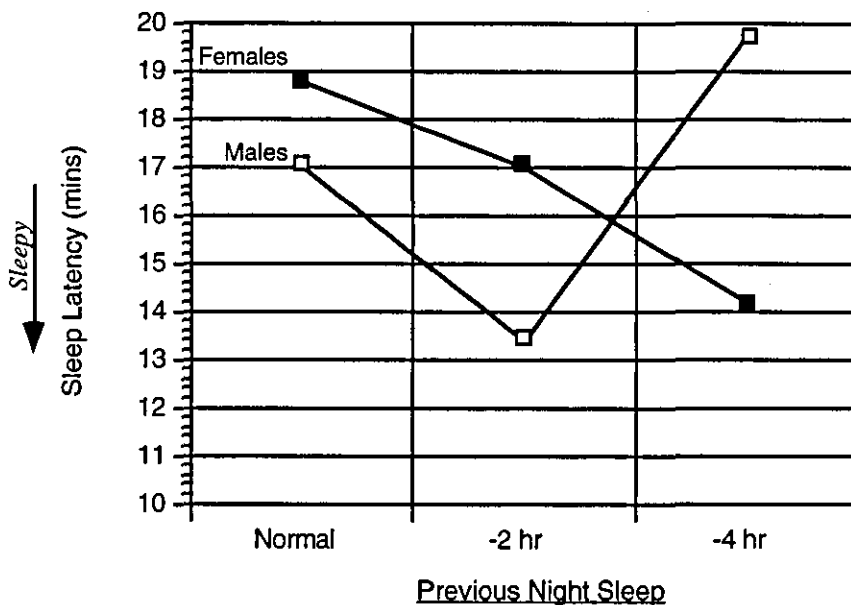


Fig. 3.17 MSLT Interaction: Sleep Length x Gender

B. MSLT : Afternoon Post-Lunch Analysis

MSLT Main Effects:

There were no significant differences for the 3 main effects; meal size, gender or prior sleep length.

MSLT Within-Factor : Post-lunch Tests 1-3

A significant main effect was found for the repeated measure – the three post-lunch testing times, $F=7.44 [2,72]$; $p<0.001$. The fastest sleep latency occurred in the first test to follow lunch (13.30h). At subsequent tests (15.00h and 16.30h) sleepiness declined.

These final two tests, suggesting a recovery toward alertness, were significantly different from the 13.30h post-lunch latency test, according to post-hoc analysis. This result is plotted below (with the morning prelunch score for comparative interest).

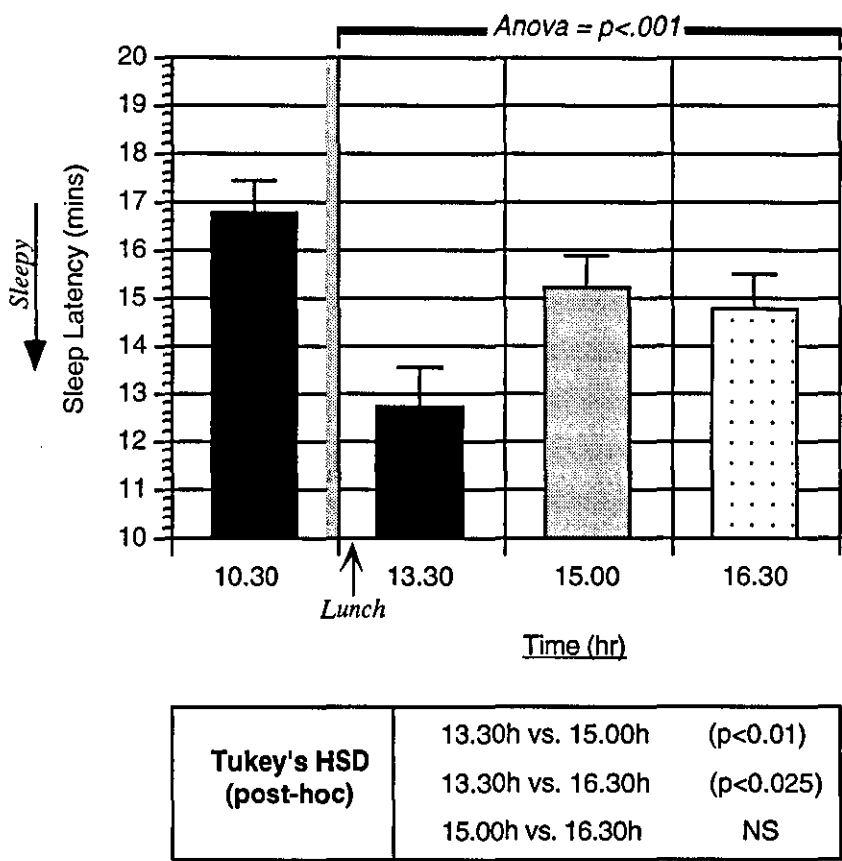


Fig. 3.18 MSLT: Mean Changes Between Post-lunch Tests (with se)

MSLT Interactions:

The interaction between ‘gender x time’ was significant, $F=3.23 [2,72]; p<0.05$; see graph overleaf. Both male and female subjects had the shortest sleep latency at 13.30h. There was an improvement in alertness in both groups at the next 15.00h test – in the case of females this change was significant (post-hoc comparisons: 15.00h vs. 13.30h; $p<0.05$). Male subjects continued to improve in a linear fashion, and by the final 16.30h test, the level of alertness was significantly different from the sleepy disposition following lunch (post-hoc comparisons: 16.30h vs. 13.30h; $p<0.025$).

Also shown in the plot (fig. 3.19) is the morning pre-lunch test result – though it did not contribute to the anova, its inclusion in the graph does highlight the fact that both sexes produced near identical sleep latencies in the morning, before food consumption.

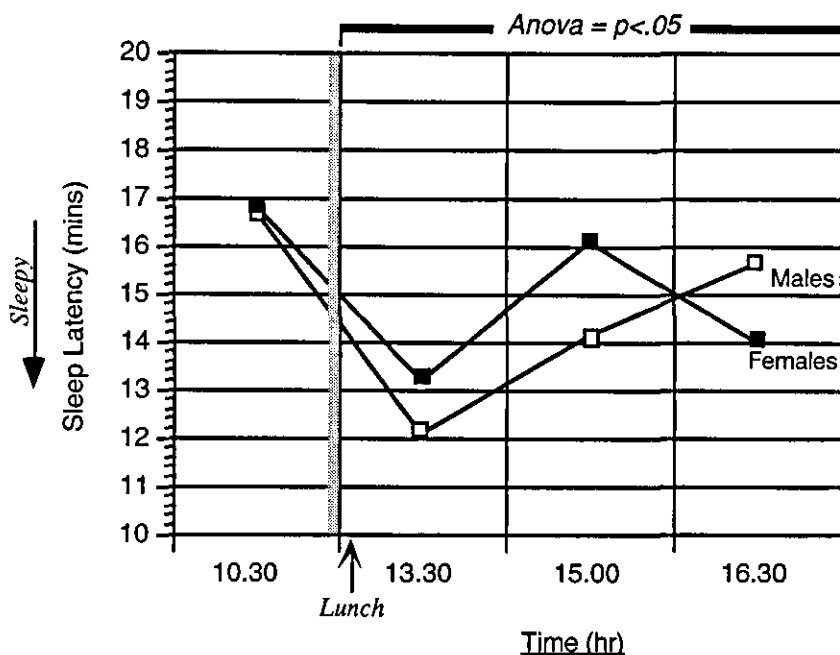


Fig. 3.19 MSLT Interaction: Gender x Time

Although the interaction ‘previous night sleep x time’ did not achieve significance, it produced trends which were interesting; see fig. 3.20 below. Irrespective of their primary sleep budget, all groups were maximally sleepy soon after lunch, at 13.30h, though the dip was emphasised in the sleep deprived group. A recovery by 15.00h was evident, but further signs of improving alertness was moderated or reversed at 16.30h.

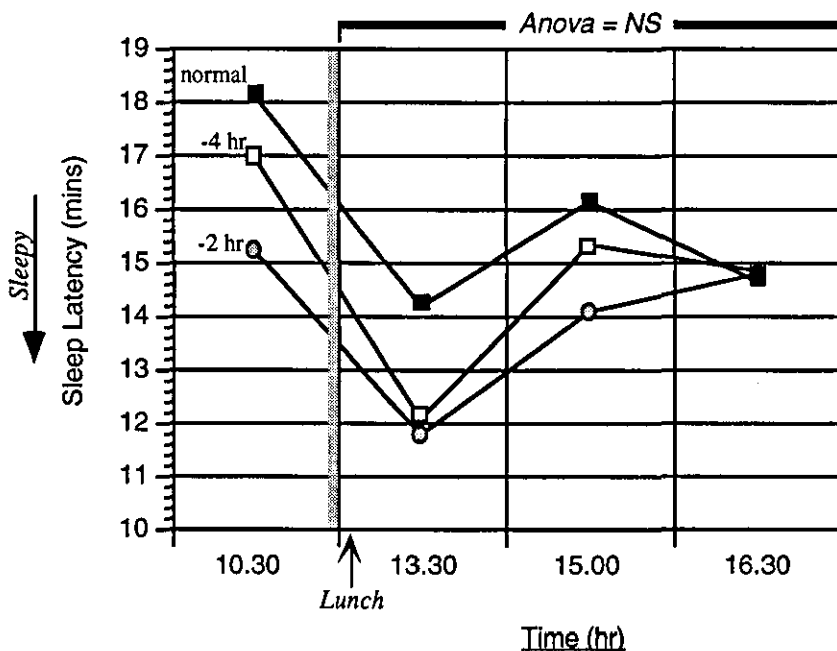


Fig. 3.20 MSLT Interaction: Sleep Length x Time

In view of the findings from other tests, a separate analysis was conducted to see whether the 1330h dip was exacerbated for female subjects consuming a large meal as against a small meal. The rationale and procedure is explained in Box 3 (see page 155). Results from t-tests indicated that the extent of the 1330h dip was not significantly affected by the size of the meal eaten ($t=1.31$ [24] $p=.10$).

C. MSLT T-test Comparisons: Pre-lunch (1030h) vs Post-lunch (1330h)

The immediate effects of the intervening lunch was examined by comparing the mean scores of the pre-lunch (10.30h) test with the post-lunch (13.30h) test. This was carried out on the 'big lunch' group and the 'small lunch' group.

Table 3.12 MSLT : Means and T-tests To Compare Morning and Post-lunch (1300h) Scores

Sleep	Big Lunch [n = 24]			Sleep	Small Lunch [n=24]		
	10.30h	13.30h	Change		10.30h	13.30h	Change
Normal	17.38 (1.8)	13.38 (2.6)	- 4.0	Normal	18.81 (1.2)	15.00 (2.1)	- 3.81
-2 hr	13.57 (2.0)	13.07 (1.9)	- 0.5	-2 hr	18.00 (1.2)	11.69 (1.2)	- 6.31
-4 hr	16.69 (1.5)	9.88 (1.8)	- 6.81	-4 hr	17.19 (1.6)	14.31 (1.6)	- 2.88
Overall	15.89 (1.0)	12.07 (1.2)	- 3.77	Overall	18.00 (0.8)	13.67 (1.1)	- 4.33
$t = -13.90$ [46] ; $p < .0001$				$t = -18.86$ [47] ; $p < .0001$			

The overall sleep latency was significantly reduced (by 3.8 mins.) in the case of the big lunch group. A similar, significant, reduction in sleep latency of 4.3 minutes occurred in the small lunch group. Because both lunch conditions produced similar falls in sleep latency, we cannot associate the magnitude of post-prandial sleepiness with meal size. In the 2hr. sleep reduction group, one 'outlier' score was removed from analysis.

Examining the change in sleep latency for each of the three 'sleep groups', it appears that under normal sleep regimens, the extent of post-lunch sleepiness is similar after both big or small lunch conditions. If sleep is severely curtailed (-4hr), a large lunch is more soporific than a small lunch (the converse is true when sleep is reduced by 2hrs.).

Stanford Sleepiness Scale (SSS)

A. SSS : Morning Pre-Lunch Analysis

SSS: Main Effects (@ 10.30 h)

For the main effect 'previous night' sleep length, there were significant changes to subjective sleepiness, $F=4.88$ [2,42]; $p<0.01$. Subjects who obtained their normal sleep quota were significantly more alert than subjects who had experienced sleep reductions — the difference was significant when compared to the 2-hr sleep reduction group, and almost significant in comparison to the 4-hr sleep restricted group. Subjects who reduced their nocturnal sleep by 4-hrs. were marginally more alert than the 2-hr. sleep deprived group; though the differences between these two groups were not significant. There were no further significant differences for the other main effects, 'gender', or the interaction 'gender x sleep length'.

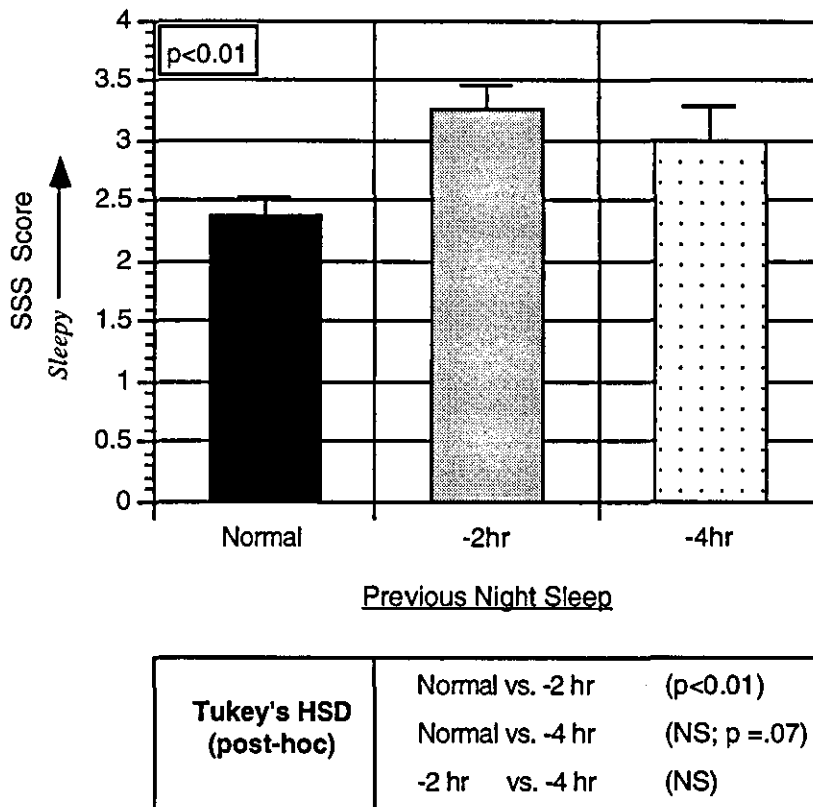


Fig 3.21 SSS-Morning (Pre-Lunch) Comparisons Following 3 Sleep Regimes

B. SSS : Afternoon Post-Lunch Analysis

SSS: Main Effects

According to anova's for post-lunch tests, no significant differences were indicated for any of the three main effects; meal size, sleep length or gender.

SSS Within-Factor : Post-lunch Tests 1-3

The repeated measure, ‘post-lunch testing times’ produced no significant changes.

SSS: Interactions

No significant interactions were found. Nevertheless, it is still instructive to view the trends associated with these (non-significant) interactions. These trends reflect those seen in the RT results, though the lack of statistical significance in the case of the SSS data, requires cautious interpretation.

The interaction ‘gender x sleep’ maps the daytime sleepiness ratings of the two sexes under contrasting sleep regimens. Both groups were similarly affected after a normal nights sleep or a sleep reduction of 4 hours. However, when female subjects curtail their sleep by 2 hours, they are reportedly more sleepy than under the severe sleep curtailment of 4 hours. Put another way, their level of alertness is remarkably high under the more harsher sleep restriction. This anomalous trend was also seen in the RT data; see fig. 3.7 and fig. 3.11.

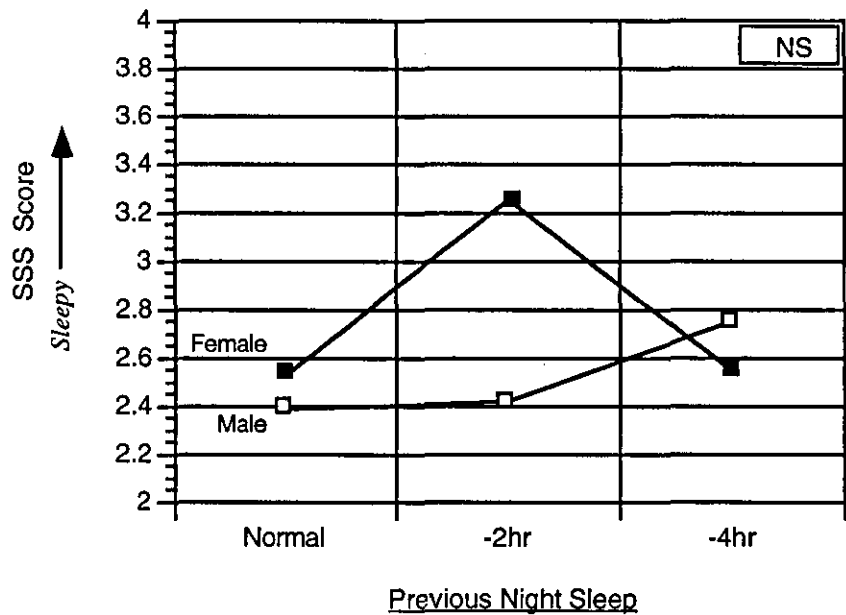


Fig. 3.22 SSS Interaction: Gender x Sleep

This greater level of sleepiness in the 2 hour sleep reduction group – when compared to the 4 hour reduction group – is further illustrated in the graph overleaf; fig 3.23. The post-lunch behaviour of the 4-hour sleep deprived group was closely allied to that of the normal sleep group.

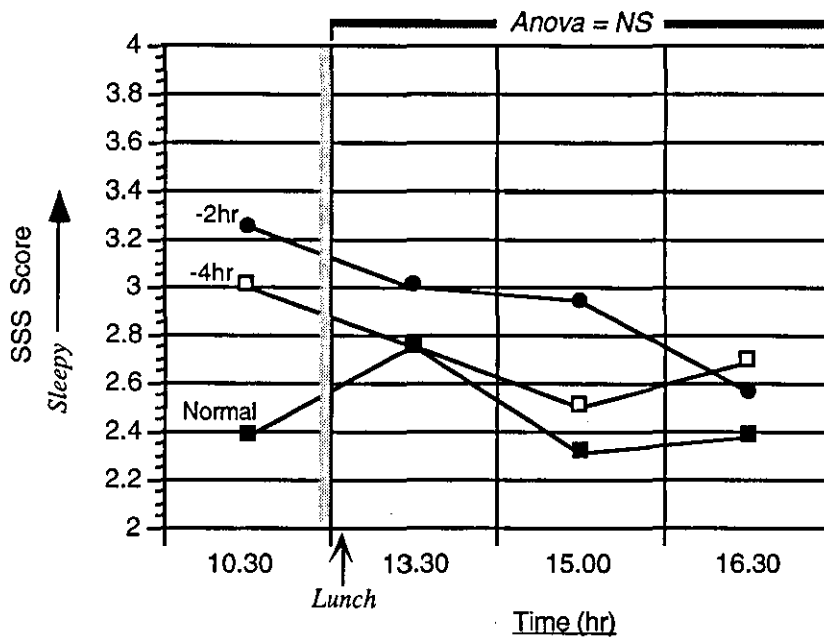


Fig. 3.23 SSS Interaction: Sleep x Time

Results from the SSS interaction “meal x gender x time” closely resembles a similar, though significant, RT interaction (cf. fig. 3.16). Female subjects were more likely to experience subjective sleepiness immediately after a large lunch, according to the trends produced by this interaction; lending further support to earlier RT findings.

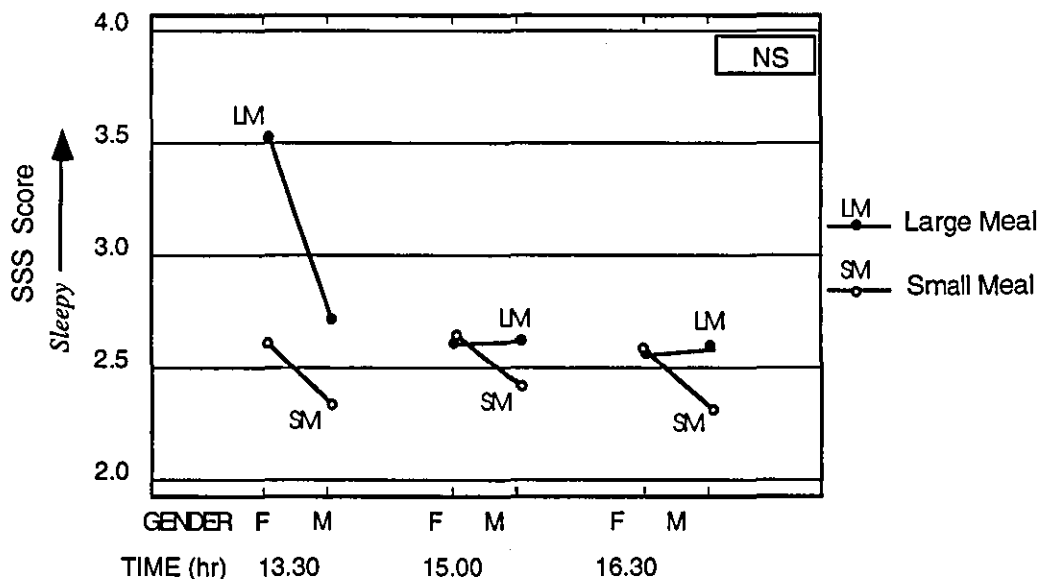


Fig. 3.24 SSS Interaction: Meal Size x Gender x Time

A separate analysis which concentrated on the female subjects, tested the prediction that the dip would be most marked at 1330h and exacerbated by a large meal (see Box 3, pp. 154–155). According to a one-tailed t-test, female subjects consuming a large meal [cf. a small meal] were significantly more sleepy at 1330h ($t=2.83$ [24]; $p<0.01$).

C. SSS T-test Comparisons: Pre-lunch (1030h) vs Post-lunch (1330h)

The 24 subjects receiving a large lunch rated themselves marginally (though significantly) more sleepy at 13.30h when compared to the morning period (10.30h). Those subjects presented with a small midday lunch became fractionally more alert.

Table 3.13 SSS : Means and T-tests To Compare Morning and Post-lunch (1300h) Scores

Sleep	Big Lunch [n=24]			Sleep	Small Lunch [n=24]		
	10.30h	13.30h	Change		10.30h	13.30h	Change
Normal	2.38 (.26)	3.13 (.30)	+0.75	Normal	2.38 (.18)	2.38 (.32)	0.00
-2 hr	3.13 (.13)	3.13 (.44)	0.00	-2 hr	3.38 (.42)	2.88 (.44)	- 0.5
-4 hr	3.25 (.31)	3.25 (.31)	0.00	-4 hr	2.75 (.45)	2.25 (.25)	- 0.5
Overall	2.92 (.16)	3.17 (.20)	+0.25	Overall	2.83 (.22)	2.50 (.20)	- 0.33
t= -11.31 [47] ; p<.001				t= -6.59 [47] ; p<.001			

The ‘overall’ changes between the two ‘lunch groups’ is also graphed below.

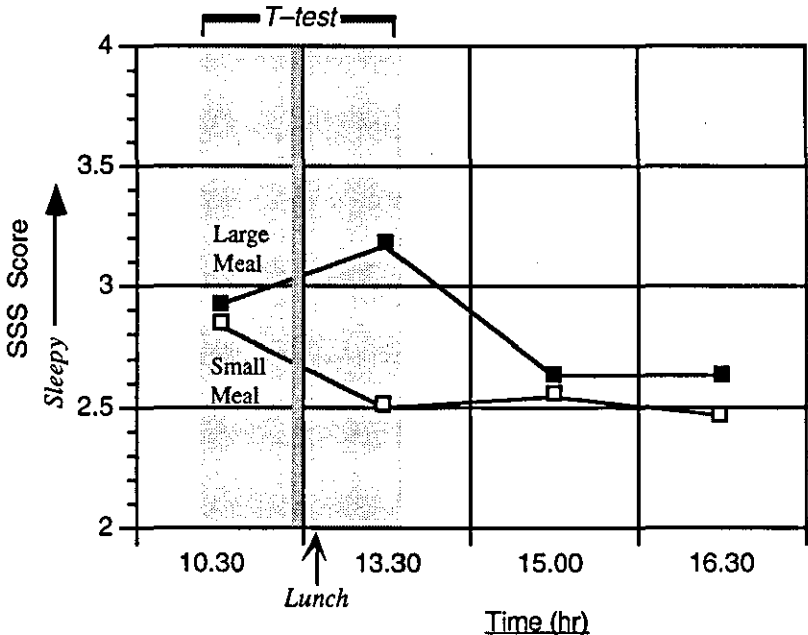


Fig. 3.25 SSS: Pre/Post-Lunch Changes and Meal Size

Visual Analogue Scale (VAS)

A. VAS : Morning Pre-Lunch Analysis

There were no significant differences in sleepiness during the morning ratings.

B. VAS : Afternoon Post-Lunch Analysis

VAS: Main Effects

Subjects consuming a large lunch reported greater post-prandial sleepiness than those receiving a small lunch, $F=4.05$ [1,36]; $p<0.05$.

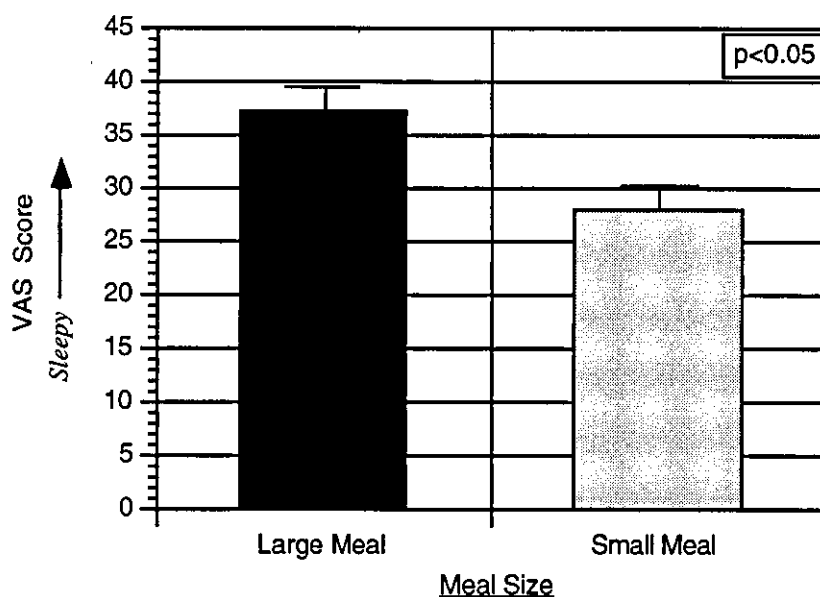


Fig. 3.26 VAS: Post-Lunch Meal Effects

VAS Within Factor: Post-lunch Tests 1-3

Significant differences were found between the afternoon tests, $F=5.28$ [2,72], $p<0.01$ (see fig. 3.27 overleaf). Essentially, at the time of the first post-lunch test (13.30h), subjects were relatively sleepy, but improved in alertness at successive afternoon tests: the improvement approached significance at 15.00h and achieved clear significance at 16.30h (relative to the 13.00h low-point).

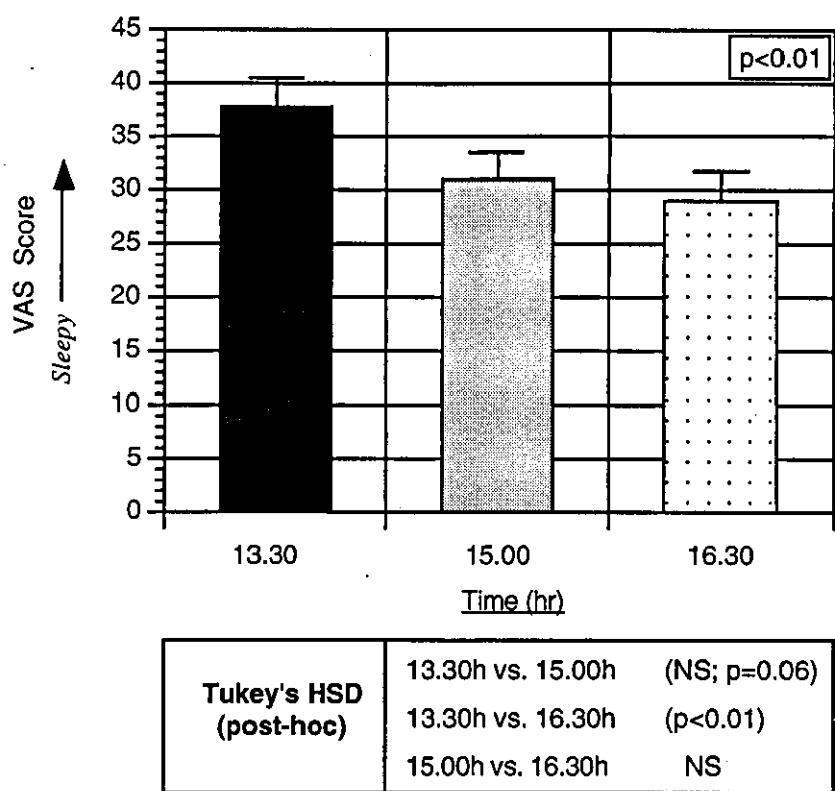


Fig. 3.27 VAS: Mean Changes Between Post-lunch Tests (with se)

VAS : Interactions

There were no significant interactions. Nevertheless, it is instructive to note from a plot of 'sleep length x time' that the 2-hr sleep reduction elicited greater reports of subjective sleepiness than the more severe 4-hr sleep loss. This is in accord with other data.

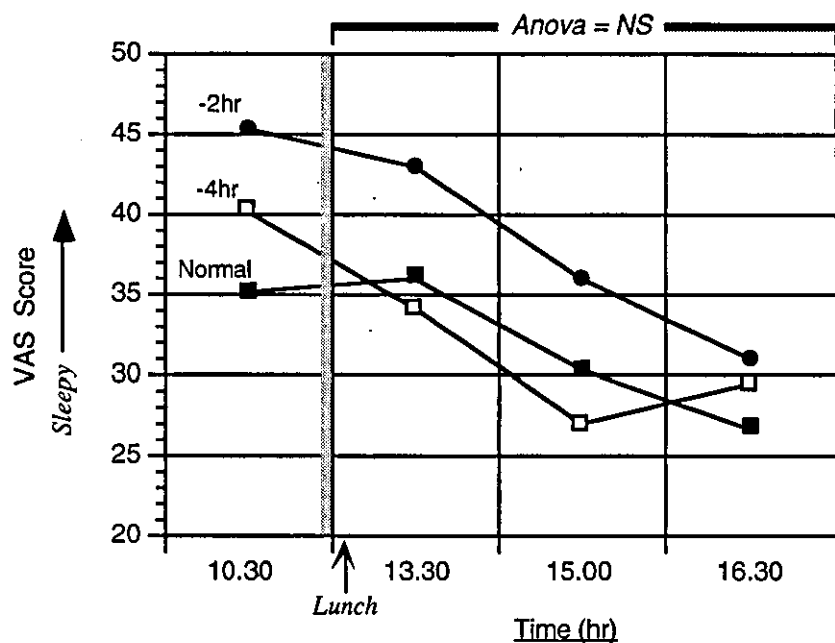


Fig. 3.28 VAS Interaction : Sleep Length x Time

Analysis of the main effects indicated that a large lunch was more likely to dampen alertness (cf. fig 3.26). Seen in the light of other factors, ie. 'meal x gender x time', it was clear that this post-lunch effect was more impressive in female subjects consuming a large lunch, and was particularly pronounced at 13.30h (cf. figs. 3.16, 3.24).

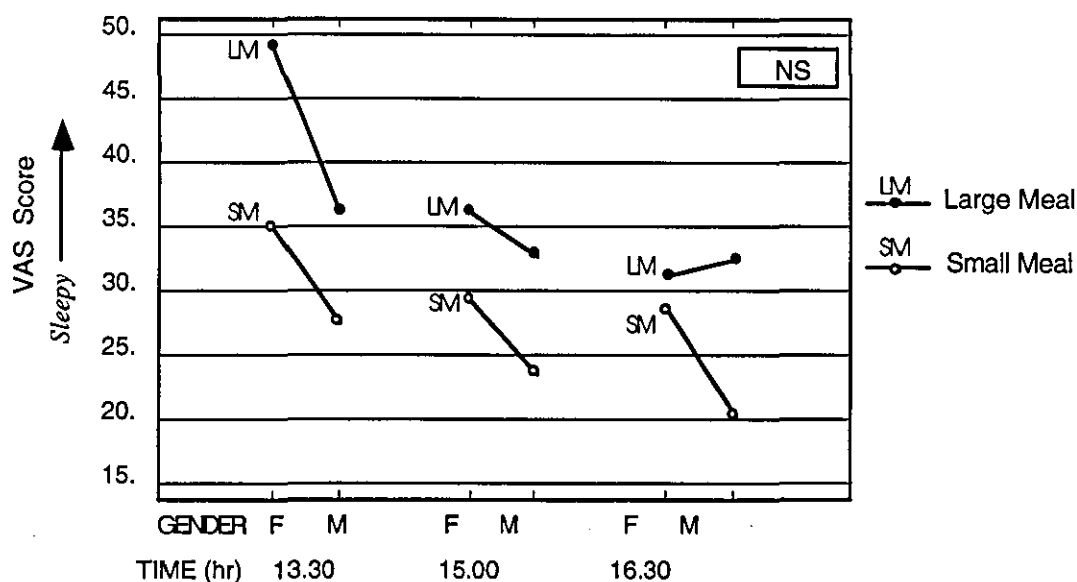


Fig. 3.29 VAS Interaction: Meal Size x Gender x Time

In a follow-up analysis to the above interaction, the extent of the dip at 1330h was examined solely for female subjects. Based on apriori knowledge and the results of other tests, it was predicted that female subjects consuming a large meal would be more severely affected (at 1330h) compared to a similar cohort consuming a small meal. The analytical procedure is explained in Box 3 (see pages 154–155).

Results from such a direct comparison, show that for *female subjects only*, the dip at 1330h is significantly greater after consuming a large meal as compared to a small meal (one tail t-test; $t=1.78$ [24]; $p<0.05$).

C. VAS T-test Comparisons: Pre-lunch (1030h) vs Post-lunch (1330h)

Table 3.14 VAS : Means and T-tests To Compare Morning and Post-lunch (1300 h) Scores

Sleep	Big Lunch [n = 24]			Sleep	Small Lunch [n=24]		
	10.30h	13.30h	Change		10.30h	13.30h	Change
Normal	36.75 (7.4)	42.13 (6.3)	+5.38	Normal	33.38 (6.8)	29.75 (7.3)	-3.63
-2 hr	45.25 (3.5)	45.75 (7.2)	+0.5	-2 hr	45.13 (7.8)	40.25 (7.6)	-4.88
-4 hr	46.75 (4.9)	42.50 (5.9)	-4.25	-4 hr	33.50 (9.8)	25.63 (6.4)	-7.87
Overall	42.92 (3.2)	43.46 (3.6)	+0.54	Overall	37.33 (4.7)	31.88 (4.1)	-5.45
$t = -17.58$ [47]; $p < .0001$				$t = -10.60$ [47]; $p < .0001$			

The data tabulated above contrasts the pre-lunch/post-lunch changes. The 'overall' data indicate that a large lunch mutes the improvement in alertness, when compared to the changes that follows a small lunch. This trend is also plotted below.

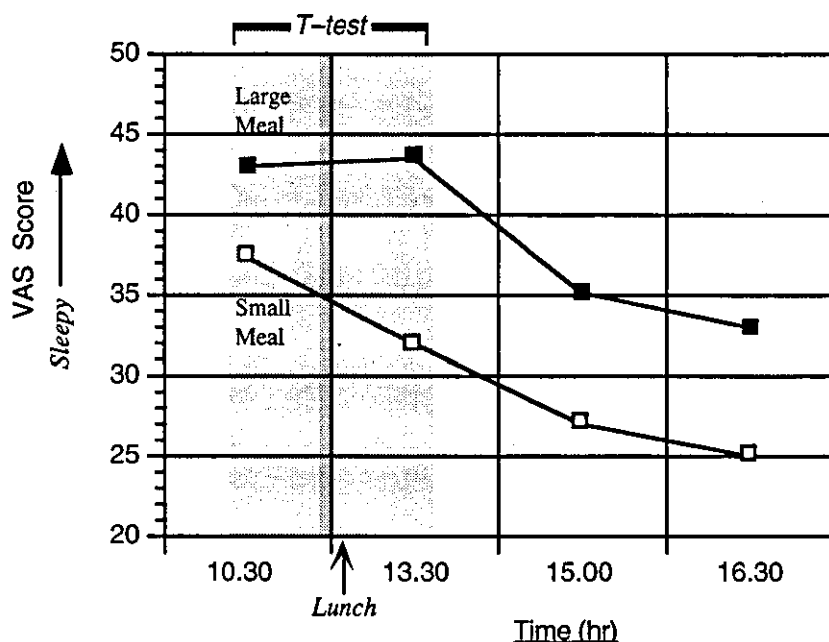


Fig. 3.30 VAS: Pre/Post-Lunch Changes and Meal Size

Post-Sleep Questionnaire

Thirty minutes after waking, subjects completed a Post-Sleep Questionnaire, probing residual sleepiness, waking difficulty and sleep quality (see table 3.15 below). The scores for the three sleep regimens were subjected to a 1-Way ANOVA. Significant differences were obtained for previous night 'sleep quality', $p < 0.05$. Here, post-hoc tests revealed that the principal differences were between those subjects receiving a 'normal' night sleep, and those subjects on 4-hrs. sleep restriction. Subjects experiencing the severe sleep restriction reported an *improved* 'sleep quality' as compared to controls.

Table 3.15 Post-Sleep Reports: completed soon after arising on the experimental day.

Previous night sleep	Sleepiness after 30 min. awake (SSS)	Difficulty in getting up	Quality of sleep obtained
Normal	2.6 (.27)	3.2 (.25)	3.0 (0)
-2 hrs	3.5 (.22)	3.7 (.21)	2.7 (.33)
-4 hrs	3.3 (.39)	3.7 (.24)	2.4 (.16)
1-Way Anova	NS	NS	<p>$p < 0.05$</p> <p>Post-hoc</p> <p>Normal vs -2hr = NS</p> <p>* Normal vs -4hr = $p < 0.05$</p> <p>-2hr vs -4hr = NS</p>
QUESTIONS & SCALES	<p>SSS</p> <p><u>7 Point Scale</u></p> <p>2 = Functioning at high level.....</p> <p>3 = Relaxed, awake.....</p> <p>4 = A little foggy.....</p>	<p>How difficult was getting up this morning?</p> <p><u>5 Point Scale</u></p> <p>2 = Easy</p> <p>3 = Moderate</p> <p>4 = Difficult</p>	<p>Which is the best description of the quality (not quantity) of your sleep last night?</p> <p><u>5 Point Scale</u></p> <p>2 = Better than normal</p> <p>3 = Normal</p> <p>4 = Worse than normal</p>

This second study into the afternoon pressure for sleep examined the contributions made by 'meals' and 'prior sleep loss' to the induction of afternoon sleepiness. Forty-eight subjects were randomly divided into three equal groups and assigned to differing nocturnal sleep schedules: a normal nights sleep; a 2-hr sleep reduction and a 4-hr sleep reduction. At noon-time, half the members of each of the three 'sleep regimens' were given a large meal to consume, whilst the other half dined on a small lunch. Gender differences were also considered across all the groups.

The nature of the lunch menu and the level of sleep restriction were formulated to be as realistic as possible. The guiding tenet underlying the experimental design has been the principle of mimicking real-world experiences. To that end, the meals chosen were familiar and acceptable (cf. carbohydrate and macronutrient loading experiments^{219, 224-5}) and the level of partial sleep restriction bore close resemblance to the daily experiences of students, doctors, career-professionals and even members of siesta cultures.

Temporal Characteristics of the Post-Prandial Effect.

With the exception of the SSS, all the other tests (RT, RT-Slowest 10%, MSLT and VAS) were consistent in highlighting the 1300 h–1330 h period as the most vulnerable zone of afternoon impairment. The worst performance scores and the sleepiest disposition surfaced at this point in the afternoon. Focussing on the MSLT, we see that the shortest sleep latency occurred at 1330 h (fig. 3.18; p.155). Other studies confirming this 1330 h trough, include: Carskadon and Dement²³⁵, Carskadon et al.²⁸³ — a 1400 h trough was found by Zwuyghuizen-Doorenbos²⁸⁵, Hartse²⁸⁷, Carskadon and Dement³¹¹, Stepanski et al.³¹², Levine et al.²⁷⁴, Clodore et al.³¹³. Generally the afternoon window of MSLT sleepiness encompasses the period 1300–1600 h.

In the earlier experiment described in Section One, I found the afternoon dip to maximise at or around 1500 h. The veracity of such a time window was given credence by Lavie's data on the 1500 h 'Secondary Midafternoon Sleep Gate'^{111, 112} described as phase locked (180°) to the mid-point of nocturnal sleep. Field data from sleep surveys, vehicular accidents, braking errors by train drivers, and meter reading lapses would corroborate this 1500 h low point (see page 97). To dwell on the differences between 1300–1330 h and 1500–1530 h might seem superfluous. However, these differences do invite useful speculation.

To begin, Carskadon and Dement^{235, 166} collected normative MSLT data at six time points (0930–1930 h) and noticed the biggest dip occurred soon after lunch, at 1330 h. On a

second occasion, the same subjects were confined to a bed throughout the day, and fed with small, but equal portions of food, on the hour. During this 'constant routine' condition, MSLT's were carried out as before. This time however, they discovered the dip to occur at 1530 h. In another experiment consisting of 'Baseline—Sleep Loss—Recovery', Carskadon et al.²⁸³ found the lowest MSLT latencies to occur at 1330 h during baseline and recovery days. However, following sleep restriction, the shortest latency occurred at 1530 h.

From these observations, it could be argued that when the barriers to sleep are lessened either through confinement to bed as in the constant routine paradigm, or through sleep deprivation, or boredom induction in the confines of a climatic chamber, then the true, endogenous, biphasic sleep clock emerges. A 1500 h sleep propensity is clearly evident in these instances. Under normal, baseline conditions, or non-restrictive conditions as in the present experiment, masking of this true clock is feasible. Alternatively, the 1300h low point could be an artifact of the experimental procedure. Following lunch subjects returned to the laboratory, where their EOG and auricular reference electrodes were re-attached. In essence, the sedentary nature and relative inactivity of the preceding hour, together with the visually dull confines of the laboratory (see Horne¹⁷) may have contributed to the early unmasking of the inevitable APS. Also worth noting is the fact that in most tests, signs of a secondary move towards sleepiness emerges around 1600–1630 h. Perhaps, this is the true APS window (late sleeping students are known to dip late in the afternoon, around 1600 h; see Dinges²³). On the other hand, the different reports of maximal sleepiness, prominent at 1330 h, 1400 h, or 1530 h, could simply be the result of sociocultural factors, related to the time and size of habitual lunch. Americans start work earlier than Europeans, and the latter also regard lunch as a main meal, whilst many Americans, quite literally, eat a small lunch on-the-hop. These factors must be weighed when trying to localise the APS within a temporal framework.

Meals

According to main effects from the RT test (slowest 10% of scores) and the bipolar sleepiness scales (VAS), a large meal significantly lowered the level of alertness, $p < .05$ (see figs. 3.14 & 3.26). A more detailed picture emerges when one examines the interaction results from the various anova tables. From the full RT data it is clear that the effects of meals are modulated by gender. Essentially, female subjects are more prone to the deleterious effects of a large meal, $p < .05$ (figs. 3.10 & 3.16). Non-significant trends from the VAS (fig. 3.29) and SSS (fig. 3.24) support the significant RT data in highlighting the fact that females are most affected by a large meal. As a follow-up to these results, a secondary analysis was conducted: specifically, the data from female subjects were isolated and examined to see whether the effects of meal size

were more prone to occur around the first post-lunch test (c. 1300h). The subjective measures, SSS and VAS, clearly indicated that the 1330h trough was significantly exacerbated by a large meal ($p < .01$ and $p < .05$ respectively), and the scores of the 'RT-slowest 10%' lent strong support to these subjective findings ($p = .07$). The full RT data and MSLT test did not reach statistical significance in this respect. Further examples and explanations of this post-lunch susceptibility by female subjects follow below.

In a study looking at the effects of carbohydrate and protein-rich meals, Bonnie Spring and co-workers²²⁴ also examined the interactions with sex. They found that females were especially susceptible to the effects of carbohydrates compared to male subjects: in particular, females became significantly sleepy after these meals, whilst males became 'calmer'. The authors concluded that the effects of meals are 'blunted for males because of their greater body weight'.

In the present study, the negative reaction by female subjects to the large meal may well have been due to the fact that they were not used to eating such big meals. Although all the female volunteers were of normal weight (relative to height), nonetheless, it remains true that they were generally smaller in frame size and weight, than male subjects, and this could also reflect different eating habits. The 'unusualness' of the large meal was a problem mainly confined to females, and was evident by their adverse response.

A recent study on 'the post-lunch dip' by Craig and Richardson²⁰⁷ reported on the interplay between habitual meal sizes and experimental meal challenges. They presented twelve 'habitual light lunchers' and twelve 'habitual heavy lunchers' with a series of tests after they consumed a big lunch and a small lunch. A big lunch was found to increase the number of omission errors, whilst a small lunch lowered the error rate. However, the effects were found to be modulated by 'habitual meal sizes', in that the level of impairment was greater when the unusual meal was eaten. Crucially, the investigators noted that the largest impairment occurred in habitual light lunchers eating a big meal – their light lunchers would resemble the female subjects in the present study.

The adverse reaction of the female subjects to the unusual meal is best interpreted under the 'conditioning' hypothesis. In a recent paper, entitled; "The Eating Paradox: How We Tolerate Food", Woods²³² has presented a plausible case for the role of conditioning in food intake. His model offers a possible framework by which to interpret the 'gender data'. The basic premise of his thesis is that eating of food brings about disruptive and potentially dangerous changes to the delicately balanced interior milieu. For instance, the ingestion of food causes thermic changes which perturb body temperature; the sudden elevation of glucose and other fuels in the blood disturb the 'normally low blood fuel levels'; and the physical load weighted in the gut is incapacitating. Indeed, evidence is also provided for the elevation of 'stress hormones' (under sympathetic control) during meal times. In what is described as *behavioural tolerance* we are said to

have developed behavioural and physiological strategies to minimise the impact of ingested food. In its homeostatic drive, the body tries to check any major changes to the internal environment. As an example, elevations of blood glucose is countered by the release of insulin. Specifically, it has been discovered that humans release insulin (‘cephalic insulin’) in *anticipation* of eating; it is a response that is mediated by food-related cues, eg. ‘odors’ and even ‘time-of-day’. Again, if food-induced thermogenesis raises temperature, the body may, in anticipation, strive to lower temperature at lunchtime. Such bodily mobilization prior to food intake serves to hasten the elimination of fuels and dampen its ‘impact’. Craig and Richardson have also alluded to ‘physiological protection’ afforded by conditioning to meals. Returning to the present data, we see that a subject (based on his or her prior conditioning) may, in physiological terms, over-compensate or under-react, when presented with an unusual meal. According to Craig and Richardson,²⁰⁷ “the dip will be greatest for light lunchers eating a heavy meal [*eg. females*] and least for heavy lunchers eating a light snack [*eg. males*]” — italics mine.

To see if female subjects are indeed accustomed to eating smaller meals by habit, and whether male subjects do eat larger meals; I conducted a retrospective survey on the luncheon behaviours amongst the campus population — from which, incidentally, the experimental subjects were also drawn, eg. university halls of residences.

Table 3.16 Students (N=167) surveyed on the quantity of food eaten at lunchtime

<div>Question</div> <div>On average, how much food do you eat at lunchtime?</div> <div> <div>a. A Large Amount</div> <div>b. A Moderate Amount</div> <div>c. A Small Amount</div> </div>			
Subjects	Large Amount	Moderate Amount	Small Amount
Females (N=89)	3.37 %	55.06 %	41.57 %
Males (N=78)	25.64 %	65.39 %	8.97 %
All (N=167)	13.77 %	59.88 %	26.35 %

Most students profess to eating a moderate quantity. However, a closer look at the data indicate clear gender differences. It is apparent from the above table, that over a quarter of males lunch on big meals, whilst only 3% of females do the same. Moreover, 41% of females generally dine on small meals, as against just under 9% of males.

Table 3.17 Students (N=166) surveyed on the type of food eaten at lunchtime

Question			
What type of meal do you typically eat at lunchtime ?			
a. A Nibble (eg. an apple or snack bar)			
b. A Snack(eg. a sandwich)			
c. A Cooked Meal ...(eg. a meat or veg. dish)			

Subjects	Nibble	Sandwich Snack	Cooked Meal
Females (N=88)	3.41 %	77.27 %	19.32 %
Males (N=78)	1.28 %	32.05 %	66.67 %
All (N=166)	2.41 %	56.02 %	41.57 %

Another prominent difference distinguished the ‘type’ of lunch eaten. While around 67% of males consume a hot cooked meal, only 19% of females did likewise, with twice as many females favouring a lighter, sandwich type meal (77% vs. 32%).

Table 3.18 Students perception of a standard menu; akin to the one used in this study

Question			
Examine this menu		Would you consider this menu as being:	
Burger in a Bun		a. A Large Meal	
Chips		b. A Moderate Meal	
Salad		c. A Small Meal	
Fruit Yogurt			

Subjects	Large Meal	Moderate Meal	Small Meal
Females (N=34)	52.94 %	47.06 %	0.0 %
Males (N=77)	18.18 %	79.22 %	2.60 %
All (N=111)	28.83 %	69.37 %	1.80 %

In the present study, the large lunch condition contained a menu similar to the one in table 3.18. When students were confronted with this menu, their assessments differed strongly, along the lines of gender. Females were equally split in viewing the menu as large to moderate. Contrarily, most male respondents did not consider this menu to be a large one. Nearly 53% of females rated it as large, as against just 18% of males.

The results of this survey point to well-defined differences between males and females with regard to eating behaviours. If females are unused to eating large meals, then they may have reacted against the big test meal. In terms of the hypothesis outlined earlier, female subjects may well have 'over-reacted' to the raised levels of blood glucose, and the behavioural manifestation of this over-reaction, was increased reports of sleepiness. But why should sleepiness be manifest in this case? According to Woods,²³² any exercise or activity at this post prandial period is fraught with problems because there is the danger of 'sympathetic arousal' and the mobilisation of additional glucose into the blood stream. This would only add to the already abundant supply of circulating fuels. Given this scenario, the best possible solution lies in depressing behaviours after eating — and sleeping is an excellent form of immobility.

Christie and Mc.Brearty²⁰⁰ have provided data to support the proposition by Woods. If the body endeavours to maintain an optimal internal environment, and if it achieves this steady-state by acting in advance of any shock to the status quo, then we should witness important noon-time changes. For instance, body temperature should fall at lunchtime to counter the expected diet-induced thermogenesis and there should be an upsurge of insulin. Christie and Mc.Brearty monitored changes and trends in deep body temperature, and specifically recorded a lunchtime "interruption of the expected increase towards the evening zenith." Previously, Colquhoun¹⁰⁰ had also observed that those subjects who exhibit a post-lunch dip in temperature, always report the sensation of *sleepiness* to be their most prominent sensation. Christie and Mc.Brearty also measured levels of capillary blood glucose (CBG). During periods of post-prandial deactivation (as gauged by mood measures) they could not find any falls in CBG (hypoglycaemia). However, they did find the post-lunch deactivation to be accompanied by a large *change* in CBG, prompting the conclusion that "such a sharp change could reflect the operation of an insulin surge and that the lassitude is the effect, not of hypoglycaemia, but of the parasympathetic vagal initiation of the surge.." Other studies (Mejean et al.²⁸⁹) have confirmed that insulin secretion has its own endogenous rhythm, and is not slavishly looped to a blood glucose 'feedback-response'. Clearly, the noon-time temperature dip and the insulin surges, could be viewed as preparatory buffers to blunt the 'impact' of lunch-time food.

It is worth mentioning that some studies have suggested that gastric distension may promote sleepiness, through the stimulation of vagal afferents^{see 258, 212}. Perhaps, the stomach's capacity is reached sooner in smaller built female subjects. Recent work by DeCastro and Brewer (1992)²¹⁵ suggest that it is unlikely that gastric capacity is a problem — under social facilitation (dining with others), meal sizes can increase by 28% (in the company of one other diner); 41% (in the company of two); 53% (in the company of three) and 71% (in the company of five). That meal intake could be over

70% larger when dining in large groups, is clearly a remarkable affirmation of gastric and satiation flexibility, and supports the view that gastric limit is hardly ever reached.

The male subjects in the present experiment showed an improvement in RT speed following a large lunch, as against the small lunch condition. Similarly, Craig and Richardson²⁰⁷ also reported that their heavy lunchers (all male) were faster in a letter cancellation task following a large meal:

Lines completed after a Large Meal = 1857

Lines completed after a Small Meal = 1658

If male subjects are conditioned to a large lunch, then their positive performance scores, as seen here, should not seem remarkable. The improvement in performance after a large meal could also be due to the increased availability and uptake of glucose by both brain and muscle. Relative to its size, the consumption of glucose by the brain is extraordinarily high; per unit weight, its utilisation of glucose is ten times higher than the rest of the body. A study by Benton²⁹⁰ on psychological functioning following blood glucose manipulation found that glucose greatly improved performance in monitoring tasks like the simple RT test. In an extensive review on the role of glucose on CNS functioning, Siebert et al.²⁹¹ quote from studies using ¹⁴C-deoxyglucose which indicate that the attending brain places increased demands on glucose uptake. By monitoring EEG changes following glucose administration, central activation was evident (greater alpha intensity and decreased theta), so confirming the “vigilance-promoting effects of glucose on the CNS.” But, why was the physiological benefits of glucose, (viz. CNS activation and performance) only confined to males, and not evident in females? Furthermore, the hyperinsulinism that follows blood glucose elevation, also results in increased uptake of tryptophan across the blood-brain barrier, which ultimately increases serotonin synthesis, and the sleep inducing properties of this neurotransmitter are well known. The pursuance of the ‘glucose’ argument in relation to meals and psychometric performance, must be approached with extreme caution.

While a large lunch can improve performance speed, there is also evidence to indicate that it may alter performance *strategy*. In a letter cancellation task, Craig and Richardson found a significant increase in omission errors following a large lunch. Similar increases in errors were reported by Smith et al.²⁰² and Horne³¹⁴ (see table 3.19). In the current study, the nature of the simple RT test was such that it did not foster or conduce any errors; response speed simply varied. However, we know that sleepy individuals are particularly prone to ‘lapsing’ during psychological tests. Such lapses represent failures in focussed attention. A method employed by Dinges¹⁰⁶ to tease out these lapses in RT tests, involves filtering out the slowest 10% of scores. The scores in this ‘Lapse Domain’ do represent attention deficits, and thus may be used as a guide to

RT 'errors'. The treatment of long RT scores as indicators of 'lapses', is historically rooted in the Walter Reed school^{see 106}. An examination of the slowest 10% of scores in the current experiment indicated that a large lunch was responsible for the greatest lapses. So, whilst performance speed is enhanced following a large meal, it is also true that the response strategy changed towards laxity and carelessness.

Table 3.19 Recent examples highlighting the rise in errors following a large lunch

Changes Following a Large Lunch		
Study	Task	Errors
Present Study (1992)	Visual RT	↑ p<.05
Smith, A. et al (1991) ²⁰²	Search Task	↑ p<.05
Craig, A. et al (1989) ²⁰⁷	Letter cancellation	↑ p<.05
Horne, J.A. (1988) ³¹⁴	WAVT	↑ p<.05

In addition to the objective RT test, I probed subjective ratings of sleepiness with the SSS and VAS. As Craig and Richardson also employed a VAS test, I will compare the data from this particular instrument. These authors contrasted a morning pre-lunch VAS rating of sleepiness, with a single post-lunch rating. Likewise, I too conducted t-tests between a morning rating and an immediate post-lunch rating (at 13.30h). Our results were identical: namely, a large lunch was found to increase sleepiness whilst a small lunch improved alertness. These changes were not dependent on habitual meal sizes. Presented below is a summary table of the pre-lunch/post-lunch VAS scores — in addition to the 'overall' scores, I have also included the scores for the group obtaining a normal night sleep, in order to make the data more comparable with the aforementioned authors, as their subject pool were not sleep deprived.

Table 3.18 VAS Pre-lunch and Post-Lunch Scores: Big vs. Small Meals (means & se)

Sleep	Big Lunch [n=24]			Sleep	Small Lunch [n=24]		
	10.30h	13.30h	Change *		10.30h	13.30h	Change *
Normal Sleep	36.75 (7.4)	42.13 (6.3)	+ 5.38	Normal Sleep	33.38 (6.8)	29.75 (7.3)	- 3.63
Overall (Normal, -2hr, -4h)	42.92 (3.2)	43.46 (3.6)	+ 0.54	Overall (Normal, -2hr, -4h)	37.33 (4.7)	31.88 (4.1)	- 5.45

* +ve score = sleepy

-ve score = alert

In the case of the *objective* RT measures described earlier, the role of endogenous factors were prominent (eg. conditioning and individual metabolic response). With respect to *subjective* sleepiness, as gauged by the VAS, it seems that exogenous factors (meals) were more influential, than endogenous factors. In a correlational study, Freeman et al. examined the relationship amongst four sleepiness measures and concluded that perhaps subjective and objective measures “..reflect different facets of sleepiness”, particularly when diurnal sleepiness falls²⁹²; see also Hoch et al, 1992³²⁵.

The SSS failed to reveal any post-lunch differences in sleepiness. Other studies which have employed this instrument to gauge post-prandial sleepiness have also found it to be lacking in sensitivity (eg. Stahl et al.²¹², Bruck et al.²⁹³). Unlike the VAS, which specifically probes sleepiness on an ‘alert—drowsy’ continuum, the scope of the SSS is more heterogeneous. On closer examination, its seven statements (purportedly distinct), do in fact contain around 24 separate items; some of which probe ‘sleepiness’ whilst other items are more germane to elucidating degrees of ‘fatigue’ and ‘depression’ (see Horne⁸⁷, MacLean²⁹⁴, MacLean et al.³²⁶). For the subtle, post-prandial sleepiness under scrutiny here, the SSS is simply not ‘pure’ or focussed enough.

The MSLT also failed to reveal any meal-related changes. This finding is consistent with other studies. Stahl et al.²¹² could not find any lowering in sleep onset latency following a similar meal of hamburgers and chips. Likewise, polygraphic monitoring of 10 healthy control subjects (cf. narcoleptic patients) after a meal supplemented with 50g of glucose, also failed to discern any soporific properties resulting from the prescribed lunch²⁹³. More recently, an EEG study by Zammit et al. (1992)²⁹⁹ could not establish any meal-related rise in sleep propensity.

This experiment has highlighted the negative effects precipitated by a large meal — RT performance was impaired and sleepiness — as indexed by analogue scales (VAS) — increased significantly. However, these manifest signs of sleepiness were moderated by the effects of gender. A ‘conditioning’ explanation was advanced, and it was suggested that the effects of a large meal were essentially muted in male subjects because of their larger body size. To pursue the latter point further, would bring to the discussion the topic of bioenergetics, ie. metabolic need, BMR, energy expenditure etcetera. This is an area beyond the scope of the present discussion — however, the related question of thermogenesis needs comment.

One important consequence of food ingestion is the production of heat. This so-called ‘thermic effect’ of food, has been well documented by numerous studies; Piers et al.²⁹⁵ Kinabo et al.²⁹⁶, Dallosso et al.²⁹⁷, Belko et al.²⁹⁸ and Westerup-Plantenga et al.²²⁷ A recent paper by Zammit et al.²⁹⁹ takes the issue of thermogenesis one step further and

discusses its relationship to post-prandial sleepiness. Their results may have direct bearing on the present study, and will be briefly mentioned below.

Over 4 days, male subjects were given a high-calorie lunch (a liquid carbohydrate meal) on two days, and a low-calorie meal for the next two days. Following a high-calorie meal, subjects were seated on a recliner chair and asked to “stay awake”. On another occasion, following a similar high-calorie meal they were given the freedom to sleep if they so wished. The two low-calorie meals were followed by similar sleep/wake instructions on separate occasions. Baseline measures (1100–1145 h) were followed by post-lunch sleep tests (1200–1515 h). Their results were as follow:

1. Eleven of the twelve subjects fell asleep after both high and low-calorie meals. High-calorie meals did not reduce sleep latency.
2. High-calorie meals produced a greater thermic effect; increasing core temperature.
3. If sleep was allowed after meals, a sharp fall in thermogenesis occurred at sleep onset, resulting in body cooling.
4. The decline in thermogenesis at sleep onset occurred whether a high-calorie or a low-calorie meal was consumed.

Clearly, a large meal has a greater thermic effect, and this might pose a problem to subjects unused to such a meal aftermath. If high body temperatures resulting from a large meal was problematic to females, then the data from Zammit et al. would indicate that body cooling – which occasions post-prandial sleep – would be most propitious at this post-lunch hour. RT (lapse domain scores, fig. 3.16) and VAS trends (figs. 3.29, 3.26) are cogent indicators of post lunch sleepiness, and may be functionally related to this thermogenic model. The down-regulation of temperature may be a post-prandial encumbrance which is best achieved by sleep. If post-prandial sleep does have a temperature regulating mandate, then there may be wider teleological implications; it could augur well for the homeostatic view of sleep function²⁹⁹.

A summary of the main lunch-related effects seen in the afternoon are listed below:

- ❑ Female subjects are greatly impaired following a large meal, whilst male subjects show improvement in performance speed. The deleterious effects evident in female subjects, has been interpreted in terms of the ‘unusualness’ of a large meal. They are conditioned to a smaller food intake, and the mechanisms of ‘physiological protection’ aimed at minimising the impact of ingested food may have been unable to cope with the large meal. This argument was pursued in terms of the model presented by Woods with supporting data from Christie and McBrearty. By itself, the physical limits of gastric capacity would not satisfactorily explain the female response to a large meal.

- ❑ Male subjects do show an improvement in performance speed following a big meal, though this is achieved at a cost to performance accuracy (viewed from the perspective of increases to lapses in attention). Contributions to the enhanced performance speeds by elevations in central glucose uptake have been considered, but the evidence on this issue is subject to controversy.
- ❑ Subjective sleepiness show a marked increase soon after a big lunch, whilst subjective alertness tends to improve with a small meal. These subjective tests highlight the influence of exogenous factors (meals) whilst the objective RT test appear more vulnerable to endogenous influence (gender & prior conditioning).
- ❑ T-tests comparing pre-lunch scores with the first post-lunch test indicate that changes in scores may due to temporal circadian factors rather than the intervening lunch. In the MSLT and RT test, there were significant changes between the pre and post-lunch tests; but the changes for the big lunchers and small lunchers were in the same direction. So the mere passage of time, and perhaps the meal per se (*irrespective of meal size*) may be responsible for the performance impairment and lowered sleep latency at the *first* post-lunch test. However, as the afternoon progresses, additional tests, and subsequent anovas on just the afternoon changes, indicate that meal size may be influential; but any meal-related effect will be modulated by gender.
- ❑ I did not include a *no-lunch* condition because of the alerting effects of hunger; the conditioned expectation of lunch; and the zeitgeber function that meals play. Moreover, to deprive subjects of lunch, would (according to survey data on eating patterns) lack ecological validity. Therefore, no comment can be made on whether certain effects are meal-independent (cf. data from the Sussex Group; see introduction).

Sleep Restriction.

This experiment also sought to examine the effects of prior night sleep restriction on post-prandial sleepiness. The morning pre-lunch data was analysed separately from that of the afternoon (post-lunch) data. Overall, the effect of one night of partial sleep restriction was rather muted; the majority of significant findings were confined to the morning tests, with only one interaction apparent in the afternoon data (fig. 3.11).

The morning RT results, notably the slowest 10% of scores, produced the classic trend that common wisdom would predict; ie. RT became slower with increasing levels of sleep deprivation ($p < 0.05$; fig. 3.13). During the morning MSLT tests, female subjects showed a similar linear increase in sleepiness under progressive sleep reductions (see

fig. 3.17). The SSS data also indicated that sleep deprived subjects were subjectively more sleepy than normal controls ($p<.01$; fig.3.21), but curiously, it was the 2-hr sleep reduction group that was the worst affected, and not the 4-hr sleep restricted group.

The morning RT data ('overall scores') suggest that females fair worst after a 2-hr sleep reduction (whilst males improve), and that the performance of females is actually enhanced under a 4-hr sleep reduction (fig. 3.7). This paradoxical finding also extends to the afternoon data. From an examination of the non-significant trends, it becomes clear that throughout the data set, a 2-hr. sleep reduction produces the most deleterious results. This situation can be more accurately deciphered if we re-phrase our surprise and ask: why do the more severe 4-hr sleep curtailment group show signs of improvement and achieve better scores than the milder 2-hr sleep reductions. This curious result pointing to the benign effects of a 4-hr sleep reduction, is an undercurrent that runs through most of the data. These trends are distinctly illustrated in the subjective scales, SSS (fig. 3.23), VAS (fig. 3.28) and subtly evident in the MSLT scores (fig. 3.20) and RT data (fig. 3.12).

This unexpected trend purporting to show some kind of alerting response in reaction to one night of sharp sleep restriction was also evident in data presented by Hamilton, Wilkinson and Edwards³⁰⁰. They studied the effects of 2 regimens of partial sleep restriction spread over four consecutive days. Somewhat fortuitously, the regimens they instituted were very similar to the one used in the present study:

Hamilton et al	i. 7.5 hrs Sleep	ii. 6.0 hrs Sleep	iii. 4 hrs Sleep.
Present Study	i. 7.5 hrs Sleep	ii. 5.5 hrs Sleep	iii. 3.5 hrs Sleep.

Their results did suggest that a 4-hr sleep reduction was detrimental to performance, but most of the effects only occurred after Day 3 of the 4 day test. In many instances, Day 1 and Day 2 did not show any differences between the three sleep regimens. A case in point is the vigilance task which failed to show any significant differences on the first two days. Nonetheless, it is interesting to find that the subjects on a 4-hr. sleep budget out-performed the other groups in terms of detection rate (see fig. 3.31 overleaf). In a test called the Running Digit Span Task (a backward recall task), the group on a 4-hr sleep ration again performed well, and the baffled authors intoned: "...the results of this test are surprising.....Over the first three days, 4 hours of sleep gives the best performance followed by six and then 7 hours."

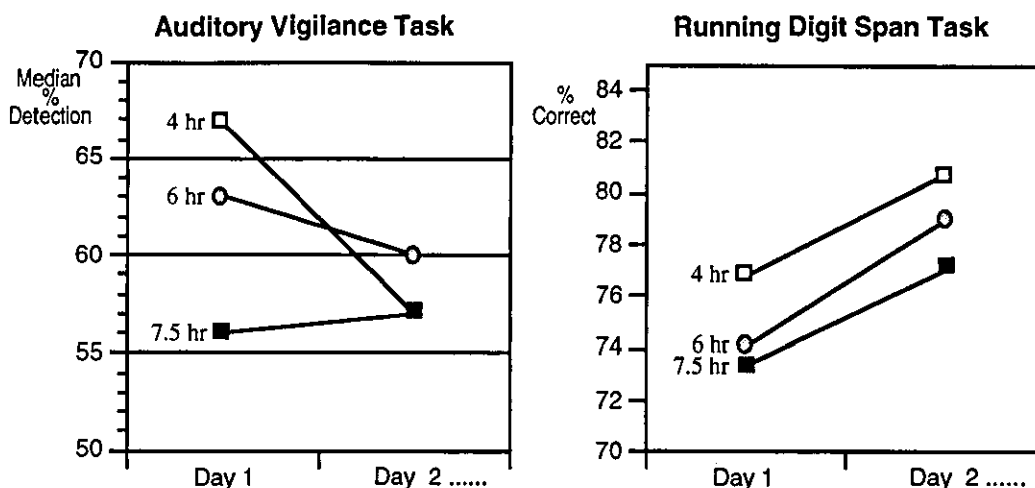


Fig. 3.31 Subset of the Hamilton et al³⁰⁰ data to show paradoxical effects of 4-hr sleep loss.

The somewhat capricious behaviour of subjects after one night of sleep loss, can partly be put down to ‘psychological factors’. A good example is provided by the pioneering work of May Smith (1916)²³⁸. In what is acknowledged to be the first study into partial sleep deprivation, she curtailed her sleep over three successive nights and diligently tested her performance, five times a week — *for 3 years!* She concluded that the fatigue resulting from sleep loss is characterised by two distinct phases. The first phase of fatigue “..acts apparently as a stimulant, so that work demanding concentrated attention is done more effectively than under normal conditions.” She also described her emotional feelings as one of ‘exhaltation’ allied with the pleasurable belief that she possessed “the power to conquer all things..” In the second phase, weakening occurs. Psychologically, the initial experience of fatigue seems to be a positive one.

We now know that sleepiness and performance impairment can be overcome, if there is an element of incentive; eg. monetary reward (Horne and Pettitt¹¹⁴), ‘knowledge-of-results’ (Wilkinson¹⁰⁹), or intrinsic mental challenge (May and Kline³⁰¹). Recently, Dinges et al. (1992)³⁰² reported that sleep deprived individuals do not necessarily lose the “willingness to perform”; if subjects felt sleepiness impinging and their performance was beginning to flag, then they usually expended more ‘effort’ to compensate.

It is also pertinent to ask whether the nature and perceptions after the 4-hr sleep differed in any way from the other sleep regimens. Soon after arising, subjects were asked to rate the ‘quality’ of their previous night sleep. Compared to the control group, subjects under 4-hr. sleep restriction, reported their sleep quality to be significantly better (see table 3.19 overleaf).

Table 3.19 Retrospective Reports of Previous Night Sleep Quality: Means (& se).

Question	Sleep Regimen	Quality of Sleep Obtained
<p><i>Which is the best description of the quality (not quantity) of your sleep last night ?</i></p> <p>1. Much better than normal</p> <p>2. Better than normal</p> <p>3. Normal</p> <p>4. Worse than normal</p> <p>5. Much worse than normal</p>	Normal	3.0 (0)
	-2 hrs	2.7 (.33)
	-4 hrs	2.4 (.16)
	<p>1-Way Anova <u>p<0.05</u></p> <p><i>Post-hoc</i> Normal vs -2hr = NS</p> <p>* Normal vs -4hr = p<0.05</p> <p>-2hr vs -4hr = NS</p>	

Recently, Van Diest and Appels (1992)³⁰³ reported that subjective assessments of previous night 'sleep quality' were not based entirely on the duration of the previous night sleep; specifically noting that reductions in EEG sleep did not necessarily result in a worsening of 'sleep quality'.

So why does a 4-hr sleep reduction lead to *improvement* in sleep quality, as evident in the results of the present study? It has been shown that subjects on a reduced sleep budget do have greater *sleep efficiencies* (TST/TIB), sometimes in excess of 90% (Roehrs et al²⁶², Levine et al²⁷⁴). The improved sleep quality could be a manifestation of changes to sleep architecture. In a study of gradual sleep reduction, Johnson and MacCleod²⁶⁷ noted that "...at 4 hours a marked increase in stages 3 and 4 was present with a decrease in REM.." The changes to sleep structure resulting from sleep reduction have now been well documented, and include the following: maintenance or increases to SWS, reductions to stages 1, 2, REM, and to wakefulness within the sleep period (see Carskadon and Dement²⁷⁶, Horne and Wilkinson²⁷⁰, Johnson and MacCleod²⁶⁷, Webb and Agnew²⁷⁷, Mullaney et al.²⁶⁹, and Carskadon et al.²⁸⁴).

Compared to a 2-hr sleep reduction, subjects under the more severe 4-hr sleep reduction, probably experienced a greater *sleep depth*, with a higher percentage of SWS and sizeable reductions to REM sleep. This much is apparent from the studies cited above. Sharp, well-phased sleep reductions, curtail REM sleep: antidepressant drugs also attenuate REM sleep. Coincidentally, mood is elevated by sleep reductions and by antidepressants. Therefore, it could be deduced, that the positive affect resulting from severe sleep loss and antidepressant drugs are caused by the shortening of REM sleep.

In 1966, Schulte observed that a teacher suffering from depression suddenly improved following a night of sleep deprivation; an observation later confirmed by Pflug and Tolle in 1971 following controlled trials (see Kuhs³⁰⁴, Wu and Bunney³⁰⁵ and Wehr³⁰⁶). A new method to treat depression, called 'Sleep Deprivation Therapy' soon emerged. A review of 61 studies by Wu and Bunney found that 59% of patients responded to this novel antidepressant therapy. It was particularly effective for endogenously depressed patients who maintained the typical diurnal variations in mood swings (morning lows, and evening highs); see Haug³⁰⁷, Wehr³⁰⁷. If full sleep is permitted (or if naps allowed) then a relapse follows. Therapies employing partial sleep deprivation (cf. total sleep loss) generally keep subjects awake in the latter half of the night. According to Szuba et al.³⁰⁸ sleep deprivation after 2.00 am is superior. The REM sleep in depressives is said to occur early (phase advanced), hence the early wake time. However, in normals, greater REM occurs towards the end of sleep time, and delaying bedtime (as in the present experiment) serves to augment and intensify delta sleep, with little time afforded to REM sleep; hence any beneficial effect is thought to be founded upon REM privation (cf. David³⁰⁹). This hypothesis remains very speculative, and to date, no satisfactory mechanism to explain Sleep Deprivation Therapy has been offered, although tentative models have been advanced (cf. Wu and Bunney³⁰⁵, Germaine³¹⁰).

In the present experiment, there were not many significant changes to post-prandial sleepiness following one night of partial sleep loss. Other studies have arrived at similar conclusions; Anch et al. have noted that: "Researchers have been unable to consistently demonstrate a behavioural effect of partial sleep loss"²⁵⁹. This is particularly true for performance tests. Carskadon et al.²⁸³ found no significant changes to performance (addition task, memory test, auditory vigilance) following sleep reductions to just 4-hrs. Likewise, Wilkinson et al.²⁷⁵ found the speed in an addition task only declined after sleep was shortened to 2–3 hours. In a chronic sleep reduction study (TIB= 5.5 hrs) Webb and Agnew²⁷⁷ could not isolate performance decrements in a battery of measures: a vigilance task did show impairment, but this was more apparent as the weeks progressed and the cumulative effects of sleep loss took hold.

Results from subjective measures, principally the sleepiness scales (SSS, VAS) and the MSLT, indicate that one night of moderate sleep restriction will not necessarily produce gross changes. In a week long study examining the effects of cumulative sleep loss, Carskadon and Dement²⁷⁶ curtailed nocturnal sleep to 5 hours (TIB= 0300h—0800h). This was preceded by 3 baseline nights. Daytime tests after the first night of sleep restriction failed to reveal significant changes from baseline for the MSLT and VAS test; the SSS did however show an immediate effect on the first day. By day 7, all tests were significantly different from baseline. The point in quoting this study is that it amply shows the cumulative effects of sleep loss. One night of partial sleep loss may

prove ineffectual in producing noticeable changes. In the study by Carskadon and Dement, sleep was reduced to 5 h. – in my study, the sleep of one group was reduced to 5.5 h. Therefore, it might prove instructive to juxtapose my results with those of the above authors; principally their data pertaining to the first night of sleep restriction.

Table 3.20 Contrasting means (sd) after normal sleep length and one night of ~2hr sleep restriction: Present study [n=16] vs. Carskadon & Dement²⁷⁸ [n=10]

Test	Normal Sleep		One Night Sleep Loss (TIB = 5.0 & 5.5 hr)	
	Carskadon & Dement	Present Study	Carskadon & Dement	Present Study
MSLT	16.7 (3.1)	15.8 (5.7)	14.3 (5.6)	14.0 (5.0)
VAS	32.3 (9.8)	32.0 (19.0)	37.9 (7.8)	38.6 (19.0)
SSS	2.4 (.5)	2.4 (.9)	2.7 (.4)	2.9 (1.0)

* Carskadon & Dement Study: MSLT scores are averages of 6 tests (cf. Present Study=4) and their subjective scales span 0800h – 2130h (cf. Present Study=1000h–16.30h)

The mean scores from the two studies are strikingly similar (the subjective ratings from the present study are more sensitive to the contrasting meal effects, hence the bigger observed variance). These concordant results attest to our ability to overmaster the acute effects of one night of sleep reductions.

✱

This experiment has shown that one night of acute sleep restriction will not necessarily amplify the afternoon trough that characterises the APS. The behaviour of subjects after a ‘full night sleep’ was not very different to those deprived of sleep: all groups were equally impaired at the 1300–1330 h trough. The greater dip predicted for subjects on a 4-hr. sleep loss, did not materialise — somewhat incongruously, there were indications of an improvement under this austere sleep regimen. This group also judged their nocturnal ‘sleep quality’ to be significantly improved, in marked contrast to the ‘normal sleepers’. In sum, the effects of sleep loss were muted. Psychological factors may have overridden any short-term effects of sleep restriction. We know that the effects of sleep loss are cumulative. Therefore, this experiment might have benefitted if sleep restriction was extended to 3 nights, whereupon graded sleep loss would have resulted in graded impairments, in a monotonic fashion. In siesta cultures, where the warmer climes favour evening leisure extending into the small hours, sleep loss becomes a chronic problem. Shift-workers are also prone to chronic sleep reductions. Unsurprisingly, napping during the APS period is a ubiquitous phenomenon in both these groups.

C onclusion

*

“By early afternoon, the hunt is over. A pride of lions having eaten their fill, gathers in the shade of a thorn tree. Other animals drowse, play or groom each other quietly. They have behaved this way in the early afternoon for millions of years, in a land where the heat of the sun rules all. Far from the tropics, Scandanavian factory workers make more mistakes on the job, German schoolchildren stumble over their arithmetic exercises and Greek merchants close their shops. In offices everywhere, people have returned from lunch, but alertness flags and daydreams intrude. Some people tell themselves they must have eaten too heavily...”[ref: 328]

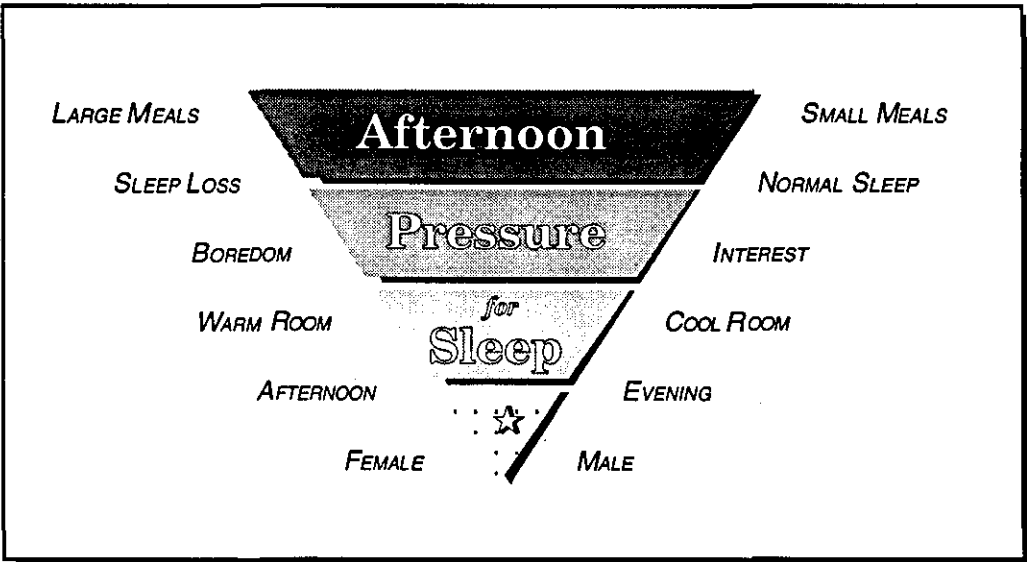
The investigations reported in this thesis represents an attempt to understand the transient, but enfeebling sleepiness that supervenes in the middle of the waking day. It was an experimental endeavour that involved over 100 individuals; all devoted a large part of their waking day to the enterprise. Laboratory studies variously spanned between 0900–2200 h.

The central hypothesis argued for the existence of a biphasic sleepiness rhythm – but more important was the contention that this secondary sleepiness was subject to powerful masking by environmental factors. It was further suggested that mere testing at various time points in the afternoon (as others have done) would not be sufficient to uncover the subtle diurnal variations in sleepiness.

In order to expose the endogenous APS, environmental agents known to thwart the expression of afternoon sleepiness had to be dampened and ‘sleepiness offered a course of least resistance’. If physiological sleepiness is present, it would be most vulnerable under a sleep-faciliatative or dearousing environment. To that end, the cognitive and physical environment was manipulated; at a time phased to coincide with the circadian low-point of the afternoon. Essentially, a bid was made to amplify the trough.

A number of experimental variables – hypothesised as sleep-facilitating – were selected for study. They were all deemed ecologically valid, in that they were common to daily experience (eg. boredom, hot stuffy rooms, big lunches).

Fig. 4.0 The range of variables that were manipulated in a bid to unmask the APS.



The first experiment attempted to use acute 'cognitive' boredom as an agent to unmask daytime changes in alertness, specifically targeting the advancing APS. Two independent groups were exposed to boring stimuli in the afternoon and early evening. Significantly, the soporific property of boredom only appeared during the APS window — exposure to identical conditions in the early evening failed to elicit comparable impairments. Interestingly, the baseline scores for the afternoon group (taken at 1235h) and the baseline scores for the evening group (taken at 1835h) were virtually identical. However, once inside the climatic chamber, and following arousal manipulations, the differences between the two time conditions emerged. These findings highlight the power of masking; afternoon sleepiness was only evinced *after* arousal manipulations. The failure of other workers to isolate the afternoon dip, must be seen in the light of possible masking effects.

Discussions of afternoon sleepiness are not only a matter of scientific debate; it also generates much speculation in lay society. The 'siesta' or afternoon napping in general, has often been attributed to the lunch-time meal and/or the soaring heat of midday. Contrary to these folklore beliefs, the results from this investigation cannot confirm that either a large midday meal or indeed that high temperatures serve to modulate the APS. However, the situation is not a clear-cut one, and any inferences which may be drawn, do carry important caveats. On the question of meals, it depends on who is doing the eating, and whether the meal consumed conflicts with normal dietary habit: a female subject unused to a large meal will react negatively to such a challenge. Plainly, afternoon sleepiness is not an inevitable consequence of eating a large meal, a fact also endorsed by other cross-cultural observations. In Spain, the afternoon siesta is customary; and yet the main meal of the day is eaten late in the evening; whilst in Germany, where lunch is the largest meal of the day, siesta breaks are noticeably rare²³. Furthermore, in a siesta-taking country like Greece, Soldatos et al. found that less than 1% of nappers actually attributed their afternoon repose to a heavy lunch²⁷.

With respect to temperature, it was found that a warm room *per se*, does not necessarily lead to sleepiness; infact, high temperatures are known to disrupt and fragment nocturnal sleep. However, in a round-about way, environmental heat can encourage the resort to sleep. In agrarian cultures, the intense heat of midday can be punishing, forcing farmers to retreat indoors and consequently presenting a 'window' or 'opportunity' for the APS to be expressed. Arguably, heat has a 'behavioural' role to play in this context, and may not be the engine of any 'physiological' drive to sleep.

Sleepiness has been shown to have an 'environmental' context, but more important, is the growing appreciation of the 'temporal' context in modulating its expression. To speak of sleepiness in the *afternoon* is unduly nebulous, as the *afternoon* encompasses the hours 12 noon to 1800 h. The APS is embedded within this wide range. A hallmark

of both the studies reported in this thesis, has been the frequency of testing intervals, designed to 'trap' the transient visitation of afternoon sleepiness. The results would indicate that the APS window can extend between 1330–1500 h. In the first experiment, where the environment was closely managed, the afternoon trough maximised at 1500 h. This time period concurs with the secondary rise in vehicular accidents at that hour^{154, 155} and with Lavie's 'secondary midafternoon sleep gate', also judged to occur around 1500 h.¹¹¹

In the world at large, three o'clock would seem to be a turning point: a let-down for many. It represents a time when we temporarily pause from work. The three o'clock tea break (so aptly timed) interjects at the height of this unwelcomed afternoon malaise. The tea we imbibe stimulates and animates us from our afternoon quiescence. In a warm country like Yemen, where the afternoon temperatures are gruelling, all work stops at one o'clock in the afternoon. Following the afternoon break for lunch, the majority population take to chewing a green narcotic leaf called *quat*, a stimulant which is said to combat the emergent feelings of afternoon lassitude³²⁷.

It is interesting to view the battle against the APS from this socio-anthropological standpoint. Those countries without an 'enshrined' siesta break resort to culturally sanctioned stimulants, from the urbane tea drinking ritual to the more stronger narcotic alternative. The reality for most people in industrialised societies, is that work must continue, regardless of the APS. In many instances, however, the consequences to any midday drops in human efficiency can be dire.

On April 29th, 1985, a truckdriver ploughed his vehicle into the back of a stationary schoolbus in Arizona, injuring 28 passengers⁹⁴. The driver was fatigued and reportedly slept poorly the night before. Crucially, the time of the accident was 3.15 in the afternoon. In another example, the editors of "Fine Woodworking" magazine, surveyed its readership on injuries sustained; from small cuts to amputations. A total of 952 injuries were reported, most involved table saws. Analysis of the 'time-of-accident' indicated that 34% of the accidents occurred between noon and 3 p.m.^{see 328} Remarkably, the actual proportion of woodworking between these hours was very low, due to the intervening lunch-breaks; the reduced work output certainly did not justify the high accident rate.

The circadian troughs in the current study emerged at 1.30 and 3.00 pm: times of especial vulnerability which concur with the noon to 3 pm. accident peak reported in the above field data. (see fig. 4.1 overleaf).

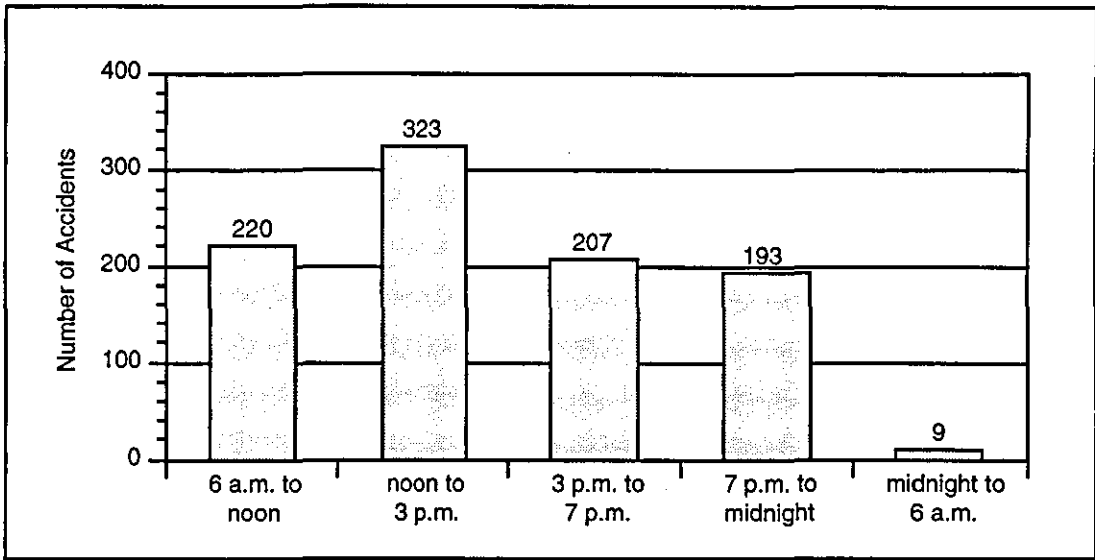


Fig. 4.1 The Post-Lunch Slip : Woodworking Accidents (Ref: 328)

Maintaining maximal alertness at all times is vital for air-traffic controllers, nurses working in ICU's, control-room workers in nuclear power plants and long-distance lorry drivers using motorways. In these occupations, especially where the role of operators is often reduced to one of 'system-monitor', boredom becomes a critical factor in predisposing performance failures. The first experiment, reported in the earlier part of this thesis, clearly demonstrated the ability of boredom to unwittingly 'expose' the intrinsic afternoon sleepiness. In a long-haul flight, eg. London to Bangkok, automation in the cockpit has reduced the pilots in-flight work load to a mere 10 minutes (landing and take-off), with the rest of the 12-hr. flight filled with unremitting boredom. Napping by the entire flight-deck personnel is not unknown.

In view of the practical issues raised by the intrusion of sleepiness at midday, it is surprising to discover the dearth in research effort to this area; a situation possibly arising from the fact that as a research discipline, the field crosses the schools of "chronobiology" and "pure sleep research". Moreover, the relationship of afternoon sleepiness to the major sleep at night, remains to be fully appreciated by researchers who still see 'sleep and wakefulness' as distinct phases, when in actual fact, they are complementary facets of circadian biology that are causally bound⁷⁵.

Another reason why afternoon sleepiness has been put on the 'back burner' for such a long time, has to do with its transient nature. It is a "now-you-see-it, now-you-dont" phenomenon, that often goes unrecognised; rousing Campbell and Zulley to ask: "What kind of rhythmic system includes one component which, in normal adults, is most conspicuous by its frequent lack of occurrence."³²⁹ Such observable inconsistencies are easily explained by social masking, and the fact that the APS window, on closing,

gives way to the rising arm of circadian activation (climbing to the early evening acrophase) which appears to mollify the earlier ennui. If one feels sluggish and listless at 2.30 p.m., but knows from experience that by 3.30 p.m. all will be well – why, (so the logic goes) make a fuss! But the issue of afternoon sleepiness is far from trivial. Recently, a committee report by the Association of Professional Sleep Societies, entitled; “Catastrophes, Sleep and Public Policy”⁹⁶ highlighted several examples of midafternoon performance failures, and urged in their conclusions: “Laboratory and field research is needed to determine the process that produce the bimodal temporal pattern in so many facets of human function. Identification of the mechanisms involved will facilitate efforts toward developing effective countermeasures to minimise the often catastrophic consequences..”

The investigations reported in this thesis do not claim to – or were ever intended to – contribute to the full scope of circadian rhythmometry. To do so would have involved 24-hr monitoring, requiring complex inferential statistical procedures in the traditional manner of chronobiology. Rather, the aim was to examine a small, but discrete window of diurnal change, as part of the larger and grander circadian phenomenon.

These investigations were also methodologically orientated and innovative, insofar as common elements of the environment were employed in a controlled (and contextually correct) fashion, to unmask the secondary component of the biphasic sleep tendency. Attempts were also made to keep the experimental manipulations as ‘naturalistic’ as possible, with real-world parallels. Notwithstanding such goals, aimed at experimental realism, any real-world recommendations that can be extrapolated from the present results, must be tempered, given the controversial nature of the tests and to an extent, the unrepresentative nature of the student volunteer groups.

Early interest in diurnal studies, like the present one, were driven by the real need to understand the daily fluctuations in industrial work output¹⁵². There were also important pedagogical questions; educationalists were concerned about school hours and the timetabling of subjects. Recently, French workers (Montagner et al., 1992³³⁰) proposed that the morning schoolday should cease all intellectually demanding work at 1130 h, and following a lengthy midday break, school should resume at 1500–1730 h. As part of their investigations, they unobtrusively monitored a class of first year primary school children using video cameras. Their observations revealed that 68% of the children were overcome by bouts of yawning between 1430 to 1500 h. Similarly, Hellbrugge et al.^{see 330} found 80% of children between 2 to 11 years of age experienced sleepiness in the early afternoon. Data from the current investigation, presented in this thesis, would not contradict these findings collected in the field, which purport to show some form of afternoon dearousal. However, a great amount of evidence will have to be amassed before serious recommendations to changes in public policy can begin.

Afternoon sleepiness has been regarded throughout as a transient and elusive biological phenomenon, that still confounds many by its apparent absence. The fact that it is not so pervasive, I have put down to masking by the social and occupational demands of an industrialised society. But the suppression of afternoon sleep may have a longer history

It has been suggested that by foregoing the afternoon nap, early Man probably created a new "midday niche" which was actively exploited by hunting and scavenging among the hapless and dozy population on the African plain³³¹. There was perhaps, very little choice in the matter; our hominid ancestors may have been compelled to open up this midday hunting niche. According to Foley³³² there was an inordinate rise in bioenergetic requirements associated with hominid evolution, which necessitated an "increased foraging efficiency" and the consumption of "higher quality foods, in substantial quantities." The opening up of the "supposedly vacant noonday scavenging niche" ^{see 333} became an important strategic window for these foraging behaviours.

Even modern day hyenas have found this midday hunting niche ideal. A recent account of that fact appeared in a report aptly titled: 'High noon on the Maasai Mara', where the authors dramatically relate; "Lunchtime is supposed to be a slack period for wildlife-watching in Africa. All animal activity seems to be inversely related to the arc of the sun....As more and more animals take a nap, the hyenas move in and lie down between 50 and 100 metres from the resting animals without attracting much attention...Though most daytime attacks are against topi, we have also seen hyenas take Thompson's gazelles and impala at midday"(ref: 334). The hunting and scavenging niche of early diurnal hominids, have recently been contrasted with those of hyenas, by Blumenschine and Cavallo, 1992³³⁵. Hominids occupying the same hunting niche may have been responsible for the extinction of numerous hyena species. Our emergence onto the open savannah, also called for sophisticated thermoregulatory controls, especially with respect to cooling the brain. Recently, Falk³³⁶ has advanced the most likely development of brain cooling mechanisms, in the evolution of hominidae.

Today, however, the active suppression of afternoon sleepiness continues, with the assault on the modern-day siesta at its greatest. In Greece and China it is currently being legislated out, by governmental decree. In a world that operates on a 24 hour basis, where we are forced to colonize even the nighttime hours, we cannot afford this midday interlude to our stream of consciousness. Nevertheless, the chronobiological urge to nap is there; a programme lying somewhere in the inner recesses of our ancient brains. If our 'rhythms still follow the African sun' of time gone by, then afternoon conflicts may yet surface as our more powerful cortex of today, say "no" to the genetic programs that first governed our daily tempos^{328, 337}. Perhaps, like a radio astronomer studying the Big Bang, this investigation into afternoon sleepiness also represents a critical attempt at tracing the faint and dying echoes of a very distant call to sleep.

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Appendix

Appendix 1

Questionnaires

1. THE STANFORD SLEEPINESS SCALE

The SSS Questionnaire

Name.....

Date.....Test.....


Condition Code.....

Listed below are a set of feelings which reflect various degrees of Alertness & 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 REMAIN AWAKE.

2. VISUAL ANALOGUE SCALE (100mm)

Please answer the question below by placing a vertical mark on the line

Wide Awake  Very Sleepy

3. THE AKERSTEDT DROWSINESS SCALE

The ADS Questionnaire

Name.....

Date.....Test.....

Condition Code.....

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

☐ 1. EXTREMELY ALERT

☐ 2.

☐ 3. ALERT

☐ 4.

☐ 5. NEITHER ALERT NOR SLEEPY

☐ 6.

☐ 7. SLEEPY, BUT NOT FIGHTING SLEEP

☐ 8.

☐ 9. EXTREMELY SLEEPY, FIGHTING SLEEP, EFFORT TO STAY AWAKE

4. THE LEATHWOOD MOOD QUESTIONNAIRE

The LMQ

Name.....

Date.....Test.....

Condition Code.....

For each of these subjective sensations, please circle the point which best represents your present state.

	Very much	Moderately	A little	Neither on nor the other	A little	Moderately	Very much	
SLEEPY	WIDE AWAKE
LETHARGIC	VIGOROUS
HUNGRY	SATIATED
MUDDLED	CLEAR HEADED
TIRED	ENERGETIC
UNIMAGINATIVE	IMAGINATIVE
TENSE	CALM
ILL AT EASE	FEELING FINE
INEFFICIENT	EFFICIENT
FEELING BORED	AROUSED

5. THE POST-SLEEP QUESTIONNAIRE

Post-Sleep Questionnaire

Name.....

Date.....

Important:

Please do not complete this until you have been awake for 30 mins. and have got up.

Which one of the following statements do you consider best describes your feelings now?

- ☐ 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 REMAIN AWAKE.

Please fill in the appropriate times as accurately as possible:

At what time did you – Fall asleep last night? _____
Wake up this morning? _____
Get up this morning? _____

How difficult was getting up this morning?

Very Difficult	Difficult	Moderate	Easy	Very easy

Which of the following do you consider to best describe the QUALITY (not quantity) of your sleep last night?

Much better than normal	Better than normal	Normal	Worse than normal	Much worse than normal

Appendix 2

Materials For Arousal Manipulation

The induction of cognitive boredom and the elevation of arousal (mental 'interest') was achieved by presenting subjects with a variety of material; from basic reading matter, to games and videos. The items were chosen on the basis of prior surveys of student leisure interests. A questionnaire booklet was completed by 89 female students.

The first questionnaire –The Yawn Sparkle Questionnaire – was of a preliminary (pilot) nature and was completed by 32 female students. This broad and cursory questionnaire probed subjects on general themes such as:

1. Name a film (or type of film) you regard as interesting?
2. Name an interesting 'light read'?
3. Name a board game you like?
4. Name a 'boring read' (no books)?

Other questions asked subjects to rate various music types (eg. classical, jazz....).

A more detailed questionnaire booklet was then developed, based on the responses from the above questionnaire, and completed by 57 volunteer subjects. For instance, subjects were asked to rate (or rank order) a selection of *specified* magazines, videos, etcetra. The list of available videos is shown on the page overleaf – subjects were asked to rate all titles. The most popular video was a BBC comedy called "Fawlty Towers"; with one exception all the subjects that were assigned to the 'interest condition' chose to view this video: all the titles listed overleaf were available in the climatic chamber.

The video named as the most boring was an "Open University programme on maths". A university lecture on 'complex numbers' and a video on 'water channel experiments', made by the university Audio Visual Department for the Engineering Faculty, was included in the stimulus battery designed to procure cognitive boredom. A full listing of the materials selected for cognitive manipulation is presented below and overleaf.

VIDEOS : "BORING"		
VIDEOS		
Sand Channels:	Venturi Oriface Models (no Audio)	Loughborough Univ.
Sand Channels:	Pradform (Audio)	Loughborough Univ.
Complex Numbers	Open University (maths)	BBC.

Videos		1	2	3	4	5
		Very Interesting	Moderately Interesting	Indifferent	Moderately Dull	Very Dull
Films						
Play It Again Sam (comedy)	- Starring Woody Allen & Diane Keaton					
Raiders of the Lost Ark (adventure)	- Harrison Ford in a Spielberg classic					
The Maltese Falcon (thriller)	- Bogart as private eye Sam Spade					
Educating Rita (comedy)	- Julie Walters ; an Open Univ. student					
The Producers (comedy)	- Starring Gene Wilder & Zero Mostel					
Roman Holiday (romantic comedy)	- With Gregory Peck and Audrey Hepburn					
Comedy						
Fawlty Towers	- John Cleese, 3 episodes available.					
Sgt. Bilko	- Phil Silvers in the fast moving comedy					
Laurel and Hardy	- Numerous short films					
Hancock's Half Hour	- Tony Hancock in the 60's comedy hit					
Documentary						
A Train Journey in Peru	- Stunning scenery; magical S. America					
The Sex Life of Plants	- Plant pollination; colourful photography					
Animal Intelligence	- Horizon programme (BBC).					
Mysteries of the Mind	- An exploration of the human brain					
Incantation: Music of the Andes	- More S. American scenery and music					
The Meaning of Dreams	- QED programme (BBC)					
Human Sexual Attraction	- QED programme (BBC)					
Eyewitness Testimony	- The psychology of human perception					
The Miracle of Life	- Development of the foetus in the womb					
Biological Rhythms	- Human biological clocks					
The Inner Eye	- 3 programmes on psychology					
The Double Helix	- Drama/documentary; discovery of DNA					
Music						
Dire Straits in Concert	- From their hit album "Brothers in Arms"					
Pop Hits	- A selection of numerous pop videos					
Paul Simon in Zimbabwe	- From the album "Graceland"					
The Early Beatles	- Documentary with music					
The Pretenders	- Their major hits					

READING MATERIAL : "INTERESTING"		
Cosmopolitan	Vogue	Womans Weekly
Woman	Punch	Nat. Geographic
Marie Claire	Womans Own	Essentials
Elle	New Society	Best
Womans World	Company	Womans Realm
New Woman	Prima	Bella
Options	She	Spectator

READING MATERIAL : "BORING"		
MAGAZINES		
Investors Chronicle	Turf and Track	Stamp Magazine
Auto Classic	FT. Share Index	The Bell Ringer
Books		
Modern Prestressed Concrete	J.R. Libby (1971)	Van Nostrand, USA.
Fluid Mixing Technology	J.Y. Oldshue (1983)	Mc.Graw-Hill, NY.
Kinematics & Dynamics of		
Planar Machinery	P. Burton (1979)	Prentice-Hall, USA.
Engineering Thermodynamics	N. Hall, W. Ibele (1960)	Prentice-Hall, USA.
Punched Cards: <i>Their Application</i>		
<i>To Science & Industry</i>	R. Casey et al. (1958)	Reinhold Publ., USA.

GAMES : "INTERESTING"		
Scrabble	- A word game	J.W. Spear & Sons, UK.
Mastermind	- A code-breaking game of logic	Waddington Games, UK.
Othello	- An international strategy game	Peter Pan Playthings, UK.
Cluedo	- A detective board game	Waddington Games, UK.
<u>Various</u>		
Draughts.....Dominoes.....Playing Cards.....Backgammon.....Chess.....Jigsaws		

Appendix 3

Experiment One: A Summary of the Significant ANOVA Results

FACTOR	RT	df	SSS	df	ADS	df	VAS	df	HR	df	T [°] au	df
(T)emp		1,40		1,40		1,35		1,34		1,38	.07	1,33
(In)terest	.05	1,40	.01	1,40	.01	1,35	.01	1,34	.05	1,38	.05	1,33
Time/(D)ay	.01	1,40		1,40		1,35		1,34		1,38		1,33
Test (S)ess.	.05	3,120	.001	3,120	.001	3,105	.001	3,102	.001	4,152		3,99
TxIn		1,40		1,40		1,35	.08	1,34		1,38		1,33
TxD		1,40		1,40		1,35		1,34	.05	1,38		1,33
InxD	.05	1,40		1,40		1,35		1,34		1,38		1,33
TxInxD		1,40		1,40		1,35		1,34		1,38		1,33
SxT		3,120		3,120		3,105		3,102		4,152		3,99
SxIn		3,120	.05	3,120	.06	3,105	.08	3,102		4,152		3,99
SxD		3,120	.05	3,120	.07	3,105		3,102		4,152	.05	3,99
SxTxIn		3,120		3,120		3,105		3,102	.07	4,152		3,99
SxTxD		3,120		3,120		3,105		3,102		4,152	.06	3,99
SxDxIn		3,120		3,120		3,105	.05	3,102	.05	4,152	.07	3,99
SxTxDxIn		3,120	.06	3,120		3,105		3,102		4,152	.06	3,99

Experiment One: A Summary of the Significant Mood (LMQ) Results

FACTOR	DF	Sleepy—Wide Awake	Bored—Aroused	Lethargic—Vigorous	Muddled—Clear Head	Tired—Energetic	Tense—Calm	Ill at Ease—Fine	Hungry—Satiated	Unimaginative—Imaginative	Inefficient—Efficient
(T)emp	1,40	.07		.05		.05					
(In)terest	1,40	.01	.001	.01		.05		.06		.01	.05
Time/(D)ay	1,40								.05		
Test (S)ess.	3,120	.001	.001	.001	.001	.001			.001		.05
TxIn	1,40				.05	.05					
TxD	1,40						.05				
InxD	1,40						.06				
TxInxD	1,40								.05		
SxT	3,120				.05						
SxIn	3,120										
SxD	3,120		.08		.05	.05		.06			.05
SxTxIn	3,120										
SxTxD	3,120				.06						
SxDxIn	3,120										
SxTxDxIn	3,120										

Experiment Two: A Summary of the Significant ANOVA Results

FACTOR	DF	RT	RT (Slow)	MSLT	SSS	VAS
(M)eal	1,36		.05			.05
(SL)eep	2,36					
(G)ender	1,36	.05				
Test (S)ess.	2,72	.05	.05	.001		.01
MxSL	2,36					
MxG	1,36	.05				
SlxG	2,36	.05				
MxSLxG	2,36					
SxM	2,72					
SxSL	4,72					
SxG	2,72			.05		
SxMxSL	4,72					
SxMxG	2,72		.05			
SxGxSL	4,72					
SxMxGxSL	4,72					

