

Original article

A comparison of sleep deprivation and narcolepsy in terms of complex cognitive performance and subjective sleepiness

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Abstract

Objectives: (i) To expose ‘normal sleepers’ to a 32-h sleep deprivation protocol and evaluate the impact of this deprivation on a complex performance task i.e. the paced auditory serial addition test (PASAT). (ii) To compare these sleep deprivation performance findings with historical data on the impact of sleepiness secondary to narcolepsy on PASAT performance measures. (iii) To investigate the recuperative effects of a brief nap period on both sleepiness and PASAT performance for the sleep-deprived subjects. (iv) To compare these post-nap effects with historical data relating to the impact of napping on both sleepiness and PASAT performance for subjects with narcolepsy.

Background: Previous research has demonstrated that sleepiness induced by sleep deprivation in normal sleepers may lead to cognitive impairment across a range of performance tasks. Sleepiness secondary to narcolepsy has also been noted to impair cognitive function especially for complex processing tasks. Direct comparison of the effects of sleepiness on performance between non-pathological and pathological sleepiness states is confounded, however, by methodological differences in research design especially in relation to levels of induced sleepiness and performance task selection. The purpose of the current study was to undertake a sleep deprivation study that achieved a methodological match with published data evaluating the impact of sleepiness on cognitive performance for subjects with narcolepsy. This methodological matching allowed for a more precise comparison of the impact of sleepiness on performance between non-pathological and pathological sleepiness groups.

Results: Normal sleepers required a 32-h deprivation protocol to develop a subjective level of sleepiness that equated with that identified by subjects with narcolepsy. This induced sleepiness in normal sleepers did not result in any significant decrement in complex performance, a finding that was in contrast to the performance decrement previously found in subjects with narcolepsy with equivalent subjective sleepiness ratings. A 20-min nap produced more improvement in both arousal and cognitive processing performance for the subjects with narcolepsy than for the current sleep-deprivation cohort.

Conclusion: This study identified significant differences in the impact of sleepiness on complex performance between non-pathological sleep-deprived subjects and subjects with narcolepsy. This paper explores these differences in relation to the potential for both quantitative and qualitative differences that exist in the nature of sleepiness between non-pathological and pathological sleepiness states. © 2002 Elsevier Science B.V. All rights reserved.

Keywords: Sleep deprivation; Narcolepsy; Sleepiness; Performance

1. Study rationale

Narcolepsy is a disorder characterised by an excessive and pervasive daytime sleepiness. Subjective reports of cognitive impairment associated with the disorder are widespread with individuals reporting difficulties with memory, concentration and general learning [1]. Despite these subjective reports of cognitive difficulties, laboratory based assessments of cognitive function in narcolepsy often fail to demonstrate any significant performance decrement [2–4]. One potential explanation of this discrepancy between subjective experience and objective findings is that

cognitive deficits associated with narcolepsy are not consequent to deficits in neurological functioning but rather reflect the more generic relationship between sleepiness and performance decline that is also evident in non-pathologically sleepy populations. The failure of general laboratory measures to identify performance decrements in narcolepsy may therefore occur as a consequence of research methodologies masking the expression of sleepiness for subjects with narcolepsy.

It has been well documented that daytime sleepiness in narcolepsy is highly labile and sensitive to environmental factors [5,6]. Laboratory test settings appear to be stimulating for subjects with narcolepsy and therefore performance test protocols for this clinical group often report no beha-

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vioural signs of sleepiness across the testing period [2,3]. To increase the external validity of laboratory based performance testing for subjects with narcolepsy, Hood and Bruck [7] developed a testing methodology that allowed for the expression of this lability of daytime sleepiness, inducing repeated states of sleepiness and non-sleepiness across the testing period. Using this methodology, Hood and Bruck [7] compared cognitive performance on a range of tasks in sleepy and rested subjects with narcolepsy (within subject design). One of the central findings of this study was that conditions during which the subjects with narcolepsy reported being sleepy were associated with significant decrements on complex processing tasks in comparison to performance measures when rested.

The empirical question that emerges from the Hood and Bruck [7] study is whether the performance decrements observed for subjects with narcolepsy are specific to the sleepiness associated with the disorder or simply reflect a more generic relationship between sleepiness and performance measures. Although there is a substantial literature base evaluating the impact of sleep deprivation on performance in normal sleepers [8], a theoretical comparison with the complex performance task decrements found in narcolepsy is confounded by both the diversity of performance tasks reported in the deprivation literature and the failure of published sleep deprivation studies to quantify the level of induced sleepiness.

The diversity of performance measures: Performance tasks utilised in the sleep deprivation literature vary across a substantial number of domains. Tasks differ across dimensions that include duration (influencing fatigue factors), intrinsic interest (impacting on motivation) and neuropsychological complexity. For example, Dinges et al. [9] report on a 30-s memory task, Lisper and Kjellberg [10] a 10-min reaction time task and Angus and Heselgrave [11] a 54-h continuous performance measure. Comparative analysis of the impact of sleepiness on performance between subjects with narcolepsy and sleep-deprived subjects therefore necessitates appropriate matching of performance tasks to eliminate the potential for task factors to confound the results.

The quantification of daytime sleepiness: In 1982, Dement and Carskadon [12] suggested that daytime sleepiness had been virtually ignored as a dependent variable in sleep deprivation research. A review of contemporary sleep deprivation research demonstrates that sleepiness continues to often be inferred rather than quantified as a dependent variable in sleep deprivation research therefore confounding the comparative evaluation of research outcomes. An ‘unspoken tradition’ exists within the published literature that the level of induced sleepiness is simply inferred as a function of the duration of the deprivation methodology and therefore there are few attempts to quantify subsequent sleepiness states, i.e. a 36-h deprivation methodology is assumed to induce a greater level of sleepiness than a 24-h protocol despite the lack of quantification of sleepiness. As knowledge of sleepiness as a physiological state has

developed, it has been demonstrated that sleep duration is not the sole determinant of sleepiness but variables such as sleep continuity and circadian timing are also powerful predictors of sleepiness. To increase the validity of comparative analyses of performance outcomes between pathological and non-pathological sleepiness, it is therefore critical to quantify and equate ‘sleepiness’ as the dependent variable of the sleep induction methodologies.

For the reasons cited above, the findings of the relationship between sleepiness and performance in narcolepsy, as demonstrated in the Hood and Bruck [7] study, cannot be theoretically compared to sleepiness and performance interactions in sleep-deprived ‘normal sleepers’. To undertake this comparison, some matching of both levels of sleepiness and performance criteria between the pathologically sleepy and non-pathologically sleepy groups is required.

2. Study aims

The aims of the current study are to:

- (i) Induce a level of sleepiness in normal sleepers that equates with the level of sleepiness of subjects with narcolepsy reported by Hood and Bruck [7].
- (ii) Evaluate the impact of this sleepiness for sleep-deprived subjects on the paced auditory serial task (PASAT). The PASAT has been demonstrated by Hood and Bruck [7] to be the most sensitive measure of sleepiness in subjects with narcolepsy.
- (iii) Compare the impact of a 20-min nap on both arousal and performance measures for sleep-deprived subjects and subjects with narcolepsy.

By establishing, in the current study, a matched total sleep deprivation protocol for normal sleepers with the historical data reported by Hood and Bruck [7] for subjects with narcolepsy, this study provides the potential for a significant comparison of the quantitative and qualitative aspects of sleepiness associated with pathological sleepiness in narcolepsy and non-pathological sleepiness states.

3. Paper organisation and nomenclature

This paper will refer to the initial Hood and Bruck [7] study on subjects with narcolepsy as Study 1 and the comparative analysis for sleep-deprived normal subjects reported in this paper as Study 2. From Study 1, the terms ‘Sleepy Narcolepsy’ and ‘Rested Narcolepsy’ will refer to the clinical subjects under different conditions of experimental sleepiness manipulation. ‘Sleepy Narcolepsy’ will represent subjects with narcolepsy who have been exposed to a non-stimulating environment involving the completion of a 25-min Wilkinson auditory vigilance task (WAVT). ‘Rested Narcolepsy’ will refer to subjects with narcolepsy who have just completed a 20-min nap. The term ‘Rested

Normals' will refer to the subjects without pathological sleepiness when they were tested without any sleep deprivation. 'Sleepy Normals' will refer to these same subjects when tested after 32 h of sleep deprivation.

The methodology section will provide a full description of the sleep deprivation protocol. Summary aspects of the methodology and results from Study 1 on narcolepsy will be incorporated where the information appears necessary to contextualise the sleep deprivation study. This inclusion will also allow for direct comparison of relevant findings. For full details on any aspect of the comparative study on subjects with narcolepsy, the reader is referred to the initial Hood and Bruck [7] paper.

4. Method

4.1. Study 1: narcolepsy protocol

A brief summary of the historical comparison study is provided here. Eight subjects with narcolepsy and eight controls participated. Subjects with narcolepsy were withdrawn from stimulant medication for 18 h prior to the commencement of the study. The testing day incorporated four testing sessions with each session structured into a sleepy and rested state. To induce the sleepy state, subjects with narcolepsy were located in a non-stimulating environment for 25 min during which they were free to read or listen to music. Following the free-time, subjects completed a 15-min WAVT. This task has been demonstrated to be sleep inducing for subjects with narcolepsy [5]. Following this task, defined as the sleepy condition, subjects underwent a 20-min test protocol. Subjects with narcolepsy were then allowed a brief nap, a 5-min refractory period, and then repeat testing was undertaken under rested conditions. This test protocol was repeated in the initial study four times across the testing day to allow for performance testing of a wide range of tasks under different arousal conditions.

4.2. Study 2: sleep deprivation protocol

4.2.1. Subjects

Sixteen subjects participated in the sleep deprivation study. Eight subjects undertook the sleep deprivation protocol and eight subjects acted as controls. Whilst no formal sleep testing of participants was undertaken, all subjects underwent a clinical interview prior to participation and selected subjects presented with stable sleep patterns and no evidence of sleep disorders. Control subjects were matched to the sleep-deprived subjects using the criteria of age (M sleep-deprived subjects = 30.5 years; M sleep deprivation controls = 32.13 years); IQ, as measured using the K-Bit Brief Intelligence Scale (M sleep-deprived subjects = 93.38; M sleep deprivation controls = 103.25), and gender (both sleep-deprived and control subject groups comprised six female and two male participants). Using

paired t -test analyses, no significant differences existed between deprivation and control groups on the variables of age ($t(7) = 1.08$, ns) or IQ ($t(7) = 0.79$, ns)

4.2.2. Performance tasks

The central focus of this study is a comparative analysis of sleep deprivation effects on performance for the PASAT. This task, which in Study 1, was shown to be most sensitive to sleepiness effects for subjects with narcolepsy, was originally developed to provide a measure of sustained attention and speed of information processing. The task requires the subject to respond verbally to an externally paced auditory addition task and simultaneously inhibit the automatic encoding of their response and direct attentional resources to the next incoming stimulus [13]. Matching of testing protocols between studies was achieved by incorporating two filler tasks with the PASAT in Study 2 to allow for consistent testing duration (approximately 20 min) to that utilised for subjects with narcolepsy. Both the timing of testing sessions in relation to circadian factors and test/retest frequency and duration were matched between the two study protocols.

4.2.3. Procedure

As no estimates of the quantitative relationship between sleepiness associated with narcolepsy and sleepiness secondary to sleep deprivation in normal sleepers exist in the literature, substantial pilot work was undertaken to identify the period of deprivation necessary to equate with the levels of sleepiness reported by subjects with narcolepsy in the Hood and Bruck [7] study. A sleep deprivation period of 32 h was derived from this pilot testing. (See Section 5 for details.)

Subjects participating in the sleep deprivation protocol were woken by telephone at 6 am on day 1 of the study. They were instructed to remain awake for the day and to abstain from alcohol, nicotine and caffeinated drinks across the duration of the day. Apart from these restrictions, subjects were free to engage in their normal activities. By 11 pm on day 1, subjects reported to the university sleep laboratory. Overnight the subjects were free to engage in any activities they chose but were required to remain under observation in the laboratory to ensure no naps occurred.

From 11 pm on the first night of their attendance at the laboratory, subjects completed a visual analogue scale (VAS) assessing their subjective level of sleepiness across the experimental period. The scale comprised a 100 mm line with anchor points of 0 = lost struggle to remain awake and 100 = alert wide-awake. Subjects marked their subjective level of sleepiness every hour across the deprivation period.

At approximately 2 pm on day 2 of the study, subjects were tested on the performance tasks. The testing schedule took approximately 20 min and incorporated the PASAT and two filler tasks. Order of presentation of tasks was randomised.

Following the initial testing session, subjects were given the opportunity to sleep. The sleep period was monitored

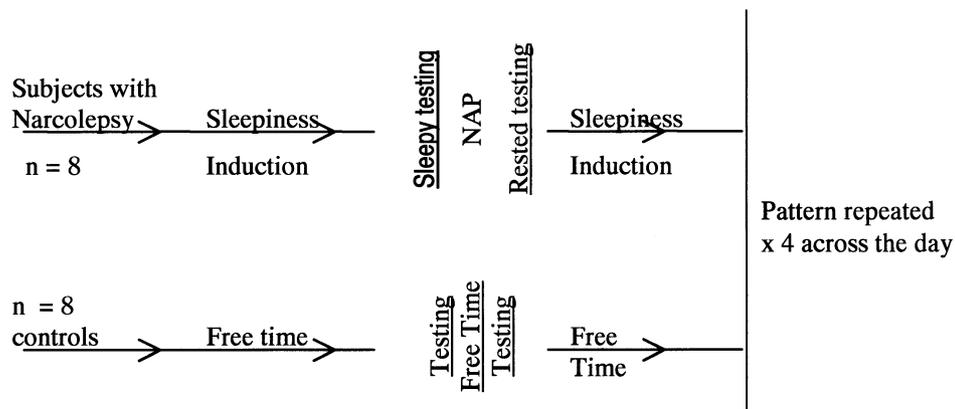
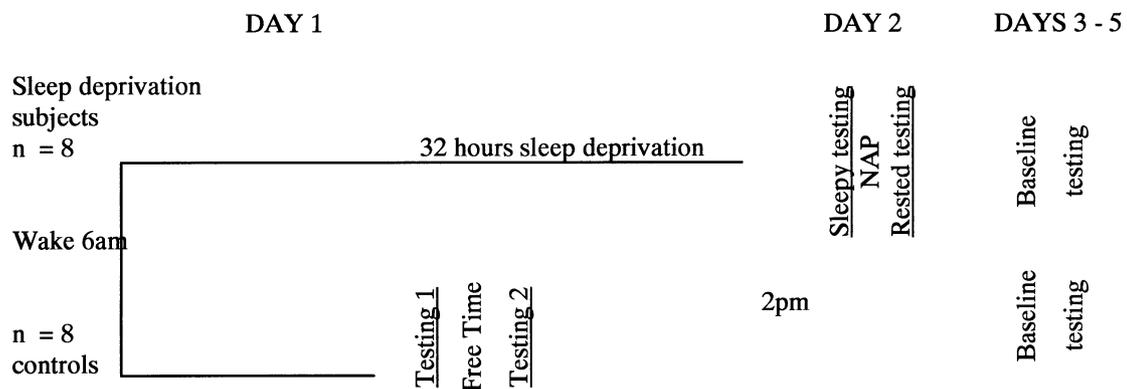
PROTOCOL STUDY 1**PROTOCOL STUDY 2**

Fig. 1. Summary of Study 1 and Study 2 protocols.

using the polysomnograph and subjects were awoken after 20 min of EEG defined sleep. All subjects were able to sleep during this period with time in bed ranging from approximately 25 to 45 min.

After being awoken from the nap, again in line with the protocol utilised for subjects with narcolepsy (Study 1), subjects were given a 5-min refractory period prior to the repeat testing session. Baseline performance measures for the sleep deprivation subjects were recorded at a third testing session scheduled between 3 and 5 days after the deprivation period.

As the sleep deprivation protocol required subjects to complete the set tasks three times, practice effects were estimated by parallel testing of control subjects. To minimise circadian confounds, the first test session for control subjects was undertaken at 2 pm and repeated 30 min later. The third testing session was completed, as for the sleep-deprived subjects, between 3 and 5 days later.

Fig. 1 provides a visual summary of the comparative protocols of Study 1 and Study 2.

5. Results*5.1. Comparative subject demographics between sleep-deprived subjects and subjects with narcolepsy*

Comparison of subject demographics between the sleep deprivation study, and the comparative study of subjects with narcolepsy, indicates that whilst no significant difference in IQ exists between normal sleepers ($M = 104$) and subjects with narcolepsy ($M = 102$; $t(7) = 0.58$, ns), for the variable of age, normal sleepers are significantly younger ($M = 30$ years) than the subjects with narcolepsy ($M = 53$ years; $t(7) = 3.71$, $P = 0.008$).

5.2. Comparative manipulation of arousal between sleep deprivation and narcolepsy protocols

Both sleep deprivation subjects and subjects with narcolepsy recorded subjective sleepiness ratings every hour in the laboratory using the VAS reported above. The rating for

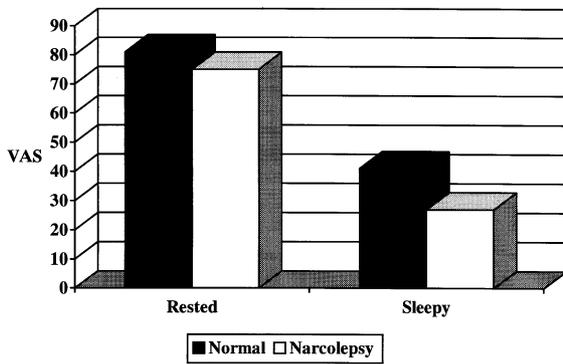


Fig. 2. Comparative changes in arousal ratings between normal sleepers and subjects with narcolepsy when rested and sleepy (lower rating indicates greater sleepiness).

Rested Normals represents the mean VAS rating taken at 11 pm on the first day of testing. The Sleepy Normals rating is represented by the mean VAS rating following 32 h of sleep deprivation. Following cognitive testing, Sleepy Normals were allowed a 20-min nap and the mean VAS rating following the nap session is identified as Post-nap. For subjects with narcolepsy, in the comparative study, the Sleep Narcolepsy rating represented the subjective VAS sleepiness rating preceding the nap period and immediately subsequent to the completion of the WAVT. The Rested Narcolepsy rating was recorded following the nap and a refractory period.

Fig. 2 provides a visual representation of comparative changes in manipulated arousal conditions across both study protocols.

Visual analysis of Fig. 2 suggests that both normal sleepers and subjects with narcolepsy demonstrated a decrease in arousal as a consequence of the experimental manipulation. The statistical significance of these arousal manipulations are summarised in Table 1.

Four major findings emerge from the statistical analyses

of the arousal manipulations:

- (i) Across the sleep deprivation period (Rested Normals vs Sleepy Normals), a significant decrease in subjective arousal ratings occurred.
- (ii) No significant difference exists between Study 1 and Study 2 sleepiness ratings between matched high (Rested Normals vs Rested Narcolepsy) and low (Sleepy Normals vs Sleepy Narcolepsy) sleepiness conditions.
- (iii) Comparative evaluation of the change in arousal conditions for sleep-deprived and narcolepsy subjects (Rested Normals to Sleepy Normals vs Rested Narcolepsy to Sleepy Narcolepsy) indicates no significant difference in induced sleepiness levels between subject groups.
- (iv) No significant difference exists in sleepiness ratings measured pre- and post-nap (Sleepy Normals vs Post-nap) for sleep-deprived subjects.

In summary, the sleep deprivation manipulation resulted in a significant decrease in subjective arousal ratings and this arousal decrement is statistically equivalent to the arousal decrement experienced by subjects with narcolepsy. For the sleep deprivation subjects, the 20-min nap led to no significant change in arousal measures. For subjects with narcolepsy, however, the equivalent nap period led to a significant increase in subjective arousal measures.

5.3. Impact of 32 h of sleep deprivation on complex cognitive performance

The above results demonstrate that for sleep-deprived subjects, the deprivation protocol resulted in significant decrements in subjective arousal. Of primary significance to this study is the comparative evaluation of the impact of this arousal manipulation on complex performance between sleep-deprived subjects and subjects with narcolepsy. The

Table 1
Analyses of comparative arousal manipulations both within the sleep deprivation protocol and between the sleep deprivation and narcolepsy protocols (Condition 1 vs Condition 2)

	Condition 1	Condition 2	t	df	P
Mean	Rested Normal	Sleepy Normal	Dependent		
SD	81.37	41.25	4.83	7	0.002
	16.92	27.84			
Mean	Rested Normal	Rested Narcolepsy	Independent		
SD	81.37	74.73	-0.94	32	0.352
	16.92	17.54			
Mean	Sleepy Normal	Sleepy Narcolepsy	Independent		
SD	41.25	27.34	-1.59	32	0.123
	27.84	19.62			
Mean	Sleepy Normal	Post-nap Normal	Dependent		
SD	41.25	54.12	-0.99	7	0.355
	27.84	30.09			
Mean	Rested Normal → Sleepy Normal	Rested Narcolepsy → Sleepy Narcolepsy	Independent		
SD	40.12	47.38	0.87	32	0.893
	23.49	19.92			

Table 2
Matrix summarising *t*-test results comparing PASAT performance scores across both sleepiness and subject groups

	Sleepy Normals	Post-nap Normals	Rested Normals	Sleepy Narcolepsy
Sleepy Normals				
Post-nap Normals	$T(7) = 0.03, P = 0.98$			
Rested Normals	$T(7) = 0.38, P = 0.71$	$T(6) = 0.18, P = 0.86$		
Sleepy Narcolepsy	$T(14) = 2.74, P = 0.016$	–	–	
Rested Narcolepsy	–	–	$T(13) = 0.22, P = 0.831$	$T(14) = 3.57, P = 0.009$

sleep deprivation protocol resulted in three performance measures for the PASAT – Rested, Sleepy, and Post-nap. As repeat testing of subjects potentially results in practice effects confounding performance outcomes, the current study utilised the scores associated with repeat testing of control subjects to estimate the degree of practice between each testing session. This percentage change associated with repeat control testing was then subtracted from the performance score to provide a residual performance measure presumed to reflect changes subsequent to the arousal manipulation. This protocol, derived from the work of May and Kline [14] was also used to separate sleepiness and practice effects in the comparative study on subjects with narcolepsy. Table 2 provides an analysis of the significance of the difference in complex performance for sleep-deprived subjects between the various testing conditions (sleepy, rested and post-nap). Comparative scores from the narcolepsy study are also included.

Table 2 demonstrates that the Sleepy Normals did not have any decrement in performance compared to Rested Normals scores. Similarly, no significant change in performance was noted between the Sleepy Normals (i.e. before a nap) and Post-nap testing conditions. (This analysis of the impact of napping on performance for sleep-deprived subjects is included for completeness but, theoretically, no change in performance is predicted, as the pre-/post-nap manipulation was not associated with significant change in arousal ratings for the sleep deprivation group – refer Table 1.) This lack of effect on complex performance, for sleepiness induced through sleep deprivation, contrasts with the significant performance decrements associated with equivalent levels of sleepiness for subjects with narcolepsy.

6. Discussion

The sleep deprivation study reported in this paper represents the first attempt to equate the sleepiness associated with narcolepsy to sleepiness experienced as a consequence of sleep deprivation for non-pathological sleepers. For both sleep-deprived subjects and subjects with narcolepsy, sleepiness was quantified using a subjective VAS rating scale. There has been some suggestion that the use of subjective sleepiness scales may have diminished validity in pathologically sleepy subjects as they may lose an appropriate frame of reference by which to measure sleepiness [15].

This was not, however, seen to be a limitation of the current study as this work evaluated the impact of the manipulation of sleepiness on subjective ratings of ‘change in sleepiness’ rather than absolute sleepiness measures. Perceptions of change are assumed to remain valid within the subject’s own frame of reference.

Several interesting findings emerge from the comparative analyses described in this paper.

6.1. The induction and discharge of sleepiness

Study 1 induced sleepiness in subjects with narcolepsy by withdrawing subjects from stimulant medication and then exposing them to the sleep inducing WAVT task. The findings from Study 2 suggest that the intensity of this sleepiness is statistically equated with 32 h of sleep deprivation for normal sleepers. Study 1 was further able to continuously induce this level of sleepiness for subjects with narcolepsy using a 1-h manipulation across repeated testing sessions. Volk et al. [6] have previously shown the sensitivity of arousal states in narcolepsy to environmental influences. Their work demonstrated that confining subjects with narcolepsy to bed increases the amount of daytime sleep by a factor of two to three times that of subjects with narcolepsy who sit at a table across the day. Besset et al. [16] reiterate this sensitivity of narcoleptic sleepiness to environmental factors suggesting “narcoleptic subjects appear more sensitive to environmental conditions than normal sleepers” (p. S32). The comparative analysis reported in this paper highlights the intensity of this environmentally induced arousal fluctuation for subjects with narcolepsy.

Not only do subjects with narcolepsy appear highly sensitive to the induction of sleepiness, but as previously reported [7,17,18], the brief nap period appears to discharge this sleepiness, re-establishing a state of high arousal. In contrast, for sleep-deprived subjects, the 20-min nap period was not associated with any significant change in arousal conditions. As subjective levels of sleepiness measured pre-nap were statistically equated in subjects with narcolepsy and sleep-deprived subjects, this lack of recuperation of arousal subsequent to the nap period for sleep-deprived subjects contrasts significantly with the subjective recuperative nature of naps for subjects with narcolepsy. One potential explanation for the observed lack of increment in arousal following the nap period for sleep-deprived subjects is the potential for residual sleep inertia effects to counteract

arousal changes. Whilst the study protocol allowed a 5-min refractory period, this may not have been sufficient for the dissipation of sleep inertia. Estimates of the duration of sleep inertia appear contentious though it seems that arousal from SWS significantly increases inertia effects [19]. As naps for subjects with narcolepsy appear to be consistently refreshing it is reasonable to suggest that sleep inertia effects may operate differentially in the disengagement of sleepiness between conditions of pathological and non-pathological sleepiness.

6.2. *The interaction of sleepiness and performance*

A further area of interest in the current study was the implication of these arousal changes on performance in the two sleepy subject groups. Significant differences in performance outcomes were found. For sleep-deprived subjects, the decreased arousal did not result in any performance decrement on the complex PASAT task. Whilst periods of sleep deprivation have been consistently associated with performance decrements [6], the literature also supports the finding that attentional resources can overcome sleepiness effects on performance. Research in the 1960s by Wilkinson [20] demonstrated that complex and exciting performance tasks are resistant to performance decrements even after 60 h of sleep deprivation. More recently, Horne and Pettitt [21] demonstrated that motivational factors are able to offset sleepiness effects across a 36 h deprivation period. Subjects in the current sleep deprivation study may well have found participation in the university-based experimental protocol intrinsically interesting and therefore have been able to counteract the effects of sleepiness on performance using this motivational factor. Subjects with narcolepsy at apparently equivalent sleepiness levels appeared to not be able to utilise attentional resources to counteract the performance decrements.

An alternate explanation for these observed differences in the effects of sleepiness on complex performance between sleep deprivation and narcolepsy, is that whilst subjective low arousal conditions were approximately equated between subjects, subjects with narcolepsy may have underestimated their level of sleepiness. Consequently, the performance decrement observed in narcolepsy may reflect this increased, yet unreported, level of sleepiness. The literature on excessive daytime sleepiness supports this possibility, arguing that sufferers of excessive daytime sleepiness typically underestimate the severity of their sleepiness [15,22]. It seems, however, that even for non-pathologically sleepy subjects, measurement strategies for assessment of daytime sleepiness have limited convergent validity. For example, Multiple Sleep Latency Test measures predict differing levels of sleepiness to either measures focusing on the maintenance of wakefulness, or subjective reporting of sleepiness states [23]. One explanation for this lack of association between sleepiness measures in non-pathologically sleepy populations is that sleepiness,

as well as varying along quantitative dimensions, may vary across as yet undefined qualitative dimensions. The lack of convergence of measures of sleepiness may simply reflect that different instruments are tapping different components of this multidimensional construct. It is therefore reasonable to argue that similarly, for subjects with narcolepsy, subjective assessments of sleepiness are not invalid, but are also linked to qualitatively distinct aspects of sleepiness to those measured by tools tapping physiological aspects of sleepiness in narcolepsy. A possible extension of this argument is that, if a lack of subjective convergence exists, between sleepiness in narcolepsy and sleepiness secondary to sleep deprivation then this may also be explained by intrinsic qualitative differences between these sleepiness states. Previous research has highlighted the potential for qualitative differences to exist between sleepiness in narcolepsy and sleepiness induced in non-pathological sleepers. For example, in contrast to normal sleepiness, sleepiness in narcolepsy is characterised by (i) the recuperative power of very brief sleep episodes or sleep attacks [17]; (ii) the ambiguity around the association of nocturnal and daytime sleep parameters [24]; (iii) the disturbance, for subjects with narcolepsy, of endogenous sleep wake timing mechanisms, leading to unstable neural states, across both sleep and wakefulness states [25–27] and (iv) subjective reports of the sudden and overwhelming nature of the sleepiness [28].

The current study represents the first attempt in the research literature to compare both arousal and performance measures between sleep-deprived subjects and subjects with narcolepsy. Several methodological limitations need, however, to be acknowledged in the interpretation of the current findings. Despite attempts to ‘match’ subjects with narcolepsy and sleep-deprived subjects, significant differences remained in age between these subject groups with the sleep deprivation study incorporating a significantly younger subject cohort than the narcolepsy group. This age difference may have impacted on both subjective sleepiness measures and performance outcomes. Performance on the PASAT has been demonstrated to decrease with age [13] though in the current study no significant difference was noted for PASAT scores between the older subject cohort in Study 1 and the younger subject group of Study 2 under rested conditions. Decreased scores on the PASAT for the older cohort of subjects with narcolepsy, under sleepy conditions, compared to the younger sleep-deprived cohort, may, however, result in part from age-related interactions between performance, sleepiness and age rather than reflect cognitive changes related to the physiological processes associated with pathological sleepiness. Replication of the current study across matched age groups is necessary to eliminate this potential study confound. A second potential confound of the current study is that subjects with narcolepsy were withdrawn from stimulant medication for the purposes of testing. This process of withdrawal may have impacted on objective sleepiness measures, subjective assessments of sleepiness and/or performance task

outcomes. Finally it is important to recognise that the disparate methodologies used to induce sleepiness i.e. an extended period of sleep deprivation for non-pathological sleepers in comparison to the repeated daytime use of the WAVT for subjects with narcolepsy may have resulted in the artificial generation of qualitatively different sleepiness states potentially confounding study outcomes.

Despite these limitations, the findings provide initial support for the suggestion that both the onset and discharge of sleepiness in narcolepsy is significantly different to that experienced in non-pathological sleepiness. The current study also extends the literature in the field by demonstrating that sleepiness subsequent to pathological and non-pathological mechanisms may have different functional outcomes in relation to performance on complex processing tasks.

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