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BRIEF COMMUNICATION

Can Normal Subjects Be Motivated to Fall Asleep Faster?

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HARRISON, Y., V. BRIGHT AND J. A. HORNE. *Can normal subjects be motivated to fall asleep faster?* PHYSIOL BEHAV **60**(2) 681–684, 1996.—The Multiple Sleep Latency Test (MSLT) is widely believed to offer an objective, physiological measure of sleepiness. The speed with which a person falls asleep throughout the day is understood to be related systematically to sleep need and circadian phase. This study examined whether normal subjects ($n = 14$ young female adults) could achieve faster MSLT sleep onsets if they were given the incentive to do so. During week 1 baseline MSLTs were determined over 1 day for all subjects. In week 2 they were randomly assigned to two groups. Control subjects underwent a second MSLT testing day identical to that of week 1, whereas Incentive subjects had an additional financial incentive to sleep. There was a significant reduction in sleep onset latency (indicating increased sleepiness) during the 1500 h trial following the incentive, when subjects also reported a significantly greater increase in sleepiness over the trial. These findings suggest that when coupled with a mid-afternoon increase in sleepiness, increased motivation to sleep can reduce sleep onset latency.

Daytime sleepiness MSLT Sleep onset

IT IS widely believed that one measure of a physiological need for sleep is the speed at which sleep occurs (5,6,11,13). This assumption underpins the Multiple Sleep Latency Test (MSLT) in which the latency to sleep onset varies with respect to sleep need and circadian phase (5). Only those factors likely to prohibit the onset of sleep are identified as possible sources of contamination (12), and it has been argued that the role of subject motivation during the MSLT is minimal (13).

Dinges (8) noted that not at all measures of sleepiness give the same result, or are equally sensitive to experimental changes in sleep. Two possible explanations for this were considered: the first assumes sleepiness to be a single physiological state for which not all measures are equally sensitive. In this respect the MSLT has been promoted as unique in directly accessing this single physiological state of sleepiness (5,6,13). The second possibility, and the one favoured by Dinges (8), is that differences between measures reflect the need to consider the specific circumstances in which sleepiness or sleep occurs. This was recently illustrated by Kribbs et al. (10), where significantly shortened sleep latencies were found for latency tests following 20-min periods of vigilance testing.

Motivating sleepy subjects to stay awake seems a relatively easy undertaking, as demonstrated (2) in a variant of the MSLT, where it was found that sleep-deprived subjects offered a financial incentive to stay awake were able to do this for significantly longer than subjects not motivated in this way. If resisting sleep is at least partially under volitional control, then the issue of whether subjects can shorten the onset of sleep through volitional processes is also of importance.

There have been various attempts to enhance the onset of sleep in humans and animals. Wilcox (14) explored the possibility of establishing sleep onset as a conditioned response in rats by offering a reward for each successful attempt to initiate sleep. This resulted in more sleep preparatory type behaviours (i.e., quiet immobility with reduced muscle tone) but no actual overall increase in the number of sleep onsets. Caruso et al. (7) had more success in human subjects when they classically conditioned a 25% reduction in sleep latency.

We examined the role of subjective effort in facilitating the onset of sleep by offering subjects a financial incentive to go to sleep during the MSLT. It was expected that this would maximise any volitional component of the sleep onset process.

METHOD

Subjects, 14 females (age 18–28 years), responded to an advertising poster displayed around the University. They slept

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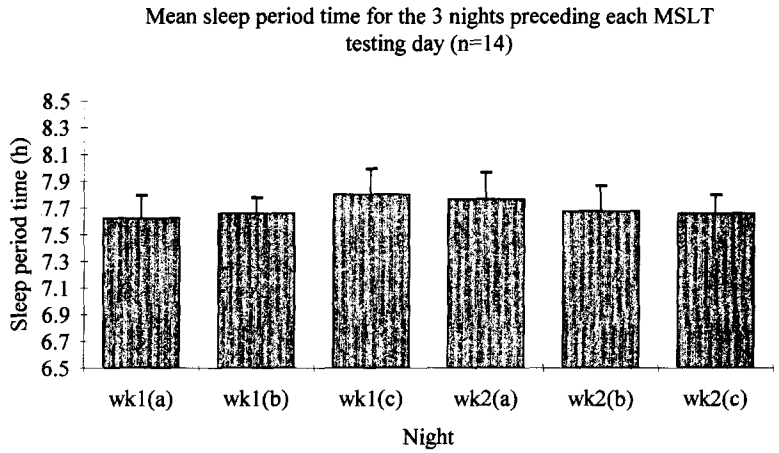

FIG. 1. Mean (SE) sleep period time for the 3 nights preceding each MSLT testing day ($n = 14$).

regularly, between 7–8 h/night, did not sleep during the day, and had no sleep complaints. Actimeters were worn each night for 10 nights, commencing 3 nights before the initial MSLT day, to ensure a regular sleep schedule and 7–8 h of sleep per night. Actimeters were worn on the dominant wrist 10 min before lights out until the final awakening the following morning. Sleep onset was determined by software previously validated against EEG recordings (9). Subjects also completed sleep diaries, including details of lights out and wake time. Sleep period time was calculated as the period between sleep onset and the final awakening. Subjects underwent 2 testing days consisting of three MSLTs performed at 2-h intervals commencing 1100 h. These 2 days were 1 week apart, during which time subjects slept at home.

For week 1, subjects were allocated to a testing day on an ad hoc basis. The MSLT protocol was as follows: a single channel of EEG (C3–A2) and two channels of EOG were recorded according to the guidelines for the administration of the MSLT (6). Subjects were asked to lie on a bed in a quiet, darkened room for a period of up to 20 min. They were instructed to: "Lie down, and with your eyes closed try to go to sleep." EEGs were printed on-line with a Grass Polygraph machine, with paper speeds set at 1 cm/s with gain equivalent to 25 V/cm. Trials were terminated following three consecutive 30-s epochs of stage 1 sleep, or a single 30-s epoch of another sleep stage. Failing this, trials were terminated following 20 min after lights out. Between trials, subjects engaged in light study, avoiding heavy meals, caffeinated drinks, and vigorous exercise.

At the first trial of the second testing day (week 2) subjects were randomly allocated to one of two groups: Control and Incentive. For both groups MSLTs were repeated using the same protocol, except that the Incentive group were further instructed before each trial, "If you can sleep faster than you did this time last week then you will be given an extra $2.00." All subjects had initially agreed to participate for a basic payment of $12.00. Subjects in the Incentive group therefore had the opportunity to increase their overall financial gain by 50%. The basic payment (minus incentive) was typical for student experimental participation at this University. As subjects participated without knowledge of the incentive throughout the first week of testing it was assumed that they were satisfied with their original expectations of payment. It was decided to offer a relative increase of a maximum of 50% incentive payment as it provided a reasonable increase to original expectations without being so excessive as to heighten anxiety during trials.

Immediately before and after each MSLT trial subjects completed the Karolinska Sleepiness Scale (KSS-1). This nine-point scale has sleepiness-related descriptors (ranging through Extremely Alert, Alert, Neither Alert Nor Sleepy, Sleepy—But No Difficulty Remaining Awake, Extremely Sleepy—Fighting Sleep).

RESULTS

Nighttime Sleep: Actimeter Data

Sleep period time was calculated as the time in minutes from sleep onset until final awakening. Figure 1 shows mean sleep period times for the 3 nights preceding each of the two MSLT testing days. Subjects had been screened prior to participation to ensure a regular 7–8 h habitual sleep schedule, with no daytime napping, and this was maintained throughout the study. Figure 1 shows that sleep period time was held relatively constant between 7.5 and 8 h each night. Sleep period time was also comparable from week to week. The mean of the 3 nights was calculated for each subject for week 1 and week 2. Mean sleep period times for week 1 and week 2 were found to be highly correlated (Pearson product moment: $r = 0.86$).

MSLT Scores

For each MSLT session a sleep latency score was determined as the time to the first of three consecutive 30-s epochs of stage 1 sleep or 30 s of another sleep stage, according to the guidelines for scoring the experimental version of the MSLT (6). The

TABLE 1
MEAN MSLT SCORES (WITH STANDARD ERRORS) ACROSS TRIALS FOR EACH SUBJECT GROUP

	Control		Incentive	
	Week 1	Week 2	Week 1	Week 2
1100 h	12.6 (2.6)	12.8 (2.7)	15.7 (2.0)	15.6 (1.7)
1300 h	12.8 (2.4)	15.7 (2.0)	14.6 (2.3)	16.0 (1.4)
1500 h	12.3 (2.7)	12.8 (2.3)	13.6 (1.8)	*8.3 (1.7)
Daily mean	12.6	13.8	14.6	13.3

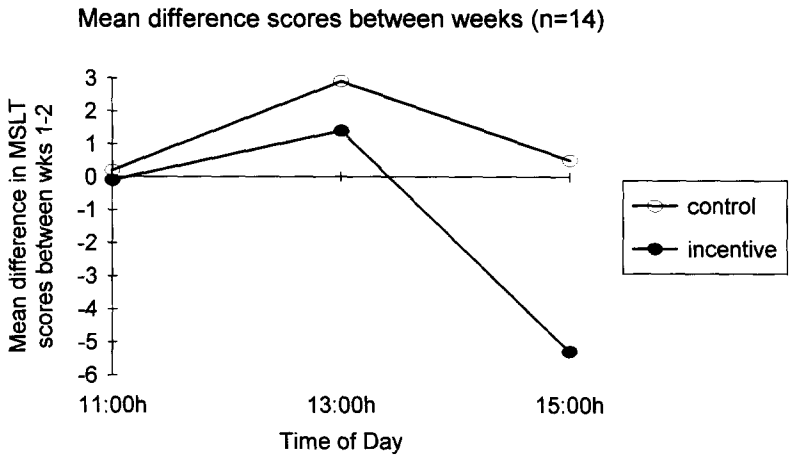

FIG. 2. Mean difference in MSLT scores (SE) between week 1 and week 2 for each time of day for Control and Incentive groups. At 1500 h there was a significant reduction in sleep latency scores for the Incentive group.

accumulation of sleep as the day progressed and the potentially distorting effect of this on subsequent sessions was avoided by terminating the test at this point. Failure to satisfy these requirements was recorded as a score of 20 min and the test terminated. Daily mean MSLT scores were calculated for each group (see Table 1).

A multivariate analysis of variance (MANOVA) was calculated for: a within-subject factor of Week (two levels), a within-subject factor of time of day (ToD, three levels), and a between-subject factor of Incentive (two levels). Prior to the MANOVA, scores were log transformed. Analysis of the main effects revealed: i) a significant main effect of ToD, $F(2, 24) = 3.99$, $p = 0.03$; ii) a nonsignificant effect of Week; iii) a nonsignificant effect of Incentive.

Analysis of the interactions revealed: i) a significant interaction between ToD and Incentive, $F(2, 24) = 3.43$, $p = 0.05$; ii) a near significant interaction between Incentive and Week, $F(1, 12) = 3.92$, $p = 0.07$; iii) a near significant interaction between Week and ToD, $F(2, 24) = 3.09$, $p = 0.06$; iv) a nonsignificant interaction between Incentive and Week and ToD.

The significant effect of ToD might be expected because of diurnal changes in sleepiness levels regardless of instruction. Paired t-tests explored the main ToD effects. Significant differences were found between 1100 and 1500 h, $t(13) = 2.20$, $p = 0.04$, and between 1300 and 1500 h, $t(13) = 2.71$, $p = 0.01$.

The relative stability of latency scores across weeks for the Control group (Table 1) contributes towards the nonsignificant effect of Week. As the Incentive was confined to the second week then the main effect of Incentive regardless of week might also be expected to be nonsignificant. On the other hand, as the experimental intervention was varied across weeks, our hypothesis would have predicted some effect of Week but this was revealed as only a near-significant interaction for both the Week by Incentive and Week by ToD interactions.

The mean differences in MSLT scores between week 1 and week 2 are shown in Fig. 2 for both subject groups for each time

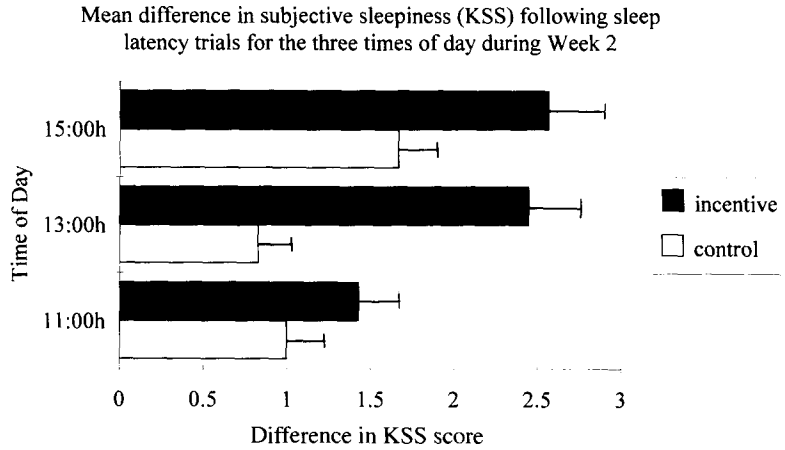

FIG. 3. Mean changes in the difference between pre- and post-MSLT subjective sleepiness scores on the Karolinska Sleepiness Scale (KSS). For both groups sleepiness increased during the trial. But the Incentive group showed a significantly greater change (see text).

of day.

Post hoc paired t-tests were used to explore changes within groups from week 1 to week 2 for each ToD. A significant difference between scores, $t(6) = 3.03$, $p = 0.02$ (two-tailed), was found between 1500 h trials for the Incentive group. All other latency score differences were found to be nonsignificiant. As differences were confined to a single trial this might account for why the influence of Week in the Week by ToD and Week by Incentive interactions was only found to reach near significance.

Subjective Sleepiness

Subjective ratings were transformed by subtracting the pretrial score from the posttrial score. A positive difference score indicated that subjective sleepiness had increased throughout the duration of the trial—the higher the score the greater the increase in sleepiness. Figure 3 gives these data for the second week, where it can be seen that the incentive group experienced an increased relative change in sleepiness over each of the three ToD sessions. Here, the overall mean change from pre- to postsession across the three times of day was 1.16 for the control group (on the nine-point KSS) compared with 2.14 for the incentive group. For each subject the mean daily change in sleepiness score was calculated across the three trials for both week 1 and week 2. A Mann–Whitney U-test showed the difference between degree of change for daily means to be significant for incentive and control groups during week 2 ($U = 9.5$, $p < 0.05$).

DISCUSSION

For the three daily MSLT trials a relatively high consistency between scores on repeated testing days throughout a period of regular nighttime sleep would be expected for the women involved in this study (15). However, the present finding of a reduction in sleep latency for the Incentive group at 1500 h suggests that when coupled with a sufficiently high propensity for sleep, sleep onset during the MSLT may be liable to interference from motivational influences. On the other hand, the relative influence of a mid-afternoon increase in sleepiness may have been misleading, as it was preceded by two opportunities to modify a response to the incentive instructions.

This highlights a potential difficulty with using the MSLT to assess daytime sleepiness in normal, healthy young adults. Whereas a relationship between nighttime sleep duration and subsequent daytime sleepiness has been demonstrated following sleep reduction and sleep extension (3,4,6), the sensitivity of the MSLT in subjects experiencing mild levels of sleepiness and the influence of factors other than a physiological need for sleep remain questionable.

A discussion of the precise nature of the processes involved in facilitating sleep for our Incentive subjects is beyond the scope of our report, for example, whether or not our findings reflect increased effort to fall asleep, or reduced effort to maintain wakefulness. In any event, the assumption that motivational factors act only to inhibit sleep during the MSLT (12) is undermined.

It is also of interest that the Incentive subjects perceived a greater change in sleepiness following the MSLT trial than control subjects. This is despite the fact that Incentive subjects spent less actual time in bed (due to the test being terminated at an earlier sleep onset). If this was due to subjects receiving an alternative instruction, then it would again seem that subjective sleepiness is prone to external factors (i.e., by the suggestion made by the experimenter that they would be more likely to have experienced sleepiness). Alternatively, this increase in sleepiness for the Incentive group might reflect actual success in their attempts to reduce their arousal levels and fall asleep. On debriefing it was generally remarked that subjects perceived the level of incentive to be worthwhile, without being aware of increased anxiety due to the additional emphasis on falling asleep.

A mean MSLT score at baseline of around 13–15 min was found for both groups. This represents a fairly typical level of sleepiness for young, regular sleeping normal subjects and is indicative of only mild sleepiness throughout the day. Nevertheless, with the added incentive of extra money, subjects in the experimental group were able to reduce significantly the interval between lights out and sleep onset during latency trials from baseline levels. Whereas Carskadon and Dement (5) have argued that only physiological sleepiness will lead to sleep, through increasing the propensity for sleep we have shown in this study that, for some individuals, the mechanisms for achieving sleep may also be influenced by psychological factors such as motivation.

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