ORIGINAL ARTICLE

Sleep Science

Sleep extension reduces fatigue in healthy, normally-sleeping young adults

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ABSTRACT

Objective: To assess the effects of one week of sleep extension on mood, fatigue and subjective sleepiness in normal-sleeping young adults. **Methods:** Twenty-seven adults (age 24.4±5.4 years, 11 female) participated. At-home baseline sleep/wake patterns were recorded with wrist actigraphy for 14 days. This was followed by two nights of in-lab baseline sleep with 8 hours time in bed (TIB), then 7 nights with TIB extended to 10 hours (2100-0700 hours). Fatigue, mood, and sleepiness were assessed following the 2nd and 9th nights of in-laboratory sleep (i.e., 2 nights with 8hTIB and 7 nights with 10 hours TIB, respectively) using the Automated Neuropsychological Assessment Metric and Karolinska Sleepiness Scale. Paired t-tests were used to compare mood, fatigue, and sleepiness ratings between conditions. **Results:** At-home wrist actigraphy revealed a mean nightly total sleep time (TST) of 7.53 +/- 0.88 hours of sleep per night. Mean in-lab baseline sleep duration (7.76 +/- 0.59) did not differ from at-home sleep. However, during sleep extension, mean TST was 9.36 +/- 0.37 hours per night, significantly more than during the in-lab baseline ($p < .001$). Following sleep extension, fatigue ratings were significantly reduced, relative to baseline ($p = .03$). However, sleep extension had no other significant effects on subjective ratings of mood or sleepiness. **Conclusions:** Sleep extension resulted in reduced fatigue in healthy, normal-sleeping young adults, although subjective sleepiness and mood were not improved. Implications include the possibility that (a) the effects of sleep extension on various aspects of mood depend upon the extent to which those aspects of mood are made salient by the study design and methodology; and (b) sleep extension may prove beneficial to fatigue-related conditions such as "burnout."

Keywords: Sleep; Fatigue; Adult.

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INTRODUCTION

The relationship between nighttime sleep and daytime functioning is straightforward. The less sleep that is obtained at night, the greater the next-day decrements in alertness and performance, and the greater the amount of subsequent 'recovery sleep' needed to restore alertness and performance to normal, baseline levels. However, the ratio of "sleep lost" to "sleep recovered" is not 1:1. If an individual who averages 8 hours of sleep per night is totally deprived of sleep for one night, he/she will not subsequently require 16 hours of sleep to achieve full recovery - a phenomenon that suggests that either (a) recovery sleep is more efficient (has greater minute-by-minute recuperative value) than typical nighttime sleep, and/or (b) the rate at which sleep-furnished resources are utilized during wakefulness is reduced by prior sleep loss[1].

Although such recovery sleep following sleep loss is characterized by supra-normal sleep duration, for the purposes of the present paper, it is considered conceptually distinct from 'sleep extension,' with the primary difference being that it is sleep extension that occurs *in response to* prior sleep loss. In contrast, 'sleep extension' is the volitional lengthening of nighttime sleep duration in the absence of greater-than-normal homeostatic sleep pressure. Of course, this distinction may be more quantitative than qualitative, with these phenomena representing two points on a single continuum that differ only in terms of the level of homeostatic sleep pressure. In other words, although sleep extension occurs in the absence of greater-than-normal homeostatic sleep pressure, that level of sleep pressure cannot be zero. Indeed, because volitional control of sleep onset and maintenance is limited, sleep would not even be initiated, much less extended, if the homeostatic pressure to sleep was at or near zero.

Thus, recovery sleep is conceptualized as a post-sleep loss, non-volitional process (involving both increased sleep duration and possibly an increased rate of recuperation during sleep) by which alertness and performance are restored to the normal, albeit possibly submaximal, level. In contrast, sleep extension is conceptualized as a process by which volitional extension of sleep duration produces a level of recuperation that is greater than that habitually obtained, and that is closer to maximal.

To date, the scientific literature on sleep extension, as presently defined and conceptualized here, is fairly limited. In part, this is because much of the early work on sleep extension failed to reveal any measurable (much less beneficial) effects. Indeed, in those early studies, the effects of increasing nighttime sleep duration beyond normal were found to be so paltry that it was hypothesized that there exists a 'sleep duration threshold', beyond which sleep fails to have any recuperative value[2,3].

Likewise, beneficial effects of sleep extension have recently been reported in studies with more naturalistic study designs. For example, in one study it was found that extending sleep from ~7 hours to ~9 hours for one week resulted in improved performance in college tennis players - i.e., improved accuracy when serving (although the lack of 'non-sleep extension control' condition requires that this finding be viewed with some skepticism). Prior research suggests beneficial effects of sleep on mood. In one study in which normal sleepers took a 20-minute mid-day nap, participants reported that the nap had generally positive effects on their mood relative to 20 minutes of non-sleep rest[4]. In another, similar, study it was found that individuals who napped for 30 minutes had improved mood compared to individuals who merely had a 30-minute cognitive break[5]. These findings suggest that sleep generally has salutary effects on mood, but other findings suggest that the relationship between sleep and mood is somewhat complex. Paradoxically, in certain populations (e.g., a subset of individuals with affective disorders) sleep *loss* reduces mood. Furthermore, although several studies note a positive change in mood following recovery from sleep deprivation[6,7], many have failed to find an effect[8-10].

Fatigue, a subjective state akin to "weariness", is of particular interest in the present study. Fatigue, which is itself considered an aspect of mood[11], is a complex cognitive process that is thought to result from cognitive overuse or overburden[12]. Spending a long period of time doing the same activity can create a state of subjective fatigue that is characterized by an increasing disinclination to continue the activity. Additionally, fatigue is exacerbated by sleep loss: sleep-deprived and restricted individuals report higher levels of fatigue in addition to higher levels of sleepiness[13-15]. Recovery sleep after sleep restriction minimizes such fatigue[16]. Sleep extension may therefore effectively reduce subjective fatigue.

Consistent with what is discussed above, an early study did show beneficial effects of sleep extension on fatigue but not other aspects of mood[17]. In that study, participants slept ad-libitum until their objective sleepiness (measured via Multiple Sleep Latency Test[18]) was low. It was found that fatigue decreased and vigor increased as a result of sleep extension, but no other aspects of mood were affected. Interestingly, the authors of the study did not report on subjective sleepiness in the context of sleep extension. It is not clear whether sleep extension would benefit both fatigue and subjective sleepiness in healthy normal-sleeping individuals. In the current study, we sought to assess the impact of brief period of sleep extension (7 days) on mood, fatigue and subjective sleepiness in a healthy, normal-sleeping population.

METHODS

This study was approved by the Walter Reed Army Institute of Research Human Use Review Committee and the United States Army Medical Research and Materiel Command Human Subjects Research Review Board and was performed in accordance with the ethical standards of the 1964 Declaration of Helsinki.

Participant Inclusion/Exclusion Criteria

Men and women 18 to 39 years of age were recruited via flyers posted at local colleges, universities, and military installations. After providing informed consent, volunteers completed questionnaires to determine eligibility based on physical and psychological health, and sleep habits. Volunteers then

underwent a physical examination, and an evaluation of blood and urine samples was conducted to determine general health status (including pregnancy and drug use status). Lastly, participants completed an in-lab screening night to rule out sleep disorders. During the screening night, participants slept overnight (minimum 8 hours TIB: 2300-0700) while wearing the level 3 equipment, including a nasal cannula, a pulse oximetry probe worn on the index finger, a thorax and abdomen effort belt, and a snore and position monitor. Individuals with an apnea-hypopnea index greater than 5 were excluded from study participation.

Volunteers were excluded if they reported any of the following for the preceding month: (1) habitual nightly sleep amounts outside the range of 6-9 h on weeknights, (2) average morning wake-up times later than 09:00 Monday through Friday, (3) average nighttime lights-out times earlier than 09:00 Sunday through Thursday (to exclude those with extreme "social jetlag"[19]), or (4) average habitual napping of greater than 3 times a week. Additional study exclusionary criteria included: cardiovascular disease; hypertension; past or present neurologic, or sleep disorder; psychiatric disorder within the last three years, present or past use of over-the-counter substances with purported psychoactive properties; use of sleep-aids within the last 2 years; regular nicotine use within the last year; current heavy alcohol use (> 14 drinks per week); current use of other drugs (including but not limited to benzodiazepines, amphetamines, cocaine, and marijuana); medication use during in-laboratory challenge phases (including use of vitamins or supplements; not including oral contraceptives); liver and kidney disease or abnormalities; underlying pulmonary disease requiring daily inhaler use; clinically significant values for any screening hematology or chemistry parameter, BMI ≥ 30**;** self-reported history of caffeine use > 400 mg (8 caffeinated sodas or 3-4 cups of coffee) per day on average; score ≥ 14 on the Beck Depression Inventory[20]; score ≥41 on the Speilberger State-Trait Anxiety Inventory[21], extreme scores < 31 ("definite evening") or > 61 ("definite morning") on the Horne-Ostberg morningness-eveningness questionnaire[22], or current breast feeding or pregnancy. Additionally, subjects who screened positive for nicotine, alcohol or illicit drugs at the screening visit were excluded from participation. Subjects who were unable to read and sign the informed consent document were excluded as well.

Experimental Design

Figure 1 depicts the experimental design. This study was part of a larger experiment aimed at testing the impact of sleep extension and subsequent sleep deprivation on inflammatory markers[23]. The study consisted of 1 at-home baseline phase with wrist actigraphy followed by 3 consecutive in-laboratory within-subject phases. During the first phase, subjects wore wrist actigraphs continuously and were instructed to maintain their typical sleep/wake schedules. During the second phase, participants were provided a baseline sleep opportunity (2300 to 0700, 8 hours time in bed [TIB]). Subsequently, participants underwent 7 nights of sleep extension (2100 to 0700, 10 hours TIB) followed by 1 night of total sleep deprivation (36 hours of continuous wakefulness) and a 10-hour recovery sleep period. Participants were allowed to leave the lab and continue typical day-to-day activities during the sleep extension phase but were asked to refrain from strenuous physical activity while in the lab. Actigraphy monitoring continued throughout the duration of the study. Only data from the at-home, baseline, and extension phases are presented here.

Cognitive/Sleep Measures

Automated Neuropsychological Assessment Metric

The Automated Neuropsychological Assessment Metric Version 4 (ANAM) is a computerized test battery that includes a variety of state and trait neurocognitive measures, including but not limited to, attention, learning, problem solving, subjective sleepiness and mood[24]. Within the ANAM, 42 mood items were presented to participants with the message "How much does this word describe how you feel?" Participants chose a number between 0 (not at all) and 6 (very much). Mood items fell into one of 7 constructs (Anger, Anxiety, Depression, Cognitive/Mental Fatigue, Happiness, Restlessness, Vigor), with 6 items in each category. The Cronbach's alpha ranges from .80 to .93 depending on the construct (from restlessness to depression, respectively). Test-retest reliability ranged from .75 to .93 (from restlessness to depression, respectively). Lastly, the battery is valid and correlated strongly with several other relevant scales (r-values ranging from .59-.70). There are no cut-off values in this scale. The ANAM was administered every 4 hours (at 1100, 1500, and 1900) during the last day of each phase.

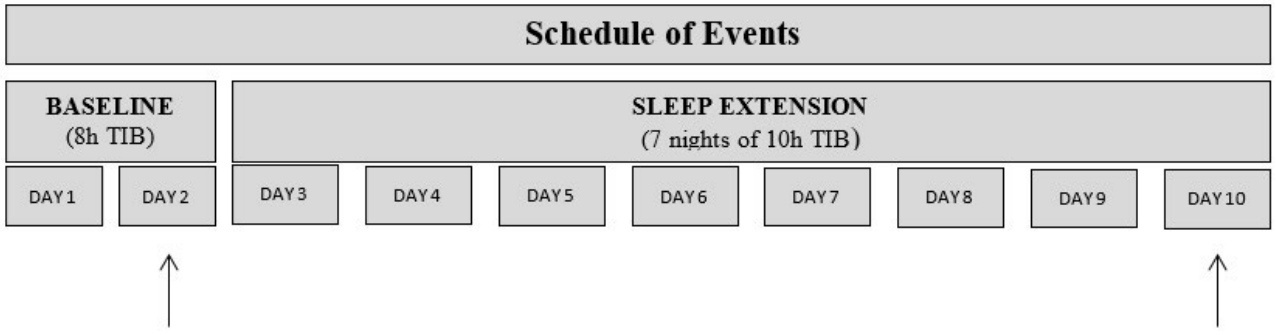

Figure 1. Study design.

Karolinska Sleepiness Scale

The Karolinska Sleepiness Scale (KSS) is a validated 9-point scale (1=extremely alert, 3 = alert, 5 = neither alert nor sleepy, 7=sleepy - but no difficulty remaining awake, and 9=extremely sleepy - fighting sleep) used for assessment of subjective sleepiness[25]. A computerized version of the KSS was administered and ratings from the three daily time points were averaged. This scale was administered every 4 hours (at 1100, 1500, and 1900) during the last day of each phase.

Actigraphy

For the entire duration of the study, participants wore an Actiwatch 2 (Philips Respironics, Murrysville, PA) wrist actigraph on their non-dominant wrist Sleep-wake status for each 30-second epoch was computed using the Actiware 6.0.9 scoring algorithm. These actigraphs have been shown to be a valid, reliable method of measuring sleep duration (statistically similar to polysomnography[26]).

Statistical Analyses

SPSS 23 (Armonk, NY) was used for statistical analyses. Descriptive statistics (mean ± standard deviation) were used to quantify demographic measures. To compare sleep parameters (e.g., total sleep time) between conditions (baseline and extension), paired-sample t-tests were used. Paired *t*-tests were also used to compare mood ratings between conditions. The three daily mood values were averaged to create one mood value for each day. P-values less than .05 were considered statistically significant.

RESULTS

Twenty-seven adults (average age 24.37 ± 5.41 years; 40.1% [11/27] female) participated. Of the 27 participants, 11 (40.1%) were White/Caucasian, 6 (22.2%) were Asian, 5 (18.5%) were Black/African American, and 5 (18.5%) were more than one racial/ethnic category. Five (18.5%) of those participants were Latino/Hispanic. Given that this study was testing the "pure" effects of sleep extension on mood and fatigue, we recruited participants free of mood disturbances. Demographics are presented in Table 1. Participants had relatively low Body Mass Index (BMI), anxiety (via the State-Trait Anxiety Inventory[21]), and depression levels (via Beck's Depression Inventory[20]). Lastly, participants, by design, were "intermediate" chronotypes on the Morningness-Eveningness Questionnaire[22]. Therefore, any detected effects of sleep extension were not due to extreme "morning" or "evening" chronotypes.

Table 1. Participant demographics.

Metric	Mean	SD
Age	24.41	5.29
BMI	24.59	3.12
MEQ	53.93	6.96
BDI	1.04	1.28
STAI	26.59	5.36

BMI: Body mass index; MEQ: Morning-eveningness questionnaire; BDI: Beck's depression Inventory; STAI: State-Trait Anxiety Inventory. SD = standard deviation of the mean.

Sleep Parameters

Mean sleep duration during the at-home, baseline, and sleep extension phases are depicted in Figure 2. In the 14 days at-home phase, participants averaged 452.32 ± 53.11 minutes (7.53 hours) of sleep per night, consistent with a "healthy" sleep duration (according to the joint task force of the American Academy of Sleep Medicine and the Sleep Research Society[27]). During the in-lab baseline nights, participants slept 465.92 ± 35.59 minutes (7.75 hours), which was not significantly different from the at-home phase ($p = .10$). During the sleep extension phase, however, participants slept an average of 561.67 ± 33.87 minutes (9.36 hours) per night, significantly more than during the at-home phase ($p < .001$).

Sleepiness

Despite a significant increase in sleep time, there was not a significant decrease in subjective sleepiness, as measured with the KSS (baseline average 3.86 ± 1.28; extension average 3.48 ± 1.72; $t(25) = 1.38$; $p = .18$), likely because normally-sleeping, healthy individuals were recruited.

Impact of sleep on mood

As shown in Table 2, counter to predictions, there was no detectable effect of sleep extension on mood ratings (vigor: ($t(26) = 1.74$, $p = .09$; restlessness: $t(26) = .24$, $p = .81$; depression: ($t(26) = -.18$, $p = .86$; anger: $t(26) = -.49$; $p = .63$; anxiety: $t(26) = .97$, $p = .34$; happiness: ($t(26) = 1.50$, $p = .15$).

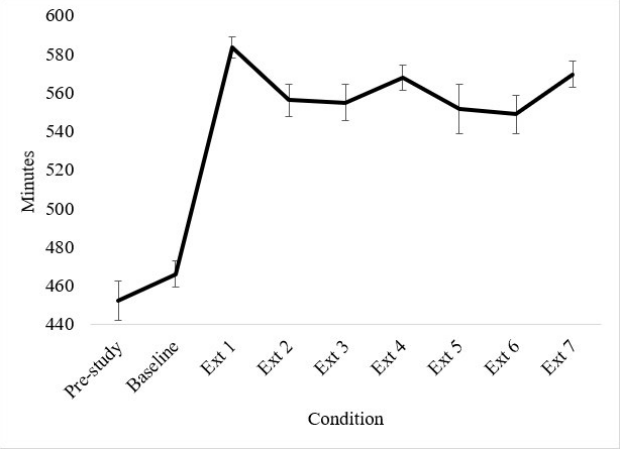

Figure 2. Total sleep time during the pre-study, baseline, and sleep extension phases.

Table 2. Mood ratings during the baseline and extension study phases.

Construct	Baseline		Extension	
	Mean	SD	Mean	SD
Vigor	3.12	1.21	2.87	1.24
Restlessness	0.69	0.82	0.67	0.82
Depression	0.24	0.52	0.26	0.68
Anger	0.25	0.49	0.23	0.51
Fatigue*	1.61	1.28	1.28	1.07
Anxiety	0.37	0.56	0.29	0.41
Happiness	4.2	1.06	3.93	1.21
Sleepiness	3.86	1.28	3.48	1.38

SD = standard deviation of the mean. * indicates a statistically significant difference between the two study phases.

However, sleep extension significantly reduced fatigue, relative to baseline ($t(26) = 2.36$, $p = .03$). Notably, fatigue was decreased despite a lack of change in subjective sleepiness, underscoring the differentiation of these constructs.

DISCUSSION

In the current study, in a sample of healthy, normally-sleeping young adults, sleep extension significantly improved fatigue below baseline levels. Interestingly, fatigue was reduced in the absence of an improvement in subjective sleepiness/alertness. Our study also demonstrated that one week of sleep extension had no detectable effect on self-reported mood. These results indicate sleep extension is a viable tool for lowering fatigue in this population, yet potentially not an effective tool for improving mood.

As discussed, previous studies have shown that sleep extension can improve cognitive and physical performance, as well as physiological markers of health beyond baseline levels. For example, one study showed that 7 days of sleep extension improved psychomotor vigilance test (PVT) performance (a proxy of attention and alertness) beyond normal levels[28]. Also, previous work has shown improvements in athletic performance following sleep extension[29]. Finally, additional evidence suggests sleep extension can improve metabolic processes such as those that determine insulin sensitivity[30]. Extending this literature, we have shown fatigue was significantly reduced following a brief bout (merely 7 days) of sleep extension, in the absence of changes in sleepiness or mood.

Our findings are consistent with another study that also assessed mood and fatigue in the context of sleep extension[17]. As mentioned, in the previous study, participants slept ad-libitum until their objective sleepiness was low. Potentially as a result of that sleep extension, fatigue decreased and vigor increased. In the current study, participants extended their sleep for just 7 days, and a significant reduction in fatigue was observed. Therefore, a brief period of sleep extension, which is more ecologically valid and feasible the previous approach, effectively reduces fatigue.

Differentiating fatigue and mood

Contrary to predictions, we did not find a detectable change in subjective mood scores (despite reductions in self-reported fatigue) following sleep extension. However, the observation that sleep extension impacted fatigue and not mood is perhaps not surprising considering fatigue is often considered separate from other facets of mood. In fact, when using the ANAM mood scale and the Profile of Moods Scale (POMS), which also contains both mood and fatigue items, researchers often separate fatigue from other items for analyses. Nevertheless, fatigue has been considered a negative mood because it is highly inter-correlated with depression, confusion, and anger[31,32]. The current results suggest - at least in the context of sleep extension - fatigue is a construct that is distinguished from the other mood items in these batteries.

It is also important to note that there are other plausible explanations for why we may not have detected changes in mood. First, it is possible that these mood constructs were simply not impacted by sleep extension because they were at "ceiling" prior to sleep manipulation. Alternatively, the effect of sleep extension on mood may have simply been too small to be detected by this tool. Further, changes in mood may not have been detectable by participants themselves. Previous work has shown that individuals are aware of changes in brain state when those changes are large but not when they are gradual[33]. For instance, when participants went from normal sleep to total sleep deprivation, there was a large change in subjective mood. However, when participants lost only one hour of sleep per night over the course of several nights (totaling the same amount of sleep deprivation overall), reports of well-being remained relatively stable over time, potentially because the brain was able to adapt to the small state changes. Therefore, given the gradual accumulation of extra sleep in the current protocol, it is possible that participants could not subjectively detect or articulate changes in mood.

Differentiating fatigue and sleepiness

The reduction of subjective fatigue in this sample is particularly interesting considering that subjective sleepiness levels were unchanged following sleep extension. This dissociation of results highlights the separateness of these complex constructs. Sleepiness is typically considered a phenomenon directly related to the sleep/wake cycle. Johns defined sleepiness as a physiological state that combines several wake- or sleep-promoting components: circadian rhythmicity, homeostatic drive, environmental, and behavioral factors[34]. Fatigue, on the other hand, which is seemingly more difficult to describe, has been defined as "weariness" related to a lack of motivation, weakness or depleted energy[35], or, alternatively, a failure to initiate or sustain tasks requiring motivation[36].

Several investigations have found poor sleep influences fatigue independent of sleepiness. For instance, a previous study found individuals with insomnia had abnormally high levels of fatigue in the absence of sleepiness[37]. Similarly, Chervin and colleagues found individuals with untreated obstructive sleep apnea were more likely to report fatigue than sleepiness[38]. Lastly, individuals with a history of traumatic brain injury (TBI), who have characteristic sleep alterations[39], experience higher levels of fatigue than uninjured controls, but not higher levels of sleepiness[36]. These results indicate fatigue is increased by poor sleep quality, even when sleepiness remains relatively unaffected. Adding to these findings, in our study, we found sleep extension differentially impacted fatigue and sleepiness.

Additionally, cross-sectional studies have shown that longer sleep is associated with feelings of fatigue and lethargy[40], leading some to speculate that long sleep might cause these negative consequences[41]. Although our findings do not provide information on whether there is a positive or negative impact of "chronic" (i.e., long-term) extended sleep, they do support the

idea that longer-than-usual sleep affords benefits, rather than leading to negative consequences. Taken together, our findings indicate that some of the previously observed associations between longer sleep and fatigue may not be due to increased sleep length, but rather caused by another factor that influences both long sleep time and fatigue (e.g., inflammation, depression)[42].

Neural underpinnings of fatigue and sleep extension

Fatigue is a physiologically complex, multifaceted symptom. Nevertheless, several attempts have been made to quantify the neural signature of subjective fatigue in healthy, young adults. For instance, one investigation found subjective fatigue was related to decreased oxygenated hemoglobin concentration in the frontal cortex and superior temporal cortex[43]. Similarly, in a study in which fatigue was induced by a continuous performance task, there were detectable alterations in cerebral blood flow in the frontal lobe when fatigue was present[44]. Taking these results together, it seems subjective fatigue is related to alterations in frontal lobe functioning. Therefore, for sleep extension to be effective in decreasing fatigue (i.e., reversing these effects), sleep extension would need to improve frontal lobe functioning. Indeed, a recent study demonstrated that sleep extension increases frontal lobe activation and connectivity with other critical regions[45], suggesting increased frontal lobe activity may be the underlying mechanism behind the reduction of fatigue observed in our study.

Strengths and weaknesses

There are several strengths of this study that should be noted. The young adult participants in this study were, by design, exceptionally healthy. Individuals with co-morbidities and sleep disorders were ineligible for participation. Furthermore, critically, individuals with high levels of anxiety and depressive symptoms were not included, allowing us to isolate mood and fatigue in absence of these confounding factors. The observed effects of sleep extension on fatigue therefore cannot be attributed to any of the aforementioned disorders. This point is critical, as fatigue has been previously associated with depression[46], co-morbidities (e.g., diabetes)[47], and obstructive sleep apnea[48], among many other disorders.

This study is also strengthened by the fact that it was relatively ecologically valid. Although participants slept in the lab during the baseline and sleep extension phases of the study, they were not required to remain in the lab during the daytime. During the study, participants continued with their typical day-to-day activities while also extending their sleep. This demonstrates (1) sleep extension is a feasible addition to one's typical routine, and (2) fatigue can be reduced by sleep extension in a real-world setting even when typical daily stressors (e.g., work and social stressors) are present.

Our study has a number of limitations that should be addressed as well. First, there was no control group and therefore it is not possible to rule out the possibility that any of the observed (and unobserved) changes are due to other factors. However, the within-subject design used here allowed us to compare participants with themselves, minimizing the chances of an extraneous factor causing these results.

Additionally, as mentioned, this study comprised a sample of healthy, young adults. Although we feel this is one of the strengths of the study, it is also potentially a weakness, as this sample may not be generalizable to older or younger populations. There are known differences in emotional processing in older adults[49], and younger populations, such as adolescents[50]. Therefore, sleep extension may differentially impact those populations. Future work should aim to assess sleep extension as a tool for fatigue, sleepiness, or mood improvement in different clinical populations as well as in the general population. Future work should also account for potential confounders, such as occupation, and dietary patterns.

Conclusions

In summary, the present results show that sleep extension significantly lowers subjective fatigue levels but does not significantly improve other aspects of mood or alertness levels in healthy, non-sleepy young adults. Sleep extension could therefore be used as an adjunctive treatment for individuals experiencing a variety of problems such as high levels of stress and burn out. Future work should aim to assess whether sleep extension is a viable tool in clinical populations (e.g., those with Chronic Fatigue Syndrome).

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REFERENCES

1. Banks S, Van Dongen HP, Maislin G, Dinges DF. Neurobehavioral dynamics following chronic sleep restriction: dose-response effects of one night for recovery. Sleep. 2010;33:1013-26.
2. Horne J, Anderson C, Platten C. Sleep extension versus nap or coffee, within the context of 'sleep debt'. J Sleep Res. 2008;17:432-6.
3. Horne J. Sleepiness as a need for sleep: when is enough, enough? Neurosci Biobehav Rev. 2010;34:108-18.
4. Hayashi M, Ito S, Hori T. The effects of a 20-min nap at noon on sleepiness, performance and EEG activity. Int J Psychophysiol. 1999;32:173-80.
5. Tamaki M, Shirota A, Hayashi M, Hori T. Restorative effects of a short afternoon nap (<30 min) in the elderly on subjective mood, performance and eeg activity. Sleep Res Online. 2000;3:131-9.
6. Wiegand M, Berger M, Zulley J, Lauer C, von Zerssen D. The influence of daytime naps on the therapeutic effect of sleep deprivation. Biol Psychiatry. 1987;22:389-92.
7. Kaida K, Takahashi M, Otsuka Y. A short nap and natural bright light exposure improve positive mood status. Ind Health. 2007;45:301-8.

8. Dinges DF, Whitehouse WG, Orne EC, Orne MT. The benefits of a nap during prolonged work and wakefulness. Work & Stress An International Journal of Work, Health & Organisations 1988;2:139-53.
9. Caldwell L, Caldwell J, Colon J, S. Ruyak P, Ramspott S. Recovery of Sleep, Performance, and Mood Following 38 Hours of Sleep Deprivation Using Naps as a Countermeasure1998.
10. Daiss SR, Bertelson AD, Benjamin LT, Jr. Napping versus resting: effects on performance and mood. Psychophysiology. 1986;23:82-8.
11. Curran SLA, Michael A. Studts, Jamie L. Short Form of the Profile of Mood States (POMS-SF): Psychometric information. 1995;7:80-3.
12. Balkin TJ, Wesensten NJ. Differentiation of sleepiness and mental fatigue effects. Cognitive fatigue: Multidisciplinary perspectives on current research and future applications. Washington, DC, US: American Psychological Association; 2011. p. 47-66.
13. Belenky G, Wesensten NJ, Thorne DR, Thomas ML, Sing HC, Redmond DP, et al. Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: a sleep dose-response study. J Sleep Res. 2003;12:1-12.
14. Brice C, Smith A. The effects of caffeine on simulated driving, subjective alertness and sustained attention. Hum Psychopharmacol. 2001;16:523-31.
15. Schmid SM, Jauch-Chara K, Hallschmid M, Schultes B. Mild sleep restriction acutely reduces plasma glucagon levels in healthy men. J Clin Endocrinol Metab. 2009;94:5169-73.
16. Ikegami K, Ogyu S, Arakomo Y, Suzuki K, Mafune K, Hiro H, et al. Recovery of cognitive performance and fatigue after one night of sleep deprivation. J Occup Health. 2009;51:412-22.
17. Kamdar BB, Kaplan KA, Kezirian EJ, Dement WC. The impact of extended sleep on daytime alertness, vigilance, and mood. Sleep Med. 2004;5:441-8.
18. Carskadon MA, Dement WC, Mitler MM, Roth T, Westbrook PR, Keenan S. Guidelines for the multiple sleep latency test (MSLT): a standard measure of sleepiness. Sleep. 1986;9:519-24.
19. Wittmann M, Dinich J, Merrow M, Roenneberg T. Social jetlag: misalignment of biological and social time. Chronobiology international. 2006;23:497-509.
20. Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. Arch Gen Psychiatry. 1961;4:561-71.
21. Spielberger CD, Gorsuch, R.L., Lushene, R., Vagg, P.R. and Jacobs, G.A. . Manual for the State-Trait Anxiety Inventory. Consulting Psychologists Press, Palo Alto. 1983.
22. Horne JA, Ostberg O. A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. Int J Chronobiol. 1976;4:97-110.
23. Simonelli G, Mantua J, Gad M, St Pierre M, Moore L, Yarnell AM, et al. Sleep extension reduces pain sensitivity. Sleep Med. 2018;54:172-6.
24. Johnson DR, Vincent AS, Johnson AE, Gilliland K, Schlegel RE. Reliability and construct validity of the Automated Neuropsychological Assessment Metrics (ANAM) mood scale. Arch Clin Neuropsychol. 2008;23:73-85.
25. Kaida K, Takahashi M, Akerstedt T, Nakata A, Otsuka Y, Haratani T, et al. Validation of the Karolinska sleepiness scale against performance and EEG variables. Clin Neurophysiol. 2006;117:1574-81.
26. Mantua J, Gravel N, Spencer RM. Reliability of Sleep Measures from Four Personal Health Monitoring Devices Compared to Research-Based Actigraphy and Polysomnography. Sensors (Basel). 2016;16.
27. Consensus Conference P, Watson NF, Badr MS, Belenky G, Bliwise DL, Buxton OM, et al. Joint Consensus Statement of the American Academy of Sleep Medicine and Sleep Research Society on the Recommended Amount of Sleep for a Healthy Adult: Methodology and Discussion. Sleep. 2015;38:1161-83.
28. Rupp TL, Wesensten NJ, Bliese PD, Balkin TJ. Banking sleep: realization of benefits during subsequent sleep restriction and recovery. Sleep. 2009;32:311-21.
29. Mah CD, Mah KE, Kezirian EJ, Dement WC. The effects of sleep extension on the athletic performance of collegiate basketball players. Sleep. 2011;34:943-50.
30. Leproult R, Deliens G, Gilson M, Peigneux P. Beneficial impact of sleep extension on fasting insulin sensitivity in adults with habitual sleep restriction. Sleep. 2015;38:707-15.
31. Watson D, Clark LA. Measurement and mismeasurement of mood: recurrent and emergent issues. J Pers Assess. 1997;68:267-96.
32. Watson D, Tellegen A. Toward a consensual structure of mood. Psychol Bull. 1985;98:219-35.
33. Drake CL, Roehrs TA, Burduvali E, Bonahoom A, Rosekind M, Roth T. Effects of rapid versus slow accumulation of eight hours of sleep loss. Psychophysiology. 2001;38:979-87.
34. Johns M. Rethinking the assessment of sleepiness. Sleep Med Rev. 1998;2:3-15.
35. Pigeon WR, Sateia MJ, Ferguson RJ. Distinguishing between excessive daytime sleepiness and fatigue: toward improved detection and treatment. J Psychosom Res. 2003;54:61-9.
36. Beaulieu-Bonneau S, Morin CM. Sleepiness and fatigue following traumatic brain injury. Sleep Med. 2012;13:598-605.
37. Hossain JL, Ahmad P, Reinish LW, Kayumov L, Hossain NK, Shapiro CM. Subjective fatigue and subjective sleepiness: two independent consequences of sleep disorders? J Sleep Res. 2005;14:245-53.
38. Chervin RD. Sleepiness, fatigue, tiredness, and lack of energy in obstructive sleep apnea. Chest. 2000;118:372-9.
39. Mantua J, Grillakis A, Mahfouz SH, Taylor MR, Brager AJ, Yarnell AM, et al. A systematic review and meta-analysis of sleep architecture and chronic traumatic brain injury. Sleep Med Rev. 2018;41:61-77.
40. Grandner MA, Kripke DF. Self-reported sleep complaints with long and short sleep: a nationally representative sample. Psychosom Med. 2004;66:239-41.
41. Grandner MA, Drummond SP. Who are the long sleepers? Towards an understanding of the mortality relationship. Sleep Med Rev. 2007;11:341-60.
42. Mantua J, Spencer RMC. Exploring the nap paradox: are mid-day sleep bouts a friend or foe? Sleep Med. 2017;37:88-97.
43. Suda M, Fukuda M, Sato T, Iwata S, Song M, Kameyama M, et al. Subjective feeling of psychological fatigue is related to decreased reactivity in ventrolateral prefrontal cortex. Brain Res. 2009;1252:152-60.
44. Lim J, Wu WC, Wang J, Detre JA, Dinges DF, Rao H. Imaging brain fatigue from sustained mental workload: an ASL perfusion study of the time-on-task effect. Neuroimage. 2010;49:3426-35.
45. Motomura Y, Kitamura S, Nakazaki K, Oba K, Katsunuma R, Terasawa Y, et al. Recovery from Unrecognized Sleep Loss Accumulated in Daily Life Improved Mood Regulation via Prefrontal Suppression of Amygdala Activity. Front Neurol. 2017;8:306.
46. Demyttenaere K, De Fruyt J, Stahl SM. The many faces of fatigue in major depressive disorder. Int J Neuropsychopharmacol. 2005;8:93-105.
47. Fritschi C, Quinn L. Fatigue in patients with diabetes: a review. J Psychosom Res. 2010;69:33-41.
48. Harris M, Glozier N, Ratnavadivel R, Grunstein RR. Obstructive sleep apnea and depression. Sleep Med Rev. 2009;13:437-44.
49. Jones BJ, Schultz KS, Adams S, Baran B, Spencer RMC. Emotional bias of sleep-dependent processing shifts from negative to positive with aging. Neurobiol Aging. 2016;45:178-89.
50. Yurgelun-Todd D. Emotional and cognitive changes during adolescence. Curr Opin Neurobiol. 2007;17:251-7.