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The Effects of Experimental Sleep Extension in Middle-to-Older-Aged Healthy Sleepers

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Abstract

Objectives To determine the feasibility for middle-aged and older adults to extend their time in bed by 2 h per night for 3 consecutive weeks. Other aims were to examine the effects of sleep extension on mood, cognitive performance, and cardiovascular health.

Methods Ten healthy middle-aged to older adults (9 women; $M = 65.20 \pm 4.78$ years) who reported regularly sleeping 6 to 8 h per night participated in a randomized controlled cross-over study: 3 weeks of both habitual sleep and extended sleep (1-week recovery between treatments). Participants were asked to spend 2 additional hours in bed per night during sleep extension. Cognitive (e.g., errors, response time), psychological (e.g., depression, anxiety, mood), and physiological measures (e.g., inflammation, glucose, triglycerides, blood pressure) were assessed.

Results Compared with habitual sleep, time in bed increased 81.63 ± 33.11 min and total sleep time increased 66.33 ± 28.64 min during sleep extension; these variables did not significantly change during baseline or the habitual sleep treatment. No significant treatment differences were found in the cognitive, psychological, or physiological measures.

Discussion Neither significant positive nor negative effects of sleep extension were found for any of the variables. In terms of feasibility, it was difficult for the participants to extend their time in bed and, subsequently, attain more sleep by the targeted amount. Sleep extension by a greater degree or longer period of times might be more likely to elicit positive or negative effects.

Keywords
- inflammation
- cognition
- healthy volunteers
- middle-aged
- sleep

Introduction

Epidemiological studies have consistently shown that both short (< 6h) and long sleep (> 8h) are associated with mortality[1] and morbidity, including cardiovascular disease,[2] diabetes,[3] depressed mood,[4] and impaired cognitive function,[5] whereas approximately 7 to 8 h of sleep has been associated with the lowest health risks.[3,6–8] Additionally, prospective studies (e.g., Ferrie et al.[9]) have found that compared with baseline, decreases and increases in sleep

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duration have been associated with increased mortality risk.

Multiple studies have indicated that the risks associated with long sleep are at least as great, if not greater, than the risks of short sleep.[10,11] Nonetheless, the notion that long sleep is an underlying cause of mortality and morbidity has been challenged. The "reverse causality" explanation (i.e., illness causing long sleep) has been advanced,[12] but many studies of long sleepers have extensively controlled for health and risk factors. Moreover, some prospective studies established good health of the participants at baseline.[13,14] However, it is noteworthy that these epidemiological studies were observational; therefore, no causal inferences can be made between sleep duration and health risks from these studies.

The argument that there are no plausible mechanisms to explain how chronic long sleep could be hazardous[12,15] has also been contradicted by multiple lines of evidence. Indeed, just 2 to 5 days of bedrest, which leads to increased sleep, elicits significant impairments in insulin sensitivity and cardiovascular function[16,17]; chronic exposure to periods of greater than 1h per day of completely sedentary behavior is associated with health risks[18] and worse sleep.[19] Furthermore, long sleep is associated with relatively low levels of daytime physical activity, especially in older individuals.[20]

Long sleep is associated with increased sleep fragmentation, which is linked to poor health outcomes in epidemiological and experimental studies.[21] Long sleep has also been associated with inflammatory markers,[22] possibly attributable to sleep fragmentation. It has been suggested that the proinflammatory cytokines (e.g., interleukin-6, IL-6) may mediate feelings of lethargy[23] which could, in turn, lead to further cytokine imbalance.[23]

Reduction in light exposure as a result of extended sleep could also lead to depression,[24] delayed circadian time, and a shorter perceived day length. Reductions in photoperiods have been associated with impaired health in some species.[25] Furthermore, depression and poorer cognitive function have been associated with longer sleep duration in adults 65 years and older.[26] Poorer cognitive function has also been found in middle-aged adults compared to age-matched adults with average sleep duration,[5,27–29] and a decline in cognitive function (advanced cognitive aging) over time has been found in long sleepers.[5,28,29] One potential explanation for the link between sleep duration, cognition, and aging is increased amyloid-β burden.[27] Sleep fragmentation is another potential explanation for the link between lower cognitive function and long sleep duration.[27]

Also contradicted by the literature is the argument that there is no experimental evidence that it is harmful for a healthy adult to sleep 9 or more hours.[15] Several experimental studies of normal duration sleepers have found negative effects of 1 to 2 nights of 10+ h of sleep duration on cognitive function,[30,31] and 2h of extended sleep for one week has had negative effects on depressed mood[32] and IL-6 inflammatory markers.[32] Nonetheless, the literature is relatively sparse,[33] with multiple limitations, including limited focus on objective measures of health. However, recent studies of average sleepers conducted by Gonzales et al.[34] and Clark et al.[35] found no effects of 5 to 6 nights of sleep extension (from 8–10 hours) on multiple measures of cardiovascular health or cognitive function.

It is noteworthy that experimental manipulation of sleep duration in these studies was short-term and intended to impact rapidly reversible outcomes. The results of these studies may not be directly comparable to long-term increases in sleep duration.

Additionally, it has been hypothesized that long sleepers likely experience an increased sleep pressure that leads to the perceived need for more sleep. In experimental sleep extension studies of healthy individuals, "forcing" sleep (as opposed to increased pressure) may not necessarily be directly comparable to individuals with increased sleep pressure. On the other hand, some evidence indicates that longer sleep in older adults partly reflects merely an assumption that they need at least 8 hours of sleep.[36]

Thus, it remains debatable whether long sleep truly causes negative effects. There is a particular need to investigate this question in middle-aged and older adults ($\geq$ 65 years old) for whom the largest association of long sleep with health risks has been found. On the other hand, it has been argued that despite documented declines in objective sleep with aging,[37,38] there is an equal need for sleep with aging,[39] and that older adults would benefit from sleep extension.

One aim of this sleep extension study was to determine whether it was feasible for middle-aged and older adults to extend time in bed (TIB) and consequently increase total sleep time (TST). Other aims were to examine the effects of 3 weeks sleep extension (2 hours) on mood state, cognitive performance, and cardiovascular and metabolic health.

Materials and Methods

Trial Design

This randomized controlled trial utilized a cross-over design. The study was reviewed and approved by the University of South Carolina (USC) Institutional Review Board ethics committee (approval number: Pro00024377).

Participants

Fourteen individuals who met the inclusion criteria were assessed. Three participants dropped out of the study due to schedule changes and/or time constraints. Eleven participants completed the study and were assessed on cognitive, emotional, and inflammatory measures in a cross-over design. One participant was excluded from data analysis due to noncompliance to the study protocol (participant slept less than 5 hours per night on average), leaving 10 participants included in the final analyses (1 male; ages ranged from 54–70 years old, $M = 65.20 \pm 4.78$ years). Self-reported TST was an average of 7.00 ± 0.745, and body mass index averaged 27.33 ± 3.66.

Screening and Exclusions

Participants were initially screened with a brief phone interview to indicate interest and basic age, health, and sleeping inclusion criteria. To reduce the chance of expectancy or demand biases, prospective participants were

informed that the intervention could have positive effects, negative effects, or no effects. Prospective participants who met the initial criteria (n = 30) were invited to the laboratory for a more extensive study orientation and screening.

After signing an informed consent form approved by the University of South Carolina's Institutional Review Board (IRB), prospective participants completed screening questionnaires, tests of cognitive function, and they were interviewed about their sleep, health, and behavioral patterns (i.e., self-reported usual sleep patterns, mental health, general health, etc.). The final screen involved an interview with a sleep physician (n = 15), who assessed participants for serious health conditions and sleep disorders, such as sleep apnea. Despite the interview with a sleep physician, it is possible that participants may have had sleep conditions like sleep apnea since sleep tests or polysomnography was not administered, which is a limitation of the current study.

Inclusion criteria were ages between 50 and 79, no significant health conditions, normal cognitive function (Mini-Mental State Examination $\leq$ 26), no known sleep disorders or diseases, and typically sleeping between 6 and 8 hours per night. Sleep duration of 6 to 8 h falls within the healthy, typical sleep duration of middle-aged-to-older adults.[38] Exclusion criteria included recent shiftwork (previous 2 months) or transmeridian travel (previous 4 weeks), or plans of either during the study period; use of hypnotics or other medications to promote sleep; any medical, neurologic, or psychiatric illness causing long sleep; factors associated with significant changes in inflammation (a key outcome variable), including several medical disorders (e.g., rheumatoid arthritis), medications (e.g., steroids) current smoking, obesity (body mass index > 33), and greater than 3 hours of moderate-to-vigorous exercise per week. Demographic information was collected at the time of consent as well as a list of all medications taken.

Interventions

Baseline

During a 1-week baseline, before each treatment, participants were asked to follow their usual sleep-wake schedules. During baseline, pretreatment measures were assessed, and baseline also served as a final screen to establish that the participants slept 6 to 8 hours. Participants were excluded if actigraphic TIB was outside of the 6 to 8 hour range.

Experimental Treatments and Randomization

Following baseline, participants were randomly assigned to 1 of 2 3-week treatments: (1) extended TIB, or (2) habitual TIB: The randomization was stratified by median duration of actigraphic baseline TST ($<$ 7 vs. $\geq$ 7 h). At the end of each week, a research staff member met with the participant to download actigraph data, review the sleep log, encourage adherence to the treatments, and troubleshoot any difficulties.

Randomization

The primary author conducted the randomizations, which were stratified by median duration of actigraphic baseline TST ($<$ 7 vs. 7 h). The primary author also enrolled participants on the study and assigned them to interventions.

Blinding

The trained clinical psychology graduate students were blinded in order to remove bias during the clinical assessments. Blinding was achieved by only supplying the participant number (with no indication of treatment assignment or the condition completed). The primary author and other staff who assistedwith conducting weekly visitswere not blinded to treatment order.

Extended TIB Treatment

In the extended TIB treatment, participants were asked to follow a fixed sleep schedule and extend their TIB by 2 hours greater than their median baseline actigraphic TIB. This was accomplished by advancing bedtime, delaying arising time, or some combination of these changes in accordance with the preferences and sleep tendencies of each participant. Participants were encouraged to extend sleep in a way that was convenient for them (e.g., go to bed earlier, wake later, or a combination of the two), and consistent with their individual sleep patterns. For example, if they experienced sleepiness in the evening or trouble arising in the morning, it was suggested that they might try going to bed earlier and sleeping later, respectively. They were also asked to remain consistent throughout the sleep extension weeks. Four participants extended sleep through a combination of earlier bedtimes and later wake-times, three participants extended sleep by waking later, two participants had earlier bedtimes, and one participant's data for bedtime/wake time were unavailable. There were no patterns based on group order.

Habitual TIB Treatment

In the habitual sleep condition, participants were asked to follow a fixed sleep schedule, in which their TIB was the same as their median baseline TIB.

Cross-over Design

After completion of the baseline and the first 3-week treatment, participants completed a 1-week recovery period to avoid carry-over effects. Participants then underwent a second 1-week baseline and crossed over to the other treatment. Participants were compensated $200 (prorated) for completing the study.

Outcomes

►**Table 1** displays the time points in which each assessment occurred.

Sleep Variables

Actigraphic Sleep

Throughout each baseline and each 3-week treatment, participants wore actigraphic wrist monitors (Phillips Respironics Actiwatch Spectrum device – Philips Respironics, Murrysville, PA, USA) for continuous assessment of sleep-wake patterns. Total sleep time, TIB, sleep

Table 1 Experimental measures during 1-week baseline and 3-week sleep extension and control groups cross-over study. Recovery week occurred during week 5.

Baseline		Treatment (Control vs. Sleep Extension)		
	Follow-up appts.		Follow-up appts.	Condition follow-up 1
Daily week 1	End week 1	Daily weeks 2–4	End week 2-4	End week 4
Actigraphy	Cognitive battery	Actigraphy	Sleepiness	Blood draw
Sleep diary	Sleepiness	Sleep diary	Fatigue	Cognitive battery
Mood diary	Fatigue	Mood diary	Depression	
	Depression		Anxiety	
	Anxiety		BP HR	
	BP HR			
Baseline		**Treatment (Control vs. Sleep Extension)**		
	Follow-up appts.		Follow-up appts.	Condition follow-up 2
Daily week 6	End week 6	Daily weeks 7-9	End week 7-9	End week 9
Actigraphy	Sleepiness	Actigraphy	Sleepiness	Blood draw
Sleep diary	Fatigue	Sleep diary	Fatigue	Cognitive battery
Mood diary	Depression	Mood diary	Depression	
	Anxiety		Anxiety	
	BP HR		BP HR	

Notes: Sleepiness (weekly): Epworth Sleepiness Scale; Depression: Beck Depression Inventory II (BDI-II; weekly), Hamilton Depression Scale (end of each baseline and end of each treatment); Anxiety: State Trait Anxiety Scale (weekly), Hamilton Anxiety Scale (end of each baseline and end of each treatment); Cognitive Battery (end of first baseline and end of each treatment): Block Pattern Memory Task, Sentence Memory Task, Trail Making Test, Psychomotor Vigilance Test, Stroop Test; BP (blood pressure) and HR (heart rate) assessed weekly.

fragmentation index, sleep efficiency, and sleep onset latency were also assessed using the Cole-Kripke algorithm.[40] Actigraphic algorithms calculated sleep fragmentation index as the sum of the percentage of mobile bouts and percentage of immobile bouts less than 1 minute to the number of immobile bouts. Sleep efficiency was calculated by the actigraph as the percentage of time spent asleep within the TIB interval.

Napping behavior was evaluated by examining activity outside of the nighttime sleep times. Inactivity over 30 consecutive minutes was considered to be potential napping behavior. Although participants were encouraged to use an event marker button on the actiwatches, few participants consistently used the button outside of nighttime sleep/wake. Overall, participants likely napped minimally throughout the study, but three participants may have reduced napping behavior slightly during the sleep extension weeks. Participants were reminded to keep their same habits throughout the habitual sleep and sleep extension weeks, so if they napped during habitual weeks, they were to continue napping per their usual schedule. Actigraphy indicated that a few participants were inactive during the afternoons or evenings (which could be interpreted as napping behavior), but most napped in the morning or afternoon inconsistently at 1 to 2 times per week.

Daily Sleep Diary

In a daily morning sleep diary, participants reported TIB, wake time, time out of bed, TST, and overall quality of sleep.

Psychological Assessments

Clinical Assessment of Depressed Mood and Anxiety:

At the end of each baseline and the end of each treatment period, supervised, blinded, and trained clinical psychology graduate students rated the participants with the Hamilton Depression Rating Scale[41] and the Hamilton Anxiety Rating Scale.[42] The Hamilton Depression Scale contains 21 items (first 17 scored) and must be administered by a trained clinician; higher scores indicate more severe depression (score ranges include: >7 no depression, 7 to 17 mild depression, 18 to 24 moderate depression, and 25+ severe depression). The Hamilton Anxiety Scale scores range from 0 to 56, and it must be administered by a trained clinician; higher scores indicate more severe anxiety (score ranges include: <17 mild anxiety, 18 to 24 mild-moderate anxiety, and 25 to 30 moderate-severe anxiety).

Weekly Psychological Questionnaires

The participants completed the following questionnaires at the end of each baseline week and the end of each treatment week. The Beck Depression Inventory (BDI-II)[43] is a 21-item self-administered questionnaire that assesses depressive symptoms. Scores range from 0 to 63, with higher scores indicating more severe depressive symptoms (cut-off score ranges include 0–13 minimal, 14–19 mild, 20–28 moderate, and 29–63 severe). The Spielberger State Trait Anxiety Inventory (Form Y-1)[44] is a self-administered survey that contains 20 items and assesses symptoms of state-trait

anxiety; higher scores indicate greater severity of state and trait anxiety symptoms (score ranges include 20–37 no or low anxiety, 38–44 moderate anxiety, and 45–80 high anxiety). The Epworth Sleepiness Scale[45] is a self-administered 8-item measure of daytime sleepiness, with higher scores representing increased level of daytime sleepiness (score ranges include 0–5 normal daytime sleepiness, 6–10 higher normal daytime sleepiness, 11–12 mild excessive daytime sleepiness, 13–15 moderate excessive daytime sleepiness, and 16–24 severe excessive daytime sleepiness). The Multidimensional Assessment of Fatigue Scale (MAF)[46] is a self-administered scale of fatigue; higher scores on this scale indicate increased level of fatigue.

Daily Psychological Assessment

The participants completed a daily diary consisting of Likert scale questions pertaining to mood. They rated how they felt the previous day; a score of zero represented *normal* mood, -3 represented *low* mood, and +3 represented *elated* mood.

Cognitive Assessments

At the initial baseline and at the end of each 3-week treatment, working memory, sustained attention, and executive function were assessed with a cognitive performance battery. Two working memory tasks were used from the Stanford-Binet Intelligence Scale, 5th edition[47]: Block Pattern Memory Task, which tests nonverbal working memory, and the Sentence Memory Task, which tests verbal working memory. Composite scores were obtained by scoring both the verbal and nonverbal tasks, but individual scores were determined to compare verbal and nonverbal working memory ability. In order to maintain the integrity of the tests, the Stanford-Binet 5 working memory assessments were administered by two blinded, supervised clinical psychology doctoral students.

The Psychomotor Vigilance Test, which is sensitive to aging and changes in sleep,[48] was used to assess changes in reaction time associated with state dependent changes in sustained attention. The metrics that were assessed included median reaction time (RT), the fastest 10% of responses, the slowest 10% of responses, the number of response *lapses* (RT > 500 ms), and standard deviation of responses.

The Stroop Color-Word Test[49] was used to measure executive functioning and cognitive control and flexibility.[50] Interference was measured by subtracting the average time of the first two subtasks completion from the average time of third subtask completion (Interference = [(Stroop I + Stroop II) / 2] – Stroop III).[50,51] Reaction times and errors were also evaluated.

The Trail Making Test (parts A and B) was used in the cognitive battery. Specifically, Trail Making Test part B has been shown to be highly sensitive to cognitive aging and is a valid measure of executive function.[52] The reaction times of Trail Making Test completion were evaluated.

Physiological Measures

At the end of each 3-week intervention, participants underwent a 12-hr fast and blood draw (7 ml) to obtain lipid levels (lipoprotein, low-density lipoprotein, very low-density lipoprotein, and triglycerides), glucose, and an inflammation marker C-reactive protein. These assessments were all done in the morning to limit diurnal variation in these measures.

Following a 10-minute seated resting period, systolic and diastolic blood pressure, as well as heart rate, were recorded at the beginning of each weekly appointment with an Omron RS8 automatic wrist blood pressure monitor. The Omron Corporation, Kyoto Head Office, Shiokoji Horikawa, Shimogyo ku, Kyoto 600-8530, JAPAN RS8 device is a validated, easy to use blood pressure monitoring device.[53] At the beginning of each participant's weekly appointment, the device was placed on the participant's non-dominant wrist below the ulna wrist bone, per the manufacturer's instructions. After 10 minutes of quietly sitting, the blood pressure and heart rate readings were recorded, and then repeated after 2 minutes. If an error or large difference occurred between the two readings (i.e., > 10 units), the reading was taken a third time; averages of the recordings were reported.

Marker of Inflammation:

Recent infection/illness resulted in interpolation of data based on group values (n = 2; see *Data Screening*). Participants were carefully screened for alcohol consumption, smoking history, body mass index, physical activity, and use of medications (antidepressants, statins, and non-steroidal anti-inflammatory drug; NSAID),[54] as these factors may impact inflammatory marker results.

Randomization

The primary author conducted the randomizations, which were stratified by median duration of actigraphic baseline TST (< 7 vs. ≥ 7 h). The primary author also enrolled participants on the study and assigned them to interventions.

Blinding

The trained clinical psychology graduate students were blinded in order to remove bias during the clinical assessments. Blinding was achieved by only supplying the participant number (with no indication of treatment assignment or the condition completed). The primary author and other staff who assisted with conducting weekly visits were not blinded to treatment order.

Statistical Methods (Data Screening and Cleaning)

Data were initially screened for outliers (±3 standard deviations from the mean) and/or missing pieces of information. The reported data are from the 10 participants who completed the entire study.

Prior to analysis, sleep, cognitive, mood, and physiological measures were screened for equipment errors, accuracy of data entry, missing values, outliers, and fit between their distributions and the assumptions of analysis (including skewness and kurtosis) using the SPSS. Actigraph malfunction resulted in the loss of one week of an individual's sleep data from the last habitual sleep week treatment. The sleep data for the week were replaced by the mean from the same

participant's two other habitual sleep weeks. The actigraph algorithm resulted in consistently implausibly short sleep onset durations data; therefore, sleep onset data were removed from analyses. Actigraphic data were cleaned by one researcher, who used the sleep diaries to corroborate the actigraphic data. Missing values of two participants for the Stanford-Binet Intelligence Scale, 5th edition, at baseline and for the end of the habitual sleep condition were replaced by mean scores of participants during the same condition and in the same condition order.

Missing values of one participant for the Hamilton Anxiety and Depression Scales for the end of the habitual sleep condition were replaced by mean scores of participants during the same condition and in the same condition order. The single missing value of one response on the BDI-II was replaced by the mean response to the other BDI-II questions for that week.

Two participants reported infections during the blood draw at the end of the second condition; the two corresponding CRP cases were found to be univariate outliers. The average of the CRP values of all participants in the same condition and in the same condition order was calculated and substituted for those values. Any data that were not normally distributed were analyzed using methods appropriate for non-normally distributed data.

Data Analysis

In order to determine if TIB and TST were extended, *t*-test comparisons were completed on the actigraphic variables. Comparisons were made using *t*-tests (comparing habitual sleep vs sleep extension conditions). Effect sizes (Hedges' g) are also reported in all statistical analysis tables.

Table 2 Baseline comparisons for all variables to confirm control conditions (n = 10).

	Condition	*M* ± *SD*	*t*-value	*p*-value	Hedges' g
Actigraphic factors					
TIB (min)	Baseline 1	486.59 ± 32.96	-0.707	00.497	-0.204
	Baseline 2	493.24 ± 32.12			
TST (min)	Baseline 1	428.62 ± 29.64	-0.014	0.989	-0.004
	Baseline 2	428.76 ± 33.93			
Sleep onset latency (min)	Baseline 1	6.03 ± 4.84	-0.988	0.349	-0.286
	Baseline 2	7.15 ± 3.47			
Sleep efficiency (%)	Baseline 1	88.08 ± 2.33	1.040	0.326	0.300
	Baseline 2	86.90 ± 3.54			
Wake after sleep onset (min)	Baseline 1	47.55 ± 15.52	-1.201	.260	-0.347
	Baseline 2	52.25 ± 19.24			
Awakenings (#)	Baseline 1	23.75 ± 4.90	-0.535	0.606	-0.154
	Baseline 2	24.30 ± 4.55			
Fragmentation (%)	Baseline 1	26.87 ± 4.39	1.345	0.211	0.389
	Baseline 2	25.65 ± 5.30			
Psychological Factors					
Hamilton Anxiety Scale	Baseline 1	1.70 ± 1.70	**	0.438	-0.104
	Baseline 2	1.90 ± 1.97			
Hamilton Depression Scale	Baseline 1	1.00 ± 0.943	**	0.399	-0.259
	Baseline 2	1.60 ± 1.84			
BDI-II	Baseline 1	1.40 ± 1.35	**	0.172	0.403
	Baseline 2	0.600 ± 1.26			
STAI	Baseline 1	26.90 ± 5.24	**	0.351	0.287
	Baseline 2	24.70 ± 4.35			
Mood	Baseline 1	0.886 ± 1.01	**	0.137	0.322
	Baseline 2	0.593 ± 0.948			
ESS	Baseline 1	4.20 ± 2.94	**	0.026*	0.700
	Baseline 2	2.70 ± 2.06			
MAF	Baseline 1	4.91 ± 5.28	**	0.345	0.330
	Baseline 2	3.10 ± 3.41			

Table 2 (*Continued*)

	Condition	*M* ± *SD*	*t*-value	*p*-value	Hedges' g
Physiological factors					
Systolic BP (mm Hg)	Baseline 1	126.50 ± 13.43	-0.451	0.663	-0.130
	Baseline 2	127.93 ± 15.93			
Diastolic BP (mm Hg)	Baseline 1	74.17 ± 7.85	0.490	0.636	0.142
	Baseline 2	72.83 ± 10.03			
Heart rate	Baseline 1	66.62 ± 9.26	-0.926	0.189	-0.268
	Baseline 2	68.47 ± 9.56			

Abbreviations: BP, blood pressure; ESS, Epworth Sleepiness Scale; MAF, Multidimensional Assessment of Fatigue; TIB, time in bed; TST, total sleep time.
Notes: *$p < 0.05$; **Wilcoxon Signed Rank Test performed.

Results

Baseline Measures

►**Table 2** displays the baseline data for the two time periods. Reported sleepiness was significantly lower after the second baseline (2.70 ± 2.06) compared with the first baseline (4.20 ± 2.94; $p = 0.026$). No other significant baseline differences were found.

Sleep Extension and Manipulation Check

►**Figure 1 (a) and (b)** depicts TIB and TST data, respectively. Time in bed was significantly longer during the sleep extension treatment (559.8 ± 36.9 min) compared with the habitual sleep treatment (478.2 ± 19.7 min), with a mean increase of 81.63 ± 33.11 min. Total sleep time was significantly longer in the sleep extension treatment (486.50 ± 40.98 min) compared with the habitual sleep treatment (420.17 ± 25.42 min), with a mean increase of 66.33 ± 28.64 min (►**Fig. 2**). There were no detectable differences between participants who advanced their bedtimes, delayed wake times, or both. There were no patterns based on group order.

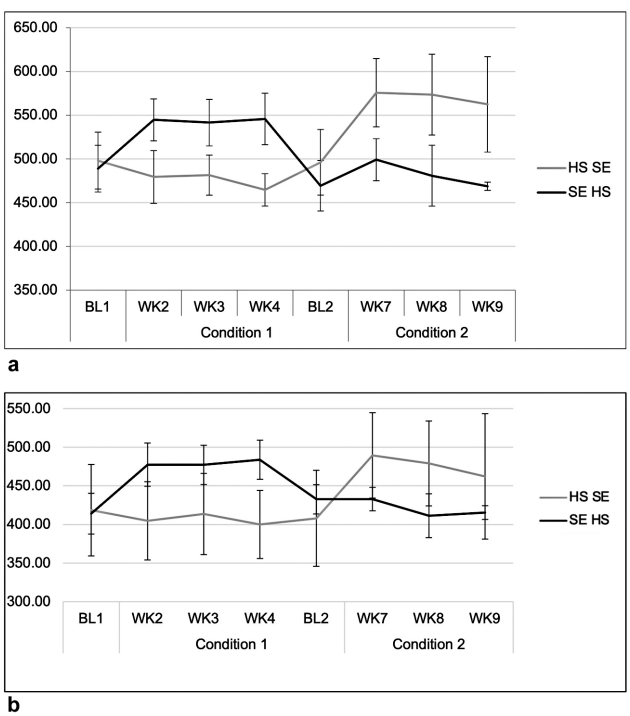

Fig. 1 (a) Figure depicts time in bed (TIB), as measured in minutes, for each condition. Gray line represents condition order of habitual sleep (HS) followed by sleep extension (SE; n = 6); black line represents condition order of SE followed by HS (n = 4). Figure 1(b) depicts total sleep time (TST), as measured in minutes, for each condition. Gray line represents condition order of SE followed by HS (n = 4). Error bars represent standard deviation.

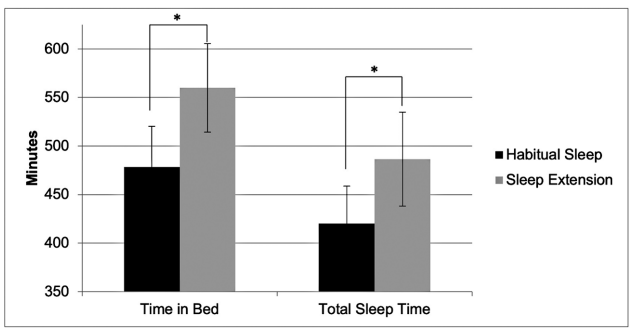

Fig. 2 Figure depicts time in bed and total sleep time, as measured in minutes, for each condition. Error bars represent standard deviation. Asterisk (*) indicates $p < 0.001$.

Actigraphic Sleep Variables

Actigraphic sleep data are presented in ►**Table 3**. Participants experienced significantly more awakenings (an average of 5.23 more awakenings) during the sleep extension treatment compared to the habitual sleep treatment ($t(9) = -4.062$, $p = 0.003$). Wake after sleep onset also increased significantly (13.79 ± 12.72 min) during the sleep extension compared with habitual sleep treatment, ($t(9) = -3.254$, $p = 0.010$). No other significant treatment effects were observed for actigraphic sleep.

Cognitive Variables

No significant treatment differences were found for Stroop Interference scores, PVT data, or Trail Making times, or Stanford-Binet 5 test scores (►**Table 4**).

Table 3 Actigraphic sleep data group comparisons.

	Condition	$M \pm SD$	*t*-value	*p*-value	Hedges g
TIB (min)	HS	478.24 ± 19.70	-7.797**	0.000027	-2.523
	SE	559.87 ± 36.88			
TST (min)	HS	420.17 ± 25.42	-7.325**	0.000044	-1.778
	SE	486.50 ± 40.98			
Sleep onset latency (min)	HS	6.37 ± 3.39	-1.027	0.331	-0.294
	SE	7.32 ± 2.44			
Sleep efficiency (%)	HS	87.86 ± 3.07	1.242	0.246	0.321
	SE	86.82 ± 2.82			
Awakenings (#)	HS	22.49 ± 4.68	-4.062**	0.003	-1.073
	SE	27.72 ± 4.22			
	HS	28.18 ± 6.36	-0.717	0.492	-0.256
Fragmentation (%)	SE	29.68 ± 4.11			
	HS	46.08 ± 12.60	-3.254*	0.010	-0.960
Wake after sleep onset (min)	SE	59.87 ± 13.64			

Abbreviations: HS, habitual sleep; SE, sleep extension; TIB, time in bed; TST, total sleep time.
Notes: *$p < 0.05$; **$p < 0.01$.

Table 4 Cognitive, mood, and psychological assessments.

	Condition	$M \pm SD$	Test Statistic	*p*-value	Hedges g*
Stroop interference (sec)	BL	37.48 ± 11.96			
	HS	33.35 ± 7.10	0.211 (*F*)	0.651	0.225
	SE	35.29 ± 9.25			
PVT mean response (log10)	BL	2.45 ± 0.048			
	HS	2.45 ± 0.053	0.013 (*F*)	0.910	0.000
	SE	2.45 ± 0.064			
PVT median response (log10)	BL	2.44 ± 0.050			
	HS	2.44 ± 0.050	0.028 (*F*)	0.869	0.000
	SE	2.44 ± 0.062			
PVT fastest 10% (log10)	BL	2.34 ± 0.036			
	HS	2.35 ± 0.036	0.121 (*F*)	0.732	-0.242
	SE	2.34 ± 0.043			
Trail making test A (sec)	BL	30.49 ± 11.83			
	HS	29.60 ± 14.09	0.352 (*F*)	0.560	-0.254
	SE	26.35 ± 10.08			
Trail making test B (sec)	BL	65.00 ± 22.49			
	HS	75.93 ± 62.20	0.140 (*F*)	0.713	-0.160
	SE	67.32 ± 38.21			
Stanford-Binet 5 nonverbal	BL	19.10 ± 2.33			
	HS	18.40 ± 2.55	0.114 (*F*)	0.739	0.075
	SE	18.60 ± 2.58			
Stanford-Binet 5 verbal	BL	19.30 ± 2.45			
	HS	20.58 ± 2.47	0.635 (*F*)	0.436	-0.341
	SE	19.80 ± 1.87			

Table 4 (*Continued*)

	Condition	*M* ± *SD*	Test Statistic	*p*-value	Hedges g*
Hamilton Anxiety Scale	BL (1&2)	1.80 ± 1.62			
	HS	2.22 ± 3.35	0.149 (*F*)	0.704	.451
	SE	2.70 ± 2.06			
Hamilton Depression Scale	BL (1&2)	1.30 ± 1.01			
	HS	1.52 ± 2.01	0.595 (*F*)	0.450	.227
	SE	2.20 ± 1.93			
Beck Depression Inventory-II	HS	1.23 ± 1.13	7.50 (*Z*)	0.527[a]	0.131
	SE	1.10 ± 1.25			
Mood scores	HS	0.729 ± 1.04	13.00 (*Z*)	0.484[a]	0.212
	SE	0.592 ± 0.806			
State-Trait Anxiety Inventory	HS	25.13 ± 5.50	18.00 (*Z*)	0.594[a]	0.124
	SE	25.34 ± 4.32			
Epworth Sleepiness Scale	HS	3.67 ± 2.76	8.50	0.182[a]	0.412
	SE	2.43 ± 1.92			
Multidimensional Assessment of Fatigue	HS	5.15 ± 4.93	13.00	0.484[a]	0.320
	SE	3.76 ± 4.02			

Abbreviations: BL, Baseline; HS, habitual sleep; SE, sleep extension.
Notes: *Hedges g comparisons between habitual sleep and sleep extension; [a]Related-Samples Wilcoxon Signed Rank Test.

Table 5 Physiological measures assessed from blood draw and blood pressure cuff.

Measure	Condition	*M* ± *SD*	Test Statistic	*p*-value	Hedges g*
CRP (log(10), mg/L)	HS	0.126 ± 0.364	0.394 (*t*)	0.349[a]	-0.030
	SE	0.134 ± 0.230			
Glucose (mg/dl)	HS	93.90 ± 5.88	-0.283 (*t*)	0.390[a]	-0.159
	SE	94.70 ± 6.73			
Trig (mg/dl)	HS	73.90 ± 14.27	-1.055 (*t*)	0.153[a]	-0.490
	SE	81.50 ± 17.75			
HDL (mg/dl)	HS	74.60 ± 20.67	-0.076 (*t*)	0.470[a]	-0.062
	SE	75.20 ± 13.80			
LDL (mg/dl)	HS	107.00 ± 48.24	-0.377 (*t*)	0.355[a]	-0.214
	SE	115.50 ± 52.39			
Systolic BP (mm Hg)	BL (1 & 2)	127.22 ± 13.85	0.040 (*F*)	0.844	-0.086
	HS	125.82 ± 13.49			
	SE	124.68 ± 11.89			
Diastolic BP (mm Hg)	BL (1&2)	73.50 ± 7.91	0.329 (*F*)	0.573	-0.246
	HS	72.19 ± 6.32			
	SE	70.53 ± 6.63			
Heart Rate	BL (1&2)	67.54 ± 8.86	0.031 (*F*)	0.861	0.076
	HS	68.89 ± 8.69			
	SE	69.67 ± 10.82			

Abbreviations: BP, blood pressure, BP; CRP, C-reactive protein; HS, habitual sleep; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SE, sleep extension; Trig, triglycerides.
Notes: *Hedges g reported for habitual sleep vs. sleep extension; [a]Independent Samples *t*-Test.

Psychological Variables

Psychological data are displayed in ►**Table 4**. No significant treatment differences were found for depression (Hamilton Depression Scale) or anxiety (Hamilton Anxiety Scale) as assessed by clinical psychology graduate students.

►**Table 4** displays the self-reported psychological data. Related-samples Wilcoxon Signed Rank tests were conducted on the subjective depression, mood, anxiety, sleepiness, and fatigue assessments since the data were not normally distributed. No significant treatment differences were found for depressed mood scores (BDI-II), Likert-rated mood scores, or anxiety scores (STAI). No significant treatment differences were found for sleepiness (ESS) or fatigue (MAF). Effect sizes indicated moderate effects for decreased subjective sleepiness after the sleep extension treatment compared to the habitual sleep treatment.

Physiological Data

►**Table 5** displays the physiological data. No significant treatment differences were found for measures of inflammation (log-transformed CRP), glucose, triglycerides, or cholesterol (HDL and LDL; see ►**Fig. 3**).

No significant treatment differences were found in systolic blood pressure, diastolic blood pressure, or heart rate.

Discussion

These relatively healthy, middle-aged-to-older average-duration sleepers were able to increase their TIB and TST sleep duration by an average of 81.63 ± 33.11 min and 66.33 ± 28.64 min, respectively, for 3 weeks in the sleep extension treatment, whereas TIB and TST in the habitual sleep treatment were similar to baseline levels. However, compared with the habitual sleep treatment, the sleep extension treatment did not significantly affect any of the dependent variables, and associated effect sizes were generally small.

Average adherence to the TIB extension was less than the target of 120 min. Only 2 of the 10 participants had TIB extension of $\geq$ 120 min, and 5 more participants had an average TIB increase of over 60 min.

Time in bed extension has been difficult to accomplish, as the average percentage of the targeted TIB extension in the present study (68%) was similar to that reported previously by our group[32] and other researchers (see Baron et al.[33] and Henst et al.[55] meta-analyses). In the weekly visits with research staff, participants were reminded to try to adhere to the TIB extension target, but since the ability to adhere to this target was a dependent variable, these reminders were mild. Anecdotally, participants indicated that they found it difficult to extend their TIB by the requested amount.

One distinct difference between experimental extension of sleep in healthy individuals and long sleepers is sleep pressure and perceived sleep need. It is possible that individuals who are obtaining healthy amounts of sleep do not experience the same level of sleep pressure, making it more difficult to achieve extra sleep when it is not needed. This particular sleep pressure factor makes experimental sleep extension much more complicated, as it becomes harder for participants to achieve their TST goals. A potentially more successful future approach may be to gradually increase TIB, perhaps based on the participants' sleep efficiency, analogous to the process of slightly and incrementally increasing TIB following the initial sleep restriction in cognitive behavioral treatment for insomnia.[56]

Other than the increase in TST, the TIB extension had minimal effect on sleep. Although wake after sleep onset and number of awakenings increased significantly in the sleep extension treatment compared to the habitual sleep

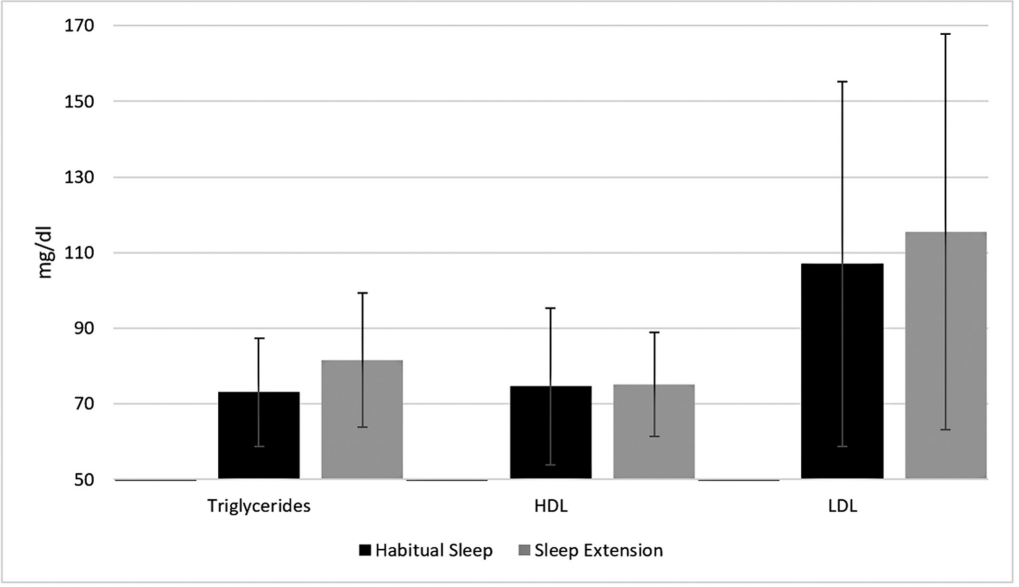

Fig. 3 Figure depicts lipid panel results; Hedges' g effect size (-0.490) indicated a moderate increase in triglycerides after the sleep extension condition. No significant differences seen in HDL (high-density lipoprotein) or LDL (low-density lipoprotein). Error bars represent standard deviation.

treatment, the amount of wake time after sleep onset (46.08 ± 12.60 and 59.87 ± 13.64 min, respectively) and number of awakenings (22.49 ± 4.68 and 27.72 ± 4.22, respectively) per hour of TIB were similar for the sleep extension and habitual sleep treatments. These data and the lack of significant difference in sleep efficiency between treatments is contrary to the notion that spending excessive TIB could lead to more sleep fragmentation, as observed, for example, in individuals with insomnia. Nonetheless, these results are consistent with previous work by our team and others (e.g., increased awakenings and wake after sleep onset[30,34]; sleep efficiency did not significantly change despite the increased wakefulness).[34]

Neither significant positive nor negative effects of sleep extension were found for any of the variables assessed. The lack of positive effects contrasts with recent studies of individuals with short sleep duration, which have found beneficial effects of sleep extension on cognition, mood, appetite, blood pressure, and prediabetes (see meta-analysis by Baron et al.[33]). However, key differences between the present study and these other studies include that participants in the present study had average age-related sleep duration and relatively good health. The majority of recent experimental sleep extension studies target short sleepers with various health problems, using sleep extension as an intervention to increase sleep duration.[33] However, for the present study, levels of blood pressure (systolic 125.82 ± 13.49 Hg, diastolic 72.19 ± 6.32 Hg), HDL (74.60 ± 20.67 mg/dl), sleepiness (3.67 ± 2.76), state anxiety (25.13 ± 5.50), and depressed mood (1.23 ± 1.13) are considered quite healthy for older adults, close to the ceiling/floor of optimal health such that significant improvements are less likely.[57]

Negative effects of sleep extension were regarded as more likely in the present study, based on our previous research[32] and other experimental sleep extension studies. For example, other studies have found impairments in performance on vigilance tasks after sleep extension (e.g., Taub et al., Taub & Berger[30,58]). The average extent and duration of TST increase (66.33 ± 28.64 min) might have been insufficient to produce the same level of grogginess and delay found in previous studies.

Although not significant, we anticipated that the triglycerides would increase after sleep extension compared to habitual sleep, which would have been consistent with other evidence that long sleep is associated with higher levels of triglycerides in women.[59] Total cholesterol levels were not significantly different between treatments, a result which was inconsistent with previous epidemiological studies examining long sleep in older adults.[60]

The current study had many significant strengths. To our knowledge, this is the first study to experimentally examine the physiological, cognitive, and psychological effects of chronic increases in sleep duration in middle-aged and older adults. The study was the first to examine sleep extension in healthy, older sleepers across such a comprehensive number of health factors through the use of a randomized, cross-over study design. The study demonstrated feasibility for middle-age and older adults to extend their sleep by extending their TIB attempting to sleep. The baseline differences in the various measures were not significant, suggesting there were not carry-over effects with a one-week washout period. These findings are important for future cross-over studies, as they provide evidence that a one-week recovery is sufficient to reduce or eliminate carry-over effects in an experimental sleep extension study.

The study also had several weaknesses. Although mitigated somewhat by a cross-over design, the sample size was small. Additionally, sleep extension was modest, and the duration of the intervention was short. More intense sleep extension (e.g., more than 2 hours) and intervention duration (e.g., more than 3 weeks) may produce different results. Participants were unable to extend their TIB or sleep duration to the extent that was planned, confirming the difficulty in voluntary sleep extension. Finally, due to the small number of participants, the impact of important social determinants (e.g., socioeconomic status) could not be evaluated and affects the generalizability of the results.

Future randomized controlled trials should examine extended sleep over a longer period of time (i.e., more than 3 weeks) and/or longer duration of TIB (i.e., more than 2 hours per night) in order to determine to what extent older adults with average sleep duration might experience negative effects following extension of sleep duration. Considering that sleep extension is difficult for healthy habitual sleepers, a greater degree of TIB extension might require extensive efforts involving gradual extension and greater troubleshooting to ensure adherence to extension protocols. However, it is acknowledged that even a longer-duration study may still not directly compare to individuals who sleep longer over several years or even decades, as increases in sleep would be acute and impact rapidly-reversible outcomes in experimental sleep extension conditions. If longer-duration studies are performed and significant effects are found, it would be imperative to conduct follow-ups (e.g., 3 months, 6 months, etc.) after study completion to determine if those significant changes are sustained over time. Finally, researchers conducting experimental sleep extension studies should be careful about comparing conclusions to epidemiological studies of long sleepers and increased health risks, as long sleepers tend to present this sleep phenotype over a significant amount of time.

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Conflict of Interests

The authors have no conflict of interests to declare.

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