



## Original Article

## Task switching in older adults with and without insomnia



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## ABSTRACT

**Background:** Task-switching deficits are common in older adults and in those with insomnia. Such deficits may be driven by difficulties with sleep continuity and dampened homeostatic sleep drive.

**Objective:** To identify the aspects of task switching affected by insomnia and its treatment, and to determine whether such effects are associated with sleep continuity and homeostatic sleep drive.

**Methods:** Polysomnographic sleep and task switching were tested in healthy older adults aged 60–93 years with insomnia ( $n = 48$ ) and normal sleeping controls ( $n = 51$ ). Assessments were repeated in the insomnia group after eight weeks of cognitive behavioral treatment for insomnia. Sleep measures included wake after sleep onset (WASO) and quantitative indices of homeostatic sleep drive (delta power during nonrapid eye movement [NREM] sleep and the ratio of delta power during the first and second NREM periods). A cued task-switching paradigm instructed participants to perform one of two tasks with varying preparatory cue-target intervals, manipulating task alternation, task repetition, and task preparation.

**Results:** The effect of preparatory cues on accuracy was diminished in the insomnia group compared with that in controls. Across the two groups, a stronger effect of preparatory cues was associated with a higher delta sleep ratio. Following insomnia treatment, task-repetition accuracy significantly improved. This improvement was associated with improvements in WASO. There were no group or treatment effects on response time or task alternation accuracy.

**Conclusions:** Effects of insomnia diagnosis and treatment apply to conditions that depend on the maintenance of a task set, rather than a domain general effect across task switching. Such effects are associated with homeostatic sleep drive and sleep continuity.

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## 1. Introduction

Deficits in executive functions are among the most prevalent cognitive changes observed with advancing age [8,47,48] and are commonly found in patients with insomnia [13,15,16,18]. For instance, a meta-analysis [16] showed negative effects of insomnia for executive functions such as maintaining and manipulating information in working memory and problem solving, though not for verbal fluency or cognitive flexibility; this suggests that there may be domain-specific effects of insomnia on executive function. Executive function deficits among older adults include difficulties selecting relevant and inhibiting irrelevant information, as well as maintaining and manipulating information in working memory

[20,22]. Task switching (a model paradigm of executive function), is often used to assess these age-related executive deficits [47]. Older adults commonly exhibit greater difficulty with managing two tasks than younger adults [30,47,49,52]. However, task-switching performance may be improved when task cues are provided, which allow for time to prepare for a given trial [43]. This “preparation effect” reflects controlled processes to maintain and reconfigure a task set in working memory. We previously reported that in older adults not selected on the basis of sleep disorders, higher wake after sleep onset (WASO) was associated with diminished preparation effects [51]. Thus, older adults with lower sleep continuity, such as those with insomnia, may be less likely to engage control processes to boost task-switching performance in the presence of preparatory cues.

Deficits in other tasks of executive function have been associated with insomnia, sleep continuity, and slow-wave

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sleep or homeostatic sleep drive. For instance, higher delta encephalographic (EEG) power (0.5–4.0 Hz), particularly within the first nonrapid eye movement (NREM) period, has been associated with better performance on tasks that tap into working memory, set shifting, and reasoning in older adults [3,50]. Delta power is often interpreted as an indicator of homeostatic sleep drive [19]; thus, less continuous sleep and lower homeostatic sleep drive in insomnia [27,31] may contribute to deficient executive control and in turn, affect task-switching performance.

Given the observed associations between these sleep measures and executive function, it is plausible that improvements in sleep continuity and homeostatic sleep drive could lead to improvements in executive processes critical for task switching in older adults. Cognitive behavioral treatment for insomnia involves restricting time-in-bed to increase sleep continuity [17,35,45] and sleep drive [6,14,40]. These treatment approaches commonly lead to significant reductions in WASO, clinically meaningful improvements or remission from insomnia [9,10,35,36], and in some reports, increased NREM delta power [11,26]. However, there is limited evidence (from a small number of studies) to suggest that these sleep improvements are also associated with improved executive function in older adults [1,32].

The present report was part of a larger study assessing sleep, arousal, and circadian rhythms among older adults with and without insomnia and, in the former group, before and after Cognitive Behavioral Treatment for Insomnia (CBTI). The parent study was designed to use CBTI as a treatment probe of neurobiological model of insomnia. The first goal of the parent study was to contrast older adults with and without insomnia, and the second goal was to determine whether improved insomnia is associated with changes in neurobiological indices of insomnia. For the present report, we aimed to identify the aspects of task switching affected by insomnia and its treatment, and whether such effects are associated with sleep continuity and homeostatic sleep drive. On the basis of our previous finding that older adults with lower sleep continuity exhibit weaker preparation effects on accuracy during task switching, we hypothesized that patients with insomnia would exhibit weaker preparation effects in accuracy than good sleeping controls. Further, we hypothesized that such deficits in preparation would be associated with reduced sleep continuity (eg, increased WASO) and homeostatic sleep drive (NREM delta power and the ratio of delta power between the first and second NREM periods). Finally, we tested whether CBTI improved performance and whether improvements in performance were associated with improvements in sleep continuity and homeostatic sleep drive. We hypothesized that preparation effects would be strengthened following CBTI and that such improvement would be associated with decreased WASO and increased whole night delta power and delta sleep ratio.

## 2. Materials and methods

### 2.1. Overview

Participants were adults with insomnia or normal sleeping controls recruited as part of the AgeWise program project (AG020677). The insomnia group participated in CBTI over eight weeks. Participants completed sleep and task-switching assessments at baseline (control and insomnia groups) and at follow-up at the end of the CBTI treatment (insomnia group only). Task-switching performance was assessed with a paradigm that included a block of trials in which participants were cued trial-by-trial to perform one of two tasks with a varying cue-target interval [51].

### 2.2. Participants

Participants were aged 60–93 years and were recruited from the greater Pittsburgh area. All participants provided informed consent as required by the University of Pittsburgh Institutional Review Board. Demographic and clinical characteristics for the control and insomnia groups are shown in Table 1. Participants were eligible to participate in the insomnia group if they met diagnostic criteria for general insomnia disorder according to DSM-IV/ICSD-2 and met a severity criteria including an Insomnia Severity Index (ISI) score  $\geq 10$ , a combined mean diary-assessed sleep latency + WASO  $> 40$  min, and sleep efficiency  $< 90\%$  [29]. Sleep efficiency was calculated with a denominator of “lights out” to final awakening and therefore required a higher threshold value than typically reported. This stringent definition of sleep efficiency was shown to have greater sensitivity and specificity in distinguishing older adults with insomnia from good sleeping controls than sleep efficiency based on time in bed to time out of bed [29]. To ensure that current sleep problems were specific to insomnia, participants who met criteria for significant sleep apnea (apnea–hypopnea index  $> 20$  events per hour) or restless legs syndrome (based on structured clinical interview) were excluded from the study. An apnea–hypopnea index of 20 was chosen to retain participants with mild-to-moderate sleep apnea, given the distribution of apnea–hypopnea indices among older adults [2,12] and evidence that the mild-to-moderate apnea–hypopnea index range has little effect on cognitive function [41,42]. Participants were eligible to participate in the control group if they did not meet criteria for a primary sleep disorder and had an ISI score  $< 10$ . Participants were not eligible for either group if they engaged in current nighttime shift work, had any untreated current severe psychiatric conditions, unstable medical conditions, or neurological disorders, including multiple sclerosis, stroke, Parkinson’s disease, Alzheimer’s disease, seizure disorder, delirium, dementia, or previous loss of consciousness  $> 24$  h. Participants with a history of psychiatric disorders were eligible except in the case of bipolar disorder or psychosis. Participants were not taking over-the-counter sleep aids, or other medications affecting sleep such as antidepressants, antipsychotic medications, anticonvulsants, steroids, or beta blockers during the study. Participants taking hypnotics at screening were given the option to enroll if they agreed to a medically supervised medication taper and discontinuation and were no longer taking hypnotics during the study. The complete list of medications reported at screening in each group is presented in the [Supplementary Materials](#) section. Participants were ineligible if they reported alcohol consumption  $> 14$  drinks per week or  $> 6$  at one sitting, or greater than three caffeine drinks per day (or 400 mg caffeine equivalent) reported in a sleep diary.

Three participants (two from the control group and one from the insomnia group) with task accuracy greater than three standard deviations below the mean were identified as outliers. These three participants were excluded from the present analyses to ensure that all participants included understood the task. One participant was excluded because of a tremor that interfered with their ability to perform the task with both hands. The present report included 51 participants in the control group and 48 participants in the insomnia group at baseline. All the 99 participants had usable task-switch data. Two insomnia participants did not complete the baseline polysomnography study and therefore had no data for polysomnographic sleep measures. One participant’s baseline polysomnographic record had excessive artifact that precluded spectral analysis within the first two NREM periods, and therefore, this participant was excluded from analyses with delta sleep ratio. Thirty-eight participants from the insomnia group returned following CBTI and completed the follow-up

**Table 1**

Demographic information and self-report and polysomnographic sleep and task-switching variables for controls at baseline (T1) and the insomnia group at baseline (T1) and at follow-up (T2). Standard deviation or percent of sample in parentheses. Switch cost units are proportion correct for accuracy and milliseconds for response time. Polysomnographic sleep variables are presented in minutes. Sleep latency complaint and WASO complaint reflect responses on the baseline survey of sleep. For T1 in the insomnia group, standard PSG variables n = 46; delta sleep ratio n = 45.

	Control T1 (n = 51)	Insomnia T1 (n = 48)	Insomnia T2 (n = 38)
Age	67.26 (5.16)	69.26 (7.87)	
No. of White/Caucasian	49 (96.1%)	42 (87.5%)	
No. of individuals with college degree or greater	38 (74.5%)	36 (75%)	
No. of females	33 (64.7%)	29 (60.4%)	
Number of medications	4.63 (2.75)	6.19 (3.76)	
Apnea–hypopnea index	4.87 (5.43)	6.23 (6.29)	
Sleep latency complaint	1 (2%)	34 (70.8%)	
WASO complaint	1 (2%)	36 (75%)	
Insomnia severity index	0.69 (1.33)	14.67 (4.56)	5.68 (4.48)
Total sleep time (min)	375.21 (46.90)	337.12 (59.82)	339.21 (46.934)
Sleep efficiency (%)	83.48 (8.29)	75.76 (11.19)	83.54 (10.61)
Sleep latency (min)	12.59 (11.75)	13.95 (13.96)	10.37 (6.70)
WASO (min)	62.28 (37.65)	95.16 (49.93)	57.42 (44.16)
N1%	7.28 (4.57)	8.50 (5.21)	7.28 (4.27)
N2%	58.83 (9.68)	58.60 (10.00)	58.73 (9.92)
N3%	11.47 (9.10)	9.78 (8.41)	10.06 (9.27)
Absolute delta power ( $\mu V^2/Hz$ )	24.93 (11.44)	22.95 (10.77)	24.60 (13.52)
Absolute delta sleep ratio (NREM 1/NREM 2)	1.30 (0.46)	1.17 (0.41)	1.08 (0.38)

polysomnographic and task-switching assessments. Reasons for attrition between baseline and follow-up included withdrawal from the study due to time commitment (n = 1), changes in availability (n = 1), being no longer interested (n = 2), poor compliance with CBTI (n = 3), and hospitalization the day of the follow-up (n = 1). Two participants were present for the follow-up session, but chose not to do follow-up assessments including the task-switching paradigm (n = 2). A final sample of 51 participants in the control group at baseline, 48 participants in the insomnia group at baseline, and 38 participants in the insomnia group at follow-up was available for data analysis.

### 2.3. Intervention

CBTI was conducted by two masters' level mental health clinicians. The treatment occurred over eight consecutive weeks with six 45–50 min in-person sessions and two telephone sessions, which included education and instruction on healthy sleep practices, sleep restriction, stimulus control, relaxation training, cognitive restructuring, and behavioral experiments regarding daytime fatigue management, and techniques to reduce worrying/thinking at night. Participants monitored their sleep with a sleep diary throughout the CBTI treatment.

### 2.4. Assessments and sleep measures

Laboratory-based polysomnography was recorded on a single night at the participants' diary-assessed habitual sleep times following an adaptation night. EEG was recorded from F3, F4, C3, and C4 electrodes, referenced to A1–A2. The sleep record was visually scored in 30-s epochs using the American Academy of Sleep Medicine criteria [23]. Spectral analysis was performed on F and C EEG channels with a 512-point fast Fourier transform using epochs scored as NREM sleep [46]. Following decimation, the EEG signal was parsed into 0.5 Hz bins over 4-s epochs and weighted by a Hamming window. Four-second epochs identified as movement artifact by an automated algorithm [7] and visual inspection were not included in the scored record. Spectral density data collapsed across the F3 and F4 channels within the 0.5–4 Hz (delta) frequency band (absolute power) were analyzed for the present report given the tendency for delta power to be the highest among

frontal channels. The present report focused on the following polysomnographic sleep variables: WASO and absolute NREM delta power assessed in two ways: whole night NREM delta power and the ratio of delta power in the first compared to the second NREM interval (delta sleep ratio). Rather than testing both absolute power and relative power across analyses, we focused on absolute power to avoid an inflated type I error rate. This measure was chosen because of observed greater interindividual variability of absolute power than of relative power, which we reasoned would be better suited for relating delta power to cognitive performance. However, to confirm consistency across absolute and relative measures (for significant results involving delta power), we confirmed whether such results could be replicated using relative power. Absolute power was calculated by first taking the average power within each 4-s epoch, and then taking the grand average across each 4-s epoch across each 0.5 Hz bin within the 0.5–4 Hz bandwidth for each channel. Relative power was calculated by dividing the area under the curve within the 0.5–4 Hz bandwidth by the area under the curve within the 0.5–32 Hz bandwidth. The delta sleep ratio was chosen to capture homeostatic sleep drive through the time course of delta power across the first 2 NREM intervals that show the highest delta power and the largest change across NREM intervals [28]. WASO and whole night NREM delta power were log transformed to correct for skewed distributions. Table 1 shows means for sleep variables at baseline and follow-up.

### 2.5. Cognitive task

The task-switching paradigm, which has been described elsewhere [51], was performed in the afternoon between 12:00 pm and 4:00 pm. Participants performed a practice block, single task block, and switching block at baseline and follow-up. In the switching block, the cue-target interval (preparation time) varied on a trial-by-trial basis. Participants were cued on each trial to perform one of two tasks that required judgments about a single-digit number presented on the screen. For one task, they judged whether the number was greater than or less than five (GL task). In the other task, they judged whether the number was odd or even (OE task). A circle preceding or accompanying the target number cued participants to perform the GL task. A square preceding or accompanying the target number cued participants to perform the OE task. The

preparation time was either zero (simultaneous cue and target), 750, or 1500 ms. Participants first completed the practice block comprised of 16 trials of each task, followed by two single-task blocks comprised of 32 trials each. Finally, participants completed the switching block. The switching block comprised a total of 96 trials: 16 trials of each cue type (GL and OE) for each of the three preparation conditions were presented randomly in the switching block with eight of each correct response type (greater than, less than, odd, even). Thus, successive trials within this block could be classified as “task repetition” or “task alternation” depending on whether successive trials used the same task. An example sequence of trials within the switching block is shown in Fig. 1.

This task-switching paradigm was used to assess accuracy and response time on task alternation and repetition trials as a function of preparation time. The 750 and 1500 ms preparation conditions were highly correlated across all conditions and time points in accuracy and response time. Thus, to facilitate interpretation, we collapsed across these two conditions. This approach was further justified by the fact that these two conditions represent an opportunity to prepare for the current task relative to the 0 ms preparation condition and by evidence that the greatest preparation benefits among older adults tend to occur within 750 ms of preparation time or less [25]. Accordingly, we report performance with and without time to prepare (preparation<sup>+</sup> and preparation<sup>-</sup>). Task-switching performance for each group and time point is shown in Table 2.

## 2.6. Statistical analyses

Primary analyses tested effects of patient group (control vs. insomnia) and time point (baseline vs follow-up) on switching and preparation for accuracy and response time. Follow-up analyses tested whether WASO, NREM delta power, and delta sleep ratio were associated with conditions found to have a significant effect of insomnia group or treatment. Ancillary analyses tested the effect of time point on each sleep variable of interest using paired t-tests to confirm which sleep variables showed significant changes after CBTI. Results are reported with an uncorrected alpha level of 0.05, as well as Bonferroni-corrected alpha levels. A corrected alpha level of 0.025 was used for each ANOVA to account for separate tests of accuracy and response time. A corrected alpha level of 0.017 was used for each follow-up correlation analysis and each ancillary

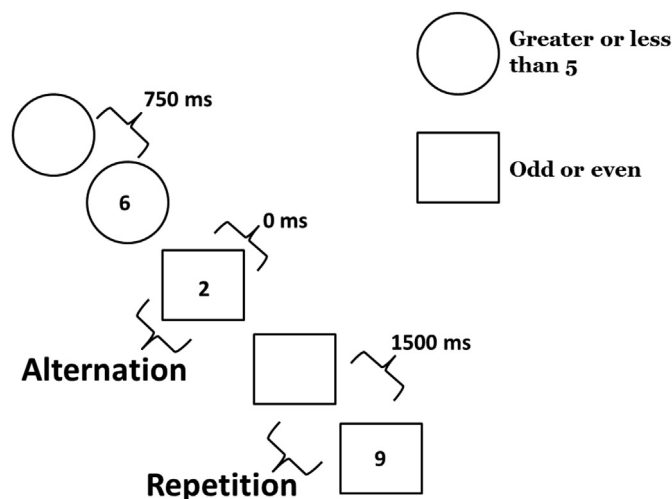


Fig. 1. Task-switching paradigm adapted from Refs. [51,52]: example of a sequence of trials including task alternations, task repetitions, and each preparation condition (0, 750, and 1500 ms preparation interval).

Table 2  
Task-switching performance for the control and insomnia groups.

	Accuracy (proportion correct)		Response time (ms)	
	Prep <sup>-</sup>	Prep <sup>+</sup>	Prep <sup>-</sup>	Prep <sup>+</sup>
<b>Controls</b>				
<b>Alternation</b>	0.61 (0.15)	0.80 (0.11)	867.67 (212.20)	616.82 (125.47)
<b>Repetition</b>	0.73 (0.15)	0.83 (0.12)	782.16 (152.93)	595.40 (108.94)
<b>Insomnia T1</b>				
	<b>Accuracy</b>		<b>Response time</b>	
	Prep <sup>-</sup>	Prep <sup>+</sup>	Prep <sup>-</sup>	Prep <sup>+</sup>
<b>Alternation</b>	0.61 (0.15)	0.76 (0.11)	814.71 (174.53)	614.95 (130.94)
<b>Repetition</b>	0.75 (0.13)	0.80 (0.15)	760.38 (144.57)	588.59 (101.77)
<b>Insomnia T2</b>				
	<b>Accuracy</b>		<b>Response time</b>	
	Prep <sup>-</sup>	Prep <sup>+</sup>	Prep <sup>-</sup>	Prep <sup>+</sup>
<b>Alternation</b>	0.58 (0.16)	0.80 (0.11)	816.44 (237.50)	621.49 (160.66)
<b>Repetition</b>	0.80 (0.11)	0.87 (0.09)	739.00 (158.58)	583.47 (120.16)

paired t-test to account for separate tests with each of the three sleep variables.

Effects of patient type and time point were tested with repeated-measures ANCOVA including interactions with switch condition (alternation vs. repetition) and preparation condition (preparation<sup>-</sup> or preparation<sup>+</sup>). Switch condition and preparation condition were included as repeated factors. For baseline analyses, patient type was included as a between-subjects factor. For time point analyses in the insomnia group, time point was included as a repeated measure.

Linear regression analyses across the control and insomnia groups were used to test the association between sleep variables of interest and task-switching conditions found to be affected by insomnia. Similarly, linear regression was used to test whether improvements in WASO, delta power, and the delta sleep ratio explained improvements following CBTI. Between baseline and follow-up, change scores were calculated to test the association between changes in sleep and changes in performance. Change scores were calculated for each sleep variable of interest; change scores in performance were only calculated for conditions that showed significant effects of time point, to focus on explaining significant effects of insomnia treatment with each sleep variable of interest.

## 2.7. Covariates

Age was included as a covariate in patient type analyses to account for slight age differences in the 2 groups. Age and sex were included as covariates in time point and regression analyses, to account variation associated with these demographic factors and sleep variables as well as changes in cognitive performance. Sensitivity analyses at baseline included the apnea–hypopnea index as a covariate to determine whether the effects of insomnia or relationships with sleep variables were affected by sleep apnea. Apnea–hypopnea index data were available for 44 participants in the control group and 46 participants in the insomnia group.

## 3. Results

### 3.1. Effects of patient type (control vs. insomnia)

Task-switching performance in each group is shown in Table 2. The main effects of patient type and interactions with switch condition and preparation condition for baseline performance are shown in Table 3. There were significant main effects of preparation and switch condition on accuracy and response time, as would be

expected with the current task. There was a significant patient type  $\times$  preparation interaction in accuracy (Fig. 2), significant at corrected  $\alpha = 0.025$ : the control group was disproportionately more accurate in the preparation<sup>+</sup> condition than the insomnia group, Cohen's  $d = 0.51$ . This effect remained significant after controlling for the apnea–hypopnea index,  $p = 0.027$ . For response time, there were no significant effects of patient type, nor did patient type interact significantly with switch condition (all  $p > 0.13$ ).

### 3.2. Baseline relationships with sleep variables

Linear regression analyses tested the associations between the preparation effect in accuracy and sleep variables across the control and insomnia groups. The delta sleep ratio was the only sleep variable significantly associated with the preparation effect,  $R^2$  change = 0.045,  $\beta = 0.22$ ,  $t = 2.13$ , uncorrected  $p = 0.035$  (Fig. 3), not significant at corrected  $\alpha = 0.017$ . This association replicated using relative power,  $R^2$  change = 0.044,  $\beta = 0.21$ ,  $t = 2.12$ ,  $p = 0.037$ . The significant uncorrected effect of patient type no longer reached significance after accounting for WASO ( $p = 0.053$ ) or the delta sleep ratio ( $p = 0.076$ ), but remained significant after accounting for delta power ( $p = 0.046$ ).

### 3.3. Effects of CBTI on sleep and task switching in insomnia

WASO showed a significant main effect of time point, indicating improvement following CBTI,  $t(37) = 6.30$ ,  $p < 0.001$ , significant at corrected  $\alpha = 0.017$ . There was no treatment-related change in absolute delta power,  $t(37) = -0.65$ ,  $p = 0.52$ , or delta sleep ratio,  $t(37) = 1.50$ ,  $p = 0.14$  (Table 1).

Task-switching performance at baseline and follow-up is shown in Table 2, and the effects of time point are shown in Table 4. There was a marginally significant main effect of time point on accuracy and a significant switch condition  $\times$  time point interaction, not significant at corrected  $\alpha = 0.025$ . This interaction reflected a greater improvement in repetition trial accuracy than in alternation trial accuracy (Fig. 4) across preparation conditions. Preparation condition did not significantly interact with time point, suggesting no significant improvement in the preparation effect following CBTI.

Linear regression analyses with change scores revealed that reductions in WASO were significantly associated with improvements in repetition trial accuracy,  $R^2$  change = 0.13,  $\beta = 0.372$ ,  $t = 2.51$ ,  $p = 0.017$ , marginally significant at corrected  $\alpha = 0.017$  as shown in Fig. 5. The association between increase in whole night delta power, the delta sleep ratio, and repetition accuracy improvement did not reach significance (delta power  $R^2$  change = 0.03,  $\beta = 0.191$ ,  $t = 1.14$ ,  $p = 0.262$ ; delta sleep ratio  $R^2$  change = 0.027,  $\beta = 0.17$ ,  $t = 1.06$ ,  $p = 0.295$ ).

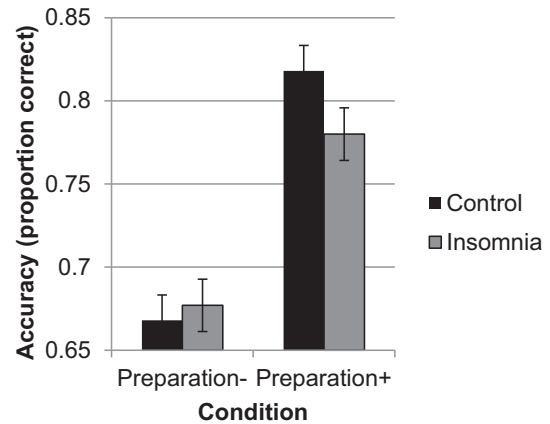


Fig. 2. Patient type  $\times$  preparation condition interaction. The preparation effect was greater in the control group than in the insomnia group, with controls exhibiting a greater increase in accuracy in the preparation<sup>+</sup> condition.

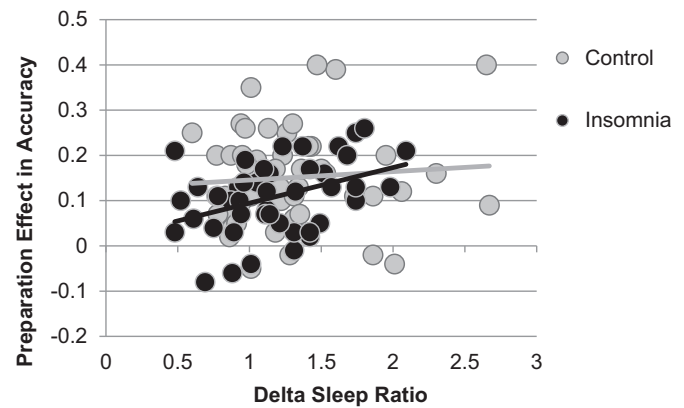


Fig. 3. Across the control and insomnia groups, the delta sleep ratio is associated with a preparation effect in accuracy. Preparation effect reflects the difference between preparation<sup>+</sup> and preparation<sup>-</sup> conditions collapsed across alternation and repetition trials.

## 4. Discussion

### 4.1. Effects of insomnia

There is growing consensus that age-related changes in sleep may contribute to deficits in goal-driven executive functions such as task switching. Here, we used a task-switching paradigm that varied preparation time to test whether insomnia and insomnia

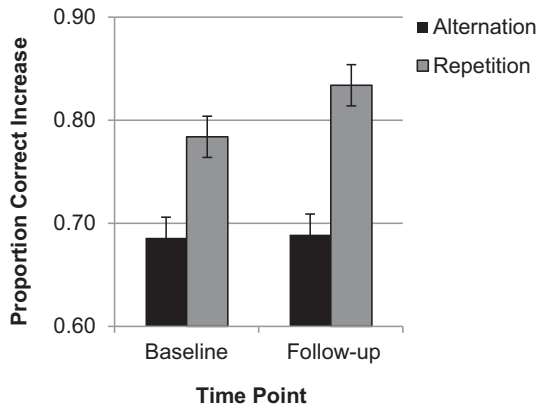
Table 3

Repeated-measures analysis of main effects of patient type and interactions with switching and preparation (prep). \* denotes significance uncorrected and corrected.

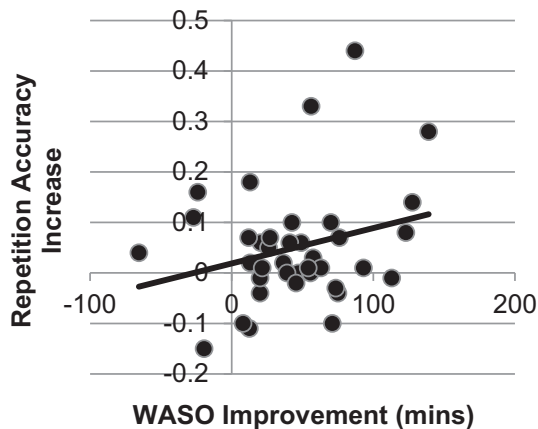
		F	df	p
Accuracy	Patient type	0.58	1, 96	0.450
	Prep	176.90	1, 96	<0.001*
	Switch	45.26	1, 96	<0.001*
	Patient type $\times$ prep	6.19	1, 96	0.015*
	Patient type $\times$ switch	0.21	1, 96	0.648
	Patient type $\times$ prep $\times$ switch	0.06	1, 96	0.802
Response time	Patient type	0.77	1, 96	0.383
	Prep	347.13	1, 96	<0.001*
	Switch	36.22	1, 96	<0.001*
	Patient type $\times$ prep	2.30	1, 96	0.133
	Patient type $\times$ switch	0.70	1, 96	0.404
	Patient type $\times$ prep $\times$ switch	1.85	1, 96	0.177

**Table 4**  
Repeated-measures analysis of main effects of time point on accuracy and response time and interactions with switch and preparation conditions in the insomnia group. \* denotes uncorrected significance, † denotes marginal uncorrected significance.

		F	df	p
Accuracy	Time	3.45	1, 35	0.072†
	Time × prep	1.80	1, 35	0.188
	Time × switch	4.80	1, 35	0.035*
	Time × prep × switch	1.66	1, 35	0.206
	Response time	Time	0.77	1, 35
Response time	Time × prep	1.42	1, 35	0.241
	Time × switch	0.61	1, 35	0.440
	Time × prep × switch	0.14	1, 35	0.712



**Fig. 4.** Time point × switch condition interaction (insomnia group). The improvement in accuracy was greater in the task repetition condition.



**Fig. 5.** In the insomnia group, improved repetition accuracy was associated with reduced WASO.

treatment affect specific aspects of task switching. Although overall task-switching performance was similar between the control and insomnia groups, preparation effects were diminished in the insomnia group relative to good sleepers. Participants with insomnia showed less of a benefit in accuracy from time to prepare than controls. This finding is consistent with our previous work in a separate sample [52] showing that greater WASO was associated with a diminished preparation effect in older adults who were not selected on the basis of sleep problems. This result suggests that normal sleepers more effectively made use of preparatory cues to adopt a task set in light of switching demands. Effective use of preparatory cues is accomplished through the engagement of

control processes during the cue-target interval to support the flexible and rapid task-set reconfiguration process [21]. This effect applied across switch conditions, and switch condition did not interact with the insomnia group, suggesting that it is not necessarily the “most challenging” task conditions that are most affected in insomnia.

#### 4.2. Baseline associations with sleep continuity and homeostatic sleep drive

Partially confirming our hypotheses, across patients and control participants, a stronger preparation effect was associated with a higher homeostatic sleep drive assessed with delta sleep ratio. The delta sleep ratio represents the ratio of delta power between the first 2 NREM periods, thus highlighting the diminishing homeostatic sleep drive between them. This finding is broadly consistent with previous studies [3,44,50]. For instance, Anderson and Horne found that slow-wave activity within the first NREM interval was associated with set shifting. The current finding extends the existing literature by demonstrating that the delta sleep ratio is associated with components of task switching that depend on the maintenance of a task set in working memory. Whether this association is driven by working memory has yet to be determined.

Contrary to our hypotheses, WASO and whole night delta power were not significantly associated with the preparation effect. Although there are a number of existing studies demonstrating a relationship between WASO and executive functions (including switching abilities [4,5,33,37,38,50–52]), we only tested baseline associations with the preparation effect in accuracy. Thus, these results simply demonstrate that WASO and whole night delta power are not associated with the preparation effect. In a prior study, we found that delta power across the whole night was associated with working memory and abstract reasoning [50]. However, that study did not examine the delta sleep ratio; thus, the strength of the association between the delta sleep ratio and the preparation effect or other aspects of task switching remains to be determined.

#### 4.3. Effects of time point and associations with sleep continuity and homeostatic sleep drive

A significant interaction between time point and switch condition reflected a greater improvement in repetition trial accuracy than in alternation trial accuracy from baseline to follow-up within the insomnia group. The improvement in repetition trial accuracy was associated with improvements in WASO. This is the first demonstration where improvements in a common symptom of insomnia following insomnia treatment are associated with significantly improved cognitive performance. In contrast to alternation accuracy, which reflects the inhibition and selection processes required for trial-by-trial switching, repetition accuracy reflects the working memory processes relevant to maintaining

two tasks throughout the task block. Similar to this finding, we previously found that actigraphically-measured WASO in older adults was associated with repetition trial response time when compared to single task performance, but not alternation trial performance [51]. These findings converge to suggest that sleep continuity may be important for working memory processes required for maintaining a task set [34,49]. In addition, given that working memory processes are considered responsible for both preparation effects and repetition trial performance, our results broadly suggest that working memory processes involved in task switching may drive associations with insomnia, WASO, and homeostatic sleep drive.

In contrast to our previous findings [50], changes in whole night delta power were not associated with changes in performance. One explanation for this discrepancy is that specific aspects of executive function not tested here are benefitted by whole night delta power. However, our previous study [50] involved a limited time frame with brief behavioral treatment for insomnia (a four-week follow-up as opposed to eight weeks here). Although neither study found a significant increase in delta power following behavioral insomnia treatment, delta power tends to remain stable from night to night [24], and therefore, it may be challenging to capture changes in delta power with a long follow-up interval, compared to what is observed with acute sleep restriction [39]. Further, single-channel EEG, as used here, may not be sensitive enough to capture important characteristics of delta power relevant to executive function. Future work using high-density EEG should examine more acute temporospatial effects of increased delta power on performance to evaluate the relevance of slow-wave sleep measures to executive deficits in older adults with sleep difficulties.

In the present study, the lack of consistency should be noted between baseline and follow-up in the cognitive conditions found that interact with patient type and time point. As a result, baseline association and change score association analyses cannot be directly compared and should be interpreted with caution. In line with our plan to test whether significant effects of patient type and time point were driven by sleep variables of interest, we did not test the association between baseline WASO and repetition trial accuracy. This approach allowed us to determine the sleep features that may explain effects of insomnia and treatment. However, it is possible that these sleep features are related to other aspects of task switching at baseline not tested here. Indeed, our previous research demonstrates that WASO is cross-sectionally associated with multiple domains of executive function in older adults including preparation effects [51]. Thus, it is important for future work to test differential effects of sleep continuity and homeostatic sleep drive on multiple domains of executive function in older adults.

#### 4.4. Limitations and future directions

Limitations of the current study are that the control group did not complete follow-up sleep or cognitive assessments, there was no separate group of untreated participants with insomnia to serve as an intervention control, and only a subset of participants with insomnia returned for follow-up assessments (38 of 48). These limitations make it difficult to confirm that sleep improvements, rather than practice or learning effects, caused changes in repetition trial accuracy in the insomnia group at follow-up. Learning curve effects may have influenced cognitive improvements and habituation to the sleep environment may have influenced sleep improvements. For instance, although WASO was similar between controls at baseline and participants with insomnia at follow-up (Table 1), some of this improvement in the insomnia group may be due to habituation to the sleep laboratory environment. Thus, although participants participated in an adaptation night before the

baseline visit, we cannot be certain that participants with insomnia improved to near-control level polysomnography-assessed WASO as a result of CBTI. Future studies should assess both groups at baseline and follow-up and test randomized control trials with an insomnia intervention control group to disentangle learning effects from treatment-related cognitive improvements and habituation effects from improved sleep.

Although the current task taps multiple dimensions of executive function, including preparation, working memory, and inhibition, only one task was assessed in this study. To better understand the domains of executive function affected by insomnia diagnosis and treatment, future work should assess cognition using multiple similarly demanding tasks. This will further address the domains of executive function that are diminished in older adults with insomnia, resulting in the effects on task switching found here.

It should be noted that the baseline association with the delta sleep ratio and interaction between time point and switch condition did not survive the Bonferroni correction for multiple comparisons. Thus, future work using larger sample sizes is needed to confirm the strength of these effects.

## 5. Conclusions

In a task-switching paradigm, insomnia and lower homeostatic sleep drive are associated with diminished preparation effects in accuracy. Following CBTI, improvements in sleep continuity are associated with improved accuracy on repetition trials that depend on the maintenance of a task set.

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## Conflict of interest

None.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: <http://dx.doi.org/10.1016/j.sleep.2016.09.002>.

## Appendix A. Supplementary data

Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.sleep.2016.09.002>.

## References

- [1] Altea E, Van Der Werf YD, Strijers RL, et al. Sleep loss affects vigilance: effects of chronic insomnia and sleep therapy. *J Sleep Res* 2008;17(3):335–43.
- [2] Ancoli-Israel S, Kripke DF, Klauber MR, et al. Sleep-disordered breathing in community-dwelling elderly. *Sleep* 1991;14(6):486.
- [3] Anderson C, Horne JA. Prefrontal cortex: links between low frequency delta EEG in sleep and neuropsychological performance in healthy, older people. *Psychophysiology* 2003;40(3):349–57.
- [4] Blackwell T, Yaffe K, Ancoli-Israel S, et al. Poor sleep is associated with impaired cognitive function in older women: the study of osteoporotic fractures. *J Gerontol Ser A* 2006;61(4):405–10.
- [5] Bonnet M. Cognitive effects of sleep and sleep fragmentation. *Sleep J Sleep Res Sleep Med* 1993;16:S65–7.
- [6] Brunner DP, Dijk D-J, Tobler I, et al. Effect of partial sleep deprivation on sleep stages and EEG power spectra: evidence for non-REM and REM sleep homeostasis. *Electroencephalogr Clin Neurophysiol* 1990;75(6):492–9.

- [7] Brunner D, Vasko R, Detka C, et al. Muscle artifacts in the sleep EEG: automated detection and effect on all-night EEG power spectra. *J Sleep Res* 1996;15(3):155–64.
- [8] Buckner RL. Memory and executive function in aging and AD: multiple factors that cause decline and reserve factors that compensate. *Neuron* 2004;44(1):195–208.
- [9] Buysse DJ. Sleep health: can we define it? Does it matter? *Sleep* 2013;37(1):9–17.
- [10] Buysse DJ, Germain A, Moul DE, et al. Efficacy of brief behavioral treatment for chronic insomnia in older adults. *Archives Intern Med* 2011;171(10):887–95.
- [11] Cervena K, Dauvilliers Y, Espa F, et al. Effect of cognitive behavioural therapy for insomnia on sleep architecture and sleep EEG power spectra in psychophysiological insomnia. *J Sleep Res* 2004;13(4):385–93.
- [12] Durán J, Esnaola S, Rubio R, et al. Obstructive sleep apnea–hypopnea and related clinical features in a population-based sample of subjects aged 30 to 70 yr. *Am J Respir Crit care Med* 2001;163(3):685–9.
- [13] Edinger JD, Means MK, Carney CE, et al. Psychomotor performance deficits and their relation to prior nights' sleep among individuals with primary insomnia. *Sleep* 2008;31(5):599.
- [14] Espie CA. Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorder in adults. *Annu Rev Psychol* 2002;53(1):215–43.
- [15] Espie CA, Kyle SD. Towards an improved neuropsychology of poor sleep? *Sleep* 2008;31(5):591–2.
- [16] Fortier-Brochu É, Beaulieu-Bonneau S, Ivers H, et al. Insomnia and daytime cognitive performance: a meta-analysis. *Sleep Med Rev* 2012;16(1):83–94.
- [17] Friedman L, Bliwise DL, Yesavage JA, et al. A preliminary study comparing sleep restriction and relaxation treatments for insomnia in older adults. *J Gerontol* 1991;46(1):P1–8.
- [18] Fulda S, Schulz H. Cognitive dysfunction in sleep disorders. *Sleep Med Rev* 2001;5(6):423–45.
- [19] Fuller PM, Gooley JJ, Saper CB. Neurobiology of the sleep-wake cycle: sleep architecture, circadian regulation, and regulatory feedback. *J Biol Rhythms* 2006;21(6):482–93.
- [20] Gazzaley A, Cooney JW, Rissman J, et al. Top-down suppression deficit underlies working memory impairment in normal aging. *Nat Neurosci* 2005;8(10):1298–300.
- [21] Gruber O, Karch S, Schlueter E, et al. Neural mechanisms of advance preparation in task switching. *Neuroimage* 2006;31(2):887–95.
- [22] Hasher L, Zacks RT. Working memory, comprehension, and aging: a review and a new view. In: Bower GH, editor. *The psychology of learning and motivation*. San Diego, CA: Academic Press; 1988.
- [23] Iber C, Ancoli-Israel S, Quan SF. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. *Am Acad Sleep Med* 2007.
- [24] Israel B, Buysse DJ, Krafty RT, et al. Short-term stability of sleep and heart rate variability in good sleepers and patients with insomnia: for some measures, one night is enough. *Sleep* 2012;35(9):1285.
- [25] Kray J. Task-set switching under cue-based versus memory-based switching conditions in younger and older adults. *Brain Res* 2006;1105(1):83–92. <http://dx.doi.org/10.1016/j.brainres.2005.11.016>.
- [26] Krystal AD, Edinger JD. Sleep EEG predictors and correlates of the response to cognitive behavioral therapy for insomnia. *Sleep* 2010;33(5):669.
- [27] Krystal AD, Edinger JD, Wohlgemuth WK, et al. NREM sleep EEG frequency spectral correlates of sleep complaints in primary insomnia subtypes. *Sleep* 2002;25(6):630–40.
- [28] Kupfer DJ, Frank E, McEachran AB, et al. Delta sleep ratio: a biological correlate of early recurrence in unipolar affective disorder. *Archives General Psychiatry* 1990;47(12):1100–5.
- [29] Levenson JC, Troxel WM, Begley A, et al. A quantitative approach to distinguishing older adults with insomnia from good sleeper controls. *J Clin Sleep Med* 2013;9(2):125–31.
- [30] Mayr U. Age differences in the selection of mental sets: the role of inhibition, stimulus ambiguity, and response-set overlap. *Psychol Aging* 2001;16(1):96–109.
- [31] Merica H, Blois R, Gaillard JM. Spectral characteristics of sleep EEG in chronic insomnia. *Eur J Neurosci* 1998;10(5):1826–34.
- [32] Miró E, Lupiáñez J, Martínez MP, et al. Cognitive-behavioral therapy for insomnia improves attentional function in fibromyalgia syndrome: a pilot, randomized controlled trial. *J Health Psychol* 2011;16(5):770–82.
- [33] Miyata S, Noda A, Iwamoto K, et al. Poor sleep quality impairs cognitive performance in older adults. *J Sleep Res* 2013;22(5):535–41.
- [34] Monsell S. Task switching. *Trends Cognitive Sci* 2003;7(3):134–40.
- [35] Morin CM, Bootzin RR, Buysse DJ, et al. Psychological and behavioral treatment of insomnia: update of the recent evidence (1998–2004). *Sleep-New York Then Westchest* 2006;29(11):1398.
- [36] Morin CM, Hauri PJ, Espie CA, et al. Nonpharmacologic treatment of chronic insomnia. *An American Academy of sleep medicine review*. *Sleep* 1999;22(8):1134–56.
- [37] Nair D, Zhang SX, Ramesh V, et al. Sleep fragmentation induces cognitive deficits via nicotinamide adenine dinucleotide phosphate oxidase–dependent pathways in mouse. *Am J Respir Crit care Med* 2011;184(11):1305–12.
- [38] Nebes RD, Buysse DJ, Halligan EM, et al. Self-reported sleep quality predicts poor cognitive performance in healthy older adults. *J Gerontol Ser B Psychol Sci Soc Sci* 2009;64(2):180–7.
- [39] Onen SH, Alloui A, Gross A, et al. The effects of total sleep deprivation, selective sleep interruption and sleep recovery on pain tolerance thresholds in healthy subjects. *J Sleep Res* 2001;10(1):35–42.
- [40] Pigeon WR, Perlis ML. Sleep homeostasis in primary insomnia. *Sleep Med Rev* 2006;10(4):247–54.
- [41] Quan SF, Budhiraja R, Batool-Anwar S, et al. Lack of impact of mild obstructive sleep apnea on sleepiness, mood and quality of life. *Southwest J Pulm Crit Care* 2014;9(1):44.
- [42] Quan SF, Wright R, Baldwin CM, et al. Obstructive sleep apnea–hypopnea and neurocognitive functioning in the Sleep Heart Health Study. *Sleep Med* 2006;7(6):498–507.
- [43] Schapkin SA, Gajewski PD, Freude G. Age differences in memory-based task switching with and without cues: an ERP study. *J Psychophysiol* 2014;28(3):187.
- [44] Scullin MK. Sleep, memory, and aging: the link between slow-wave sleep and episodic memory changes from younger to older adults. *Psychol aging* 2013;28(1):105.
- [45] Spielman AJ, Saskin P, Thorpy MJ. Treatment of chronic insomnia by restriction of time in bed. *Sleep J Sleep Res Sleep Med* 1987;10:45–56.
- [46] Vasko RC, Brunner DP, Doman JP, et al. Power spectral analysis of EEG in a multiple-bedroom, multiple-polygraph sleep laboratory. *Int J Med Inform* 1997;46(3):175–84.
- [47] Verhaeghen P. Aging and executive control: reports of a demise greatly exaggerated. *Curr Dir Psychol Sci* 2011;20(3):174–80.
- [48] Verhaeghen P, Cerella J. Aging, executive control, and attention: a review of meta-analyses. *Neurosci Biobehav Rev* 2002;26(7):849–57.
- [49] Wasylshyn C, Verhaeghen P, Sliwinski MJ. Aging and task switching: a meta-analysis. *Psychol Aging* 2011;26(1):15–20.
- [50] Wilckens KA, Hall MH, Nebes RD, et al. Changes in cognitive performance are associated with changes in sleep in older adults with insomnia. *Behav Sleep Med* 2016;14:1–16. <http://dx.doi.org/10.1080/15402002.2014.1002034>.
- [51] Wilckens KA, Woo SG, Erickson KI, et al. Sleep continuity and total sleep time are associated with task-switching and preparation in young and older adults. *J Sleep Res* 2014;23(5):508–16.
- [52] Wilckens KA, Woo SG, Kirk AR, et al. The role of sleep continuity and total sleep time in executive function across the adult lifespan. *Psychol Aging* 2014;29(3).