Sleep and cognition in older adults: Does depression matter? An actigraphy and polysomnography study

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Abstract

The impact of sleep on cognition is well recognised, although research in older adults is lacking, as are investigations into how depression might impact on this relationship. This study assessed the relationship between sleep (assessed with actigraphy and polysomnography) and cognitive performance in older adults (50–78 years) with (n = 10) and without (n = 33) a current diagnosis of Major Depressive Disorder (MDD). There were associations between sleep-wake patterns and cognition across the whole sample: time spent awake after sleep onset (WASO) was associated with speed of responding on vigilance tasks and accuracy on delayed recall tasks; sleep efficiency and total sleep time (at trend-level) were linked to working memory accuracy. Examination of the impact of depression on the relationship between sleep and cognition was explored using moderation analysis. Current depression was a significant moderator of the relationship between sleep-wake patterns (assessed with actigraphy), but not sleep architecture (assessed with PSG), and cognition. That is, there was a different pattern of association of sleep-wake patterns and cognitive performance depending on depression status. Specifically, in the MDD group, sleep-wake patterns were linked to speed of processing on cognitive tasks, whereas in the not currently depressed group, sleep-wake patterns were linked to performance accuracy. In conclusion, it was found that depression impacted on the relationship between sleep and cognition but questions remain regarding the nature of the relationship between sleep and cognition in older adults. Given that sleep problems are potentially modifiable risk factors for cognitive impairment, these findings point to the importance of assessing sleep in both depressed and healthy older adults.

Keywords: sleep; sleep-wake patterns; depression; cognition; actigraphy; older adults; polysomnography; ageing


**Introduction**

The link between sleep and cognition is now well recognised. However, fewer studies have been conducted in older adults (without sleep disorders) relative to younger adults. Understanding the importance of sleep in cognition in older adults is especially challenging because, ageing is linked to changes both in cognition – including a decline in attention, memory and executive functioning (Rabbitt & Lowe, 2000) – and in sleep (D’Ambrosio & Redline, 2014). Changes in the latter include sleep-wake cycle changes, such as increased sleep fragmentation, and sleep architecture changes (Ohayon et al., 2004; Stanley, 2005). This has led to questions regarding the nature of the relationship between sleep and cognition in the context of ageing.

One theory suggests that age-related cognitive deficits could be, at least in part, due to poor sleep (e.g., Altena et al., 2010). Evidence mostly derives from subjective studies, and from studies using actigraphy to assess sleep-wake patterns, although there is a paucity of evidence using polysomnography (PSG). Using questionnaires, sleep disturbances in older adults have been linked to poorer cognitive performance on tasks of executive functioning (e.g., Nebes et al., 2009), memory (e.g., Schmutte et al., 2007), and attention (e.g., Amer et al., 2013). Actigraphy, also commonly used, is a noninvasive method of sleep assessment, and has been recommended by the American Academy of Sleep Medicine as an accurate assessment of sleep-wake patterns (Morgenthaler et al., 2007).

Actigraphy studies in older adults are largely consistent in showing a link between sleep-wake patterns and executive functioning (Blackwell et al., 2006; Haimov et al., 2008), working memory (Miyata et al., 2013) and sustained attention (Haimov et al., 2008). Sleep duration (< 5 hours) has also been linked to simple attention and short-term memory (e.g., Miyata et al., 2013), although this is not the case in all studies (e.g., Blackwell et al., 2011; Blackwell et al., 2006), which might suggest that sleep quality, rather than quantity, is more important for cognition.

Studies using PSG, although less common, provide evidence of a link between specific stages of sleep to cognition in older adults, but the evidence is largely contradictory. For example, some studies report a positive association between SWS and memory and executive functioning performance (e.g., Anderson & Horne, 2003; Backhaus et al., 2007; Mander et al., 2013; Van der Werf et al., 2009; Van der Werf et al., 2011), while other studies report no such association (e.g., Scullin, 2013).

The reasons for the negative findings are not entirely clear. One view regarding how sleep relates to cognition is a neurodevelopmental account. This view observes that qualitative and quantitative aspects of sleep change substantially during development and that these changes parallel the learning and cognitive needs across the lifespan (Geiger et al., 2010). The assumption, therefore, is that of a close association between sleep stages (REM, SWS) and cognition and brain morphology. Evidence derives from early-life studies, where sleep duration and REM sleep is significantly increased in childhood, perhaps linked to children’s significant learning needs (e.g., Mirmiran & Van Someren, 1993). Studies in younger adults are also supportive of a continued relationship of sleep stages and cognition (e.g., Backhaus et al., 2007).

However, studies in older adults are less consistent. We know that there are significant changes in sleep architecture associated with ageing, including reductions in SWS and REM sleep (Stanley, 2005), which perhaps reflect a decreased need for learning in older adults compared to
children. But nevertheless, according to the neuro-developmental account, the tight relationship between sleep architecture and cognition should remain if human cognitive needs are so entirely dependent on sleep physiology. One prediction, based on this view, therefore, is that age-related decreases in SWS and REM are associated with corresponding decreases in cognitive performance. A suggested mechanism of action is that decreases in these sleep stages reduce Sleep Dependent Memory Consolidation (SDMC) and/or compromise the integrity of the hippocampus or prefrontal cortex (PFC; Scullin, 2013).

There is indeed some support for this view. As mentioned above, some studies indicate a role of SWS in episodic memory and executive functioning in older adults. For example, Van der Werf et al. (2009) showed that experimental reduction of SWS affected activity in the hippocampus and associated declarative memory encoding the next day. While the current study investigated the impact of sleep on cognitive performance within one testing session (“waking cognitive performance”), evidence also derives from overnight consolidation paradigms. Backhaus et al. (2007), for example, revealed that although SWS decreased with age, greater SWS was none-the-less significantly associated with better overnight episodic memory consolidation in the older adult group. SWS has also been linked to executive functioning. Lafortune et al. (2014) implicated SWS in “waking” performance on a verbal fluency task. Furthermore, another study linked experimental SWS reduction to more lapses on vigilance tasks in healthy older adults the next day (Van der Werf et al., 2011).

Rapid Eye Movement (REM) sleep has also been implicated in cognition, usually in the context of procedural or emotional memory (Maquet, 2001). Two recent studies, however, point to a possible role of REM sleep in non-emotional declarative memory in older adults. For example, a consolidation study implicated REM sleep in overnight improvement on a word recall task (Schredl et al., 2001), and another study linked longer REM sleep to better “waking cognitive performance” on a verbal learning task (Lafortune et al., 2014).

A contrasting view suggests that the nature of the relationship between sleep and cognition may change with age. For example, the benefit of sleep on cognition has been shown to be reduced in older adults (e.g., Spencer et al., 2007), and older adults appear more resilient to sleep deprivation than younger adults (e.g., Stenuit & Kerkhofs, 2005). Some studies also suggest that sleep stages important in younger adults may no longer be important in older adults (e.g., Hornung, Regen, Danker-Hopfe, Schredl, & Heuser, 2007).

Taken together, these findings suggest that sleep may no longer be as closely related to cognition in ageing, which Spiegel et al. (1986) referred to as “functional-dissociation”. The mechanisms behind the weakening sleep-cognition relationship seen in ageing, however, are not yet understood. Scullin (2013) describes an interesting study in younger adults, which found that exposure during SWS to an odor, which had been presented as context during prior learning, caused better retention of the associated memory (Rasch, Büchel, Gais, & Born, 2007). Scullin suggests that method might be useful in encouraging older adults to consolidate information during sleep, and goes on to suggest that if this intervention were successful in older adults, it would imply that the neural circuits and the ability to consolidate information remain intact in ageing. On the other hand, if the manipulation did not result in improved consolidation, this might indicate that the ability to consolidate memories in ageing has diminished, which could be the result of age-related changes in brain structure and
function (e.g., Grady, 2006), and/or neurochemical changes (e.g., Bäckman, Nyberg, Lindenberger, Li, & Farde, 2006), or other unknown age-related mechanisms. Hence, there is a need for more research into why some studies have found that the sleep–cognition association weakens in ageing.

Evidence cited in support of the “functional-dissociation” account includes studies showing a different pattern of cognitive performance in older adults relative to younger adults. For example, Scullin (2013) found that sleep benefited word-pair learning in a group of younger adults, which was correlated with greater SWS. In contrast, the opposite pattern was found in the older adults, in whom greater SWS was linked to poorer memory. Hence, results indicate that the relationship between episodic memory and SWS changed with age. Furthermore, in contrast to research in younger adults where a close association between SWS and vigilance was indicated (Jurado et al., 1989), research in healthy older adults suggested an erosion of this relationship (Crenshaw & Edinger, 1999).

Tentative evidence for the “functional dissociation” account also derives from studies showing no link between REM sleep and cognition in older adults. For example, a recent “waking” study found that REM sleep was not related to executive functioning performance in older adults (Lafortune et al., 2014), which stands in contrast to consolidation studies in younger adults, which have implicated REM in improvement on problem-solving tasks after a period of sleep (e.g., Cai et al., 2009; Walker et al., 2002). Thus, this may suggest a changing relationship between REM sleep and cognition in ageing.

Therefore, while some studies support the neurodevelopment approach suggesting a continued relationship of sleep architecture and cognition in older adults, others suggest that this relationship changes and even disappears with age (the “functional dissociation” account).

One factor that may influence the relationship between sleep and cognition differentially with age is depression. Surprisingly few studies in older adults have considered the role of depression in the association between sleep and cognition, despite evidence that depression is common in older adults (Garcia, 2008); poor sleep and depression are closely related (Sbarra & Allen, 2009); and sleep problems and depression are independently associated with cognitive impairment (Koehler et al., 2010; Naismith et al., 2009; Riemann et al., 2001; Smagula et al., 2013; Snyder, 2013).

Existing studies in older adults, however, have either assessed for subclinical levels of depression (e.g., Blackwell et al., 2011; Blackwell et al., 2006; Nebes et al., 2009; Schmutte et al., 2007), and/or have excluded older adults with clinical levels of depression (e.g., Bastien et al., 2003; Nebes et al., 2009; Sutter et al., 2012; Van der Werf et al., 2011; Vignola et al., 2000; Yu et al., 2016). A cross-sectional study using questionnaires in 107 older adults (61+ years) with varying levels of subclinical depressive symptoms on the Geriatric Depression Scale (GDS; Yesavage et al., 1983) found that poorer global sleep quality on the PSQI was linked to decreased performance on tasks of executive functioning (reasoning, semantic fluency and shifting), but only in individuals with higher levels of depressive symptoms (Sutter et al., 2012). This finding suggests that the profile of sleep-associated cognitive performance may be different depending on levels of depression.

A more recent study by Yu and colleagues (2016) found the opposite. That is, better global sleep quality on the PSQI was linked to increased performance on tasks of delayed memory, language, and general...
cognition, but only in individuals with lower levels of depressive symptoms. Despite a difference in the direction of the interaction effects, which could be due to differences in the cognitive tasks used and covariates, these important studies point to a possible moderating role of depression. This may explain some of the inconsistent research findings regarding the link between sleep and cognition since the majority of previous studies did not consider depression interaction effects.

Results have yet to be replicated using objective measures of sleep. Very few studies have assessed sleep and cognition using objective measures of sleep in older adults with depression. Naismith et al. (2011) assessed sleep-wake patterns using actigraphy in older adults with a lifetime history of clinical depression \( n = 44 \) compared to healthy controls \( n = 22 \). In the depressed individuals (currently depressed and individuals in remission), greater WASO was linked to poorer memory (verbal learning on the Rey Auditory Verbal Learning Test; RAVLT; Spreen & Strauss, 1998) and poorer executive functioning (response inhibition on the Stroop test, Delis Kaplan Executive Functioning System; DKEFS; Delis, Kaplan, & Kramer, 2001); and poorer sleep efficiency was linked to poorer memory (verbal learning on the RAVLT, and visual learning on the Ray Complex Figure Test; RCFT; Spreen & Strauss, 1998) and poorer executive functioning (response inhibition on the Stroop test, and problem-solving on the Sorting test; DKEFS).

This suggests that sleep problems are related to cognitive impairment in depression. Although the causal mechanisms remain to be understood, depression and poor sleep have been found to impact on overlapping brain areas (e.g., frontal lobes) and their associated cognitive functions (Naismith et al., 2011). While this is an important study, Naismith et al. (2011) included low levels of depression; did not separate current from past depression; and did not compare the cognitive performance of the depressed group with the control group. Furthermore, the study did not specifically examine the potential moderating effect of depression on the relationship between sleep and cognition. In other words, the authors did not examine whether the association of sleep and cognition differed as a function of depression, an important aim of the current study.

This study conducted a comprehensive investigation into the association of sleep-wake patterns and sleep architecture and daytime cognitive performance in older adults. We were particularly interested in whether this pattern of association differed between currently depressed (MDD) versus not currently depressed older adults. Given that the effect of sleep on arousal levels and attention has been suggested as a mechanism of sleep-related deficits in higher order cognitive processes (Doran et al., 2001), we controlled for vigilance in all analyses of memory and executive functioning. We also controlled for sleep-disordered breathing (SDB; Bucks et al., 2013; Malhotra & White, 2002) and the associated effects of hypoxia (Beebe & Gozal, 2002), gender (Voderholzer et al., 2003; Weiss, Kemmler, Deisenhammer, Fleischhacker, & Delazer, 2003), age (Ancoli-Israel, 2005; Bruce & Aloia, 2006), and “morningness-eveningness” preference (Ramirez et al., 2006), given that these variables can affect sleep and/or cognition.

**Method**

**Participants**

Forty-six participants took part in this study. After excluding three people due to a history of traumatic brain injury or bipolar depression, data were analysed for 43 participants (12 male, 31 female) aged 50–
78 years (M = 60.67, SD = 6.46). While the definitions of what constitutes ‘older adults’ vary, in the current study these terms pertain to individuals of 50 years of age and over. This is to ensure our findings are comparable to previous research in this area (e.g., Gildner, Liebert, Kowal, Chatterji, & Snodgrass, 2014: >50 years of age, M = 66.76; Xu et al., 2011: >50 years of age, M = 62).

Recruitment of the not currently depressed individuals (n = 33) was conducted via advertisements in community centres and mail-outs to volunteer groups. Participants with a present diagnosis of MDD (n = 10) were either outpatients from the North Metropolitan Adult Mental Health Older Adults, Osborne Park Hospital, Perth, Western Australia, or individuals recruited via advertising from the community.

Inclusion criteria for the depressed group included age (50–80 years) and a current clinical diagnosis of unipolar depression provided by a mental health professional. Exclusion criteria for all participants included: chronic infectious illness; neurological or neurodegenerative conditions; history of moderate or severe traumatic brain injury; previous loss of consciousness > 30 minutes duration; treatment for substance abuse; or any other psychiatric disorder. Ethics committee approval was granted by both the North Metropolitan Mental Health Service and the University of Western Australia Human Research Ethics Committees.

All individuals with current MDD had received a clinical diagnosis by a mental health clinician, and had their diagnosis confirmed with a research interview with the MINI (Sheehan et al., 1998) and PHQ-9 score of ≥10 (Kroenke et al., 2001). Morning cortisol levels were also collected as a potential biological index of depression. While elevated morning cortisol cannot be considered a “pure” measure of depression as it is linked to other factors such as alcohol consumption (Badrick et al., 2008), it is associated with hypothalamic pituitary adrenal axis activity, which is regarded as important in the pathophysiology of depression (Riemann et al., 2001).

Procedure

All assessments were conducted in the participant’s home. The first visit included completion of consent forms and questionnaires; clinical and sleep interviews; and the first overnight sleep study (PSG). Participants were also given an actiwatch and sleep diary. One week later, cognitive assessments were administered and the second night of PSG was conducted. The sleep studies were separated by one week in order to reduce first-night effects due to unfamiliarity with equipment (Agnew et al., 1966). Morning cortisol samples were collected on the morning of the last visit.

Sleep assessments

Sleep electrophysiology. Home-polysomnography (PSG) was conducted using the Compumedics “Somté” device. It includes electroencephalogram (EEG), electrooculogram (EOG), electromyogram (EMG), airflow, body position, thoracic and respiratory belts, and blood oxygen saturation. Data were analysed by a Senior Sleep Technologist (J. Maul) and checked by a second Sleep Technologist (A. Mellor). Only data from the second sleep study are reported. Outcome variables included the percentage of REM and SWS sleep given the implication of these major sleep stages in cognition. In order to assess for sleep-disordered breathing (SDB), analyses included the Respiratory Disturbance Index (RDI), and the Oxygen Desaturation Index (ODI-3; no. times per hour that there is a decrease of ≥3% in blood oxygen
saturation). This was to ensure that any effects of sleep on cognition were not due to SDB-associated hypoxemia.

**Actigraphy.** Participants wore an actiwatch, a wrist-worn device that non-invasively monitors sleep-wake cycles, for two weeks to ensure seven nights of viable data were collected. Output measures were total sleep time (minutes), sleep efficiency (time spent asleep as a proportion of time spent in bed, expressed as a percentage), sleep latency (minutes), and time spent awake after sleep onset (WASO; minutes). Participants kept a sleep diary to enable interpretation of actiwatch data. Actigraphic data were checked against the sleep diary by two researchers to ensure concordance (A. Mellor and F. Waters). The Actiwatch Spectrum (MiniMitter Philips) was used in this study. Seven days of actigraphy recording has been shown to be sufficient to obtain stable measures of sleep-wake patterns (Knutson et al., 2007; Tworoger et al., 2005).

Participants completed a daily sleep diary to record details such as sleep and wake times, whether the day was a work day, if they took any naps, and if the watch were removed for any period. This information was used to cross-validate and edit the actigraphy data as needed. Each actigram was visually inspected and compared to the sleep diary in order to identify any major discrepancies. Time in bed was adjusted by the scorer in cases where there was a discrepancy of greater than one hour between the diary and actigraph, so it was consistent with the sleep diary.

**Salivary cortisol assessment.** Three samples were taken: immediately after waking, wake+15 minutes, and wake+30 minutes. Participants were asked to collect their saliva in small vials and store in the freezer until collection. For each sample, salivary cortisol was assessed in duplicates using a commercial enzyme immunoassay (Salimetrics, LCC). All samples from the same participant were assessed in the same batch. The inter-assay coefficient of variation was 1.63% and 9.61% for high and low quality control standards respectively, indicating consistency between runs.

**Materials**

A demographics questionnaire asked about age, occupation, medical history (including respiratory problems, chronic pain, sleep disorders), and medication use.

The Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001) has excellent reliability ($\alpha = .94$ in the current study) and has diagnostic value as items map onto the DSM-IV criteria for diagnosis of depression (Kroenke et al., 2001). A score of $\geq 10$ (for moderate depression) is commonly used to identify clinical levels of depression (Kroenke et al., 2001).

The Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998) was used by the investigator (A. Mellor) to exclude co-morbid psychiatric disorders and to confirm diagnosis of depression.

The Morningness-Eveningness Questionnaire (MEQ; Horne & Ostberg, 1976) was used to assess individual chronotype. Studies using the MEQ report good internal consistency (Adan & Natale, 2002; $\alpha = .82$ in this study).

**Cognitive Assessments**

The National Adult Reading Test-Revised (NART-R; Nelson & Willison, 1991) is a valid index of premorbid intellectual ability that measures the ability to read aloud a list of 50 irregular English nouns. Performance on the NART correlates highly with measures of general intelligence (Crawford, Stewart, Cochrane, Parker, & Besson, 1989). We estimated IQ for each participant using a regression equation (Nelson &

The CogState is a computerized test battery used to assess cognitive functioning (http://www.cogstate.com.au) and has been described in detail elsewhere (Maruff et al., 2009). It has well-established validity (e.g., Weaver Cargin, Collie, Masters, & Maruff, 2008), is suitable in older populations (Collie et al., 2003) and is sensitive to subtle cognitive impairment (Falleti et al., 2003). It assesses psychomotor speed, long-term memory (immediate and delayed), working memory and other components of executive functioning. The CogState battery was administered using a laptop computer. Tasks were selected in order to measure a range of cognitive functions. The primary outcome measures recommended by the CogState manual were used for analyses. Additional variables were also analysed to derive measures of accuracy and speed of responding measures for each task. In this study, participants completed the following tasks:

Detection Task – A measure of simple reaction time, speed of processing and psychomotor function (Davidson et al., 2011; Olver, Ignatiadis, Maruff, Burrows, & Norman, 2008; Pietrzak, Snyder, & Maruff, 2010). Participants are instructed to respond as quickly as possible when a playing-card presented on screen turns face up (Primary outcome measure = speed of responding: mean of the log10 transformed reaction time in ms for correct responses; log10, ms. Additional outcome variable = accuracy).

Identification Task – A measure of choice reaction time, attention and vigilance (Davidson et al., 2011; Hammers et al., 2012; Lim, Ellis, Pietrzak, Ames, Darby, Harrington, Martins, Masters, Rowe, Savage, Szoeke, Villemagne, & Maruff, 2012; Maruff et al., 2009; Olver et al., 2008). Participants must decide whether the playing card is red or black. (Primary outcome measure = speed of responding; log10, ms. Additional outcome variable = accuracy).

One Back Memory Task – Widely agreed as a working memory task (Biggs et al., 2011; Davidson et al., 2011; Maruff et al., 2009; Yoshida et al., 2011). Participants are required to identify whether the current card was the same as the previously presented card (Primary outcome variable = accuracy. Additional outcome variable = speed of responding; log10, ms).

Groton Maze Learning Task – This task requires participants to navigate their way through a 10x10 grid of tiles on a computer screen using the mouse to find a hidden pathway using trial and error-based feedback. While this task taps a diverse range of functions including spatial learning and error monitoring (Pietrzak et al., 2008), rule use and spatial memory (Pietrzak et al., 2010) that rely on attention, processing speed and decision-making (Darby & Walsh, 2005), it is widely used as a measure of spatial problem-solving and therefore, executive functioning (Cockayne et al., 2011; Davidson et al., 2011; Olver et al., 2008; Yoshida et al., 2011). There are a total of five trials. (Primary outcome measure = total number of errors. Additional outcome variable = speed of responding; moves per second). There is also a delayed recall version of this task (recall is tested approximately 20 minutes later, during which participants complete the remaining cognitive tasks).

International Shopping List – A verbal learning task (Davidson et al., 2011; Thompson et al., 2011; Yoshida et al., 2011). Participants listen to a list of items (n = 12) on a shopping list and are asked to recall them aloud. There are three learning rounds. (Primary outcome measure = total number of correct responses across three consecutive trials. No speed of responding measure). There is also a delayed recall
version of this task (recall is tested approximately 30 minutes later, during which participants complete the remaining cognitive tasks).

Continuous Paired Learning – This task assesses paired-associate learning, a form of visual learning and memory (Davidson et al., 2011). In the learning phase, participants learn the location of objects on screen. In the test phase, they must find the correct location of the object across five rounds. (Primary outcome measure = total number of errors. Additional outcome measure = speed of responding; log10, ms).

The Hayling Sentence Completion Test (HSCT; Burgess & Shallice, 1997) – This is a widely used measure of executive functioning, comprising two sets of 15 sentences, each missing the last word. In section 1, participants are asked to complete the sentence with a logical word as fast as possible. In section 2, they are asked to complete the sentence with an incongruent word as fast as possible. There are three possible outcome measures: the sum of the response latencies in section 1, the number of intrusions (errors) in section 2, and the time taken to respond in section 2. In this study, we analysed the total number of intrusion errors in section 2 (Burgess & Shallice, 1997).

Statistical Analysis

Salivary Cortisol. The Area Under the Curve with respect to ground (AUC0) was calculated from the three morning cortisol saliva samples as an estimate of the total cortisol secretion over the first half an hour after awakening (y = 0; cortisol levels at time 0). This method is often used in cortisol research where there are repeated measurements over time (e.g., Fekedulegn et al., 2007; Vreeburg et al., 2010).

Cognitive variables. To reduce the number of variables in the analyses, cognitive variables were clustered into cognitive domains. We separated working memory tasks from other tasks of executive functioning based on the division of tasks in the CogState manual (also see Baddeley, 1992). Separate speed and accuracy composites were calculated by standardizing the scores for each relevant task and averaging them. See Table 1 for a complete list of composites. For composites that included tasks with primary outcome measures in different directions (e.g., total number of errors vs. total number of correct responses), z-scores were reflected prior to averaging, such that higher scores indicate better performance for accuracy measures, and longer response times for speed measures. These were then re-reflected to make all scores positive.
Table 1. Cognitive Composites derived from CogState tasks and paper and pencil measures

<table>
<thead>
<tr>
<th>Composites</th>
<th>Tasks</th>
<th>Primary Outcome Measure</th>
<th>Additional Outcome Measure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vigilance</td>
<td>Detection Task</td>
<td>Speed of responding (lmm: log10 ms)</td>
<td>Accuracy</td>
</tr>
<tr>
<td></td>
<td>Identification Task</td>
<td>Speed of responding (lmm: log10 ms)</td>
<td>Accuracy</td>
</tr>
<tr>
<td>Long-term Memory</td>
<td>Continuous Paired Learning Task</td>
<td>Total no. errors across 5 rounds</td>
<td>Speed of responding (lmm:</td>
</tr>
<tr>
<td></td>
<td>International Shopping List</td>
<td></td>
<td>log10 ms)</td>
</tr>
<tr>
<td></td>
<td>(Immediate recall)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>International Shopping List</td>
<td>No. correct responses across 3 rounds</td>
<td>N/A</td>
</tr>
<tr>
<td></td>
<td>(Delayed recall; approx. 30 mins)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Delayed-Recall</td>
<td>Groton Maze Learning Test</td>
<td>Total no. errors</td>
<td>Moves per second</td>
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<tr>
<td></td>
<td>(Delayed recall; approx. 20 mins)</td>
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<tr>
<td></td>
<td>International Shopping List</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(Delayed recall; approx. 30 mins)</td>
<td>No. correct responses across 3 rounds</td>
<td>N/A</td>
</tr>
<tr>
<td>Executive Functioning</td>
<td>Groton Maze Learning Test</td>
<td>Total no. errors</td>
<td>Moves per second</td>
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<tr>
<td></td>
<td>(Immediate recall)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hayling Sentence Completion Test</td>
<td>Total no. intrusion errors</td>
<td>N/A</td>
</tr>
<tr>
<td>Working Memory</td>
<td>One-Back Card Task</td>
<td>Accuracy</td>
<td>Speed of responding (lmm:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>log10 ms)</td>
</tr>
</tbody>
</table>

Note: The immediate recall version of the International Shopping List is a measure of long-term memory. Any measure which requires recall of information after >2 minutes is, by definition, a measure of long-term memory (Cardwell & Flanagan, 2005).
Data were analysed using SPSS Version 20 (IBM, Inc) and were screened for normality according to the criteria: skewness < 2, kurtosis < 7 (Curran, West, & Finch, 1996). Prior to analysis, data were transformed using “Log10 transformations” where appropriate (Field, 2009). Cronbach’s alpha was used as a measure of internal consistency. Estimates of effect sizes were partial η² for continuous data or Cramer’s Phi for categorical data (Tables 2 and 3).

There were two stages of analysis. Firstly, to investigate the association of sleep and cognition in the whole sample, standard regression analyses were conducted entering relevant covariates (demographic variables that were significantly associated with the dependent variable) in the first step and then predictors (sleep) in the second step. In all analyses of memory and executive functioning, vigilance (also examined as an independent variable) was added as a covariate (at Step 1).

Sleep variables assessed with actigraphy included: total sleep time (TST), sleep latency, sleep efficiency, and time spent awake after sleep onset (WASO). Sleep variables assessed with PSG included: %REM and %SWS. All correlations were one-tailed based on theory, except those where no prediction of the direction of the expected effect could be made, in which case two-tailed tests were reported. Secondly, a series of moderation analyses was conducted to investigate the effect of depression (dummy-coded for currently depressed versus not currently depressed) on the relationship between sleep and cognition using Hayes’ moderation method (Hayes, Glynn, & Hug, 2012).

Bootstrapping was used to calculate 95% bias-corrected confidence intervals (CI) using 5000 bootstrapped samples (Hayes et al., 2012). All analyses were corrected for heteroscedasticity. Means were not centred (Echambadi & Hess, 2007) – (for critical discussion see Hayes et al., 2012). According to Cohen (1988) effect sizes for values of $R^2$ can be defined as follows: small effect = .02; medium effect = .13; large effect = .26. Predictions regarding the nature of the effects were made a priori, hence no adjustments for multiple comparisons were made (Rothman, 1990). For the sake of parsimony, only significant results ($p < .050$) or trend-level findings ($p < .100$) are reported.

**Results**

Descriptive statistics for demographic and sleep variables are reported in Table 2, and cognitive data are provided in Table 3. Simple group comparisons are provided, however, they did not take into account the effect of covariates, hence the regression analyses below. Bivariate associations for all study variables can be seen in Table 4.
Table 2. Descriptive statistics for demographic and sleep variables for currently depressed versus not currently depressed participants

<table>
<thead>
<tr>
<th>Variable</th>
<th>Currently Depressed</th>
<th>n</th>
<th>Not Currently Depressed</th>
<th>n</th>
<th>p</th>
<th>Partial η² or Cramer’s Phi</th>
</tr>
</thead>
<tbody>
<tr>
<td>M (SD), range, Median [IQR], range, or N: %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>59.80(6.76), 51-74</td>
<td>10</td>
<td>60.94(6.45), 50-79</td>
<td>33</td>
<td></td>
<td>.631&lt;.001</td>
</tr>
<tr>
<td>Gender</td>
<td>4:40%</td>
<td>10</td>
<td>7:21.21%</td>
<td>33</td>
<td>.233&lt;.001</td>
<td></td>
</tr>
<tr>
<td>Taking antidepressants</td>
<td>8: 80%</td>
<td>10</td>
<td>9: 27.27%</td>
<td>33</td>
<td>&lt;.001 .46</td>
<td></td>
</tr>
<tr>
<td>PSQI Use of Sleep Medication²</td>
<td>1.80(1.55), 0-3</td>
<td>10</td>
<td>0.31(0.64), 0-3</td>
<td>33</td>
<td>&lt;.001 .33</td>
<td></td>
</tr>
<tr>
<td>Currently employed</td>
<td>5: 50%</td>
<td>10</td>
<td>20: 60.61%</td>
<td>33</td>
<td>.551&lt;.001 .09</td>
<td></td>
</tr>
<tr>
<td>PHQ-9³</td>
<td>16.60 (4.23), 11-24</td>
<td>10</td>
<td>2.84 (2.97), 0-9</td>
<td>33</td>
<td>&lt;.001 .77</td>
<td></td>
</tr>
<tr>
<td>Cortisol (AUCG)</td>
<td>598.98(469.20), 168.36-1557.42</td>
<td>8</td>
<td>334.89(186.24), 67.77-817.36</td>
<td>27</td>
<td>.040 .09</td>
<td></td>
</tr>
<tr>
<td>RDI²</td>
<td>4.75(16.20), 0.10-94</td>
<td>10</td>
<td>3.45(10.20), 0-31</td>
<td>32</td>
<td>.443&lt;.001 .02</td>
<td></td>
</tr>
<tr>
<td>ODI-3⁵</td>
<td>2.83(13.62), 0.15-23.69</td>
<td>10</td>
<td>1.83(9.46), 0-22.02</td>
<td>28</td>
<td>.907=0</td>
<td></td>
</tr>
<tr>
<td>MEQ total⁶</td>
<td>52.33(5.98), 42-61</td>
<td>9</td>
<td>57.90(9.26), 37-75</td>
<td>33</td>
<td>.099&lt;.001 .07</td>
<td></td>
</tr>
<tr>
<td>Actigraphy TST⁷</td>
<td>395.10(123.71), 144.19-553.60</td>
<td>10</td>
<td>405.89(60.67), 217.21-510.05</td>
<td>33</td>
<td>.177=0</td>
<td></td>
</tr>
<tr>
<td>Actigraphy Sleep Efficiency⁸</td>
<td>77.12(19.80), 24.36-89.35</td>
<td>10</td>
<td>83.35(8.55), 53.84-92.47</td>
<td>33</td>
<td>.058&lt;.001 .18</td>
<td></td>
</tr>
<tr>
<td>Actigraphy Sleep Latency⁹</td>
<td>21.21(16.26), 4.54-60</td>
<td>10</td>
<td>13.74(11.77), 1.27-48.14</td>
<td>33</td>
<td>.079&lt;.001 .06</td>
<td></td>
</tr>
<tr>
<td>Actigraphy WASO¹⁰</td>
<td>44.74(14.84), 23.77-70</td>
<td>10</td>
<td>53.03(28.83), 11.15-118.80</td>
<td>33</td>
<td>.153&lt;.001 .03</td>
<td></td>
</tr>
<tr>
<td>%REM</td>
<td>27.71(9.73), 14.50-43.90</td>
<td>10</td>
<td>23.48(7.31), 3.30-40.70</td>
<td>33</td>
<td>.073&lt;.001 .05</td>
<td></td>
</tr>
<tr>
<td>%SWS</td>
<td>7.68(6.35), 0-17.70</td>
<td>10</td>
<td>13.45(7.61), 1.40-36.50</td>
<td>33</td>
<td>.018&lt;.001 .10</td>
<td></td>
</tr>
</tbody>
</table>

Note. ¹Sample size varies due to missing data; PSQI = Pittsburgh Sleep Quality Index; PHQ-9 = Patient Health Questionnaire; AUCG = Area Under the Curve with reference to ground; RDI = Respiratory Disturbance Index; ODI-3 = Oxygen Desaturation Index, below 3%; MEQ = Morningness Evenness Questionnaire; TST = Total Sleep Time (mins); WASO = Wake After Sleep Onset (mins); REM = Rapid Eye Movement sleep; SWS = Slow Wave Sleep; ²Higher scores indicate greater use of sleep medication; Higher scores indicate greater depressive symptoms; ³Higher scores indicate greater respiratory disturbance; ⁴Higher scores indicate greater oxygen desaturation; ⁵Higher scores indicate morning preference; ⁶Higher scores indicate longer sleep time; ⁷Higher scores indicate better sleep efficiency (%); ⁸Higher scores indicate longer sleep latency (mins); ¹⁰Higher scores indicate more time spent awake after sleep onset; ¹¹Two-tailed: all tests were one-tailed based on theory, except those where no prediction of the direction of the expected effect could be made, in which case two-tailed tests are reported.
Table 3. Descriptive statistics for cognitive variables (z scores) for currently depressed and not currently depressed participants

<table>
<thead>
<tr>
<th></th>
<th>Currently Depressed</th>
<th>Not Currently Depressed</th>
<th>n(^1)</th>
<th>p</th>
<th>Cramer’s Phi</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>M (SD), range</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NART converted WAIS-R IQ(^3)</td>
<td>108.78(10.01), 95.88 – 119.44</td>
<td>113.58(8.72), 89.68-128.12</td>
<td>10</td>
<td>.148(^6)</td>
<td>.05</td>
</tr>
<tr>
<td>Vigilance (speed)(^4)</td>
<td>2.66(1.21), 1.15-4.69</td>
<td>2.45 (0.82), 0.10-3.90</td>
<td>10</td>
<td>.268</td>
<td>.01</td>
</tr>
<tr>
<td>Vigilance (accuracy)(^5)</td>
<td>2.26(0.76), 1.27-3.21</td>
<td>2.33(0.70), 1-3.21</td>
<td>10</td>
<td>.395</td>
<td>0</td>
</tr>
<tr>
<td>Long-term memory (speed)(^4)</td>
<td>3.57(0.10), 3.37 – 3.73</td>
<td>3.48(0.14), 3.20-3.76</td>
<td>10</td>
<td>.037</td>
<td>.08</td>
</tr>
<tr>
<td>Long-term memory (accuracy)(^5)</td>
<td>2.54(0.94), 1-3.78</td>
<td>3.06(0.76), 1.39-4.41</td>
<td>10</td>
<td>.042</td>
<td>.07</td>
</tr>
<tr>
<td>Delayed recall (speed)(^4)</td>
<td>0.17(0.07), 0.02 – 0.26</td>
<td>0.15(0.07), 0-0.3</td>
<td>10</td>
<td>.197</td>
<td>.02</td>
</tr>
<tr>
<td>Delayed recall (accuracy)(^5)</td>
<td>3.08(1.02), 1-4.41</td>
<td>3.30(0.67), 2.10-4.67</td>
<td>10</td>
<td>.215</td>
<td>.02</td>
</tr>
<tr>
<td>Working memory (speed)(^4)</td>
<td>2.87(0.11), 2.66 – 3</td>
<td>2.91(0.08), 2.75-3.05</td>
<td>10</td>
<td>.115</td>
<td>.04</td>
</tr>
<tr>
<td>Working memory (accuracy)(^5)</td>
<td>1.27(0.09), 1.16 – 1.40</td>
<td>1.34(0.16), 0.96-1.57</td>
<td>10</td>
<td>.083</td>
<td>.05</td>
</tr>
<tr>
<td>Executive functioning (speed)(^4)</td>
<td>1.42(0.15), 1.27-1.69</td>
<td>1.34(0.15), 1-1.69</td>
<td>10</td>
<td>.096</td>
<td>.04</td>
</tr>
<tr>
<td>Executive functioning (accuracy)(^5)</td>
<td>2.22(0.81), 1.09-3.54</td>
<td>2.73(0.53), 1-3.62</td>
<td>9</td>
<td>.015</td>
<td>.12</td>
</tr>
</tbody>
</table>

Note. 1Sample size varies due to missing data; NART = National Adult Reading Test; WAIS-R = Wechsler Adult Intelligence Scale Revised; 2Higher scores indicate a greater number of errors; 3Higher scores indicate higher IQ; 4Higher scores indicate slower performance; 5Higher scores indicate better performance; 6two-tailed: all tests were one-tailed based on theory, except those where no prediction of the direction of expected effect could be made, in which case two-tailed tests are reported.
Table 4. Correlation matrix (Pearson correlation coefficients) for all study variables

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>IQ</th>
<th>PSQI Use of sleep medication</th>
<th>Vigilance (speed)</th>
<th>Vigilance (accuracy)</th>
<th>RDI³</th>
<th>ODI-3³</th>
<th>MEQ⁶</th>
<th>Cortisol AUCo</th>
<th>TST (mins)¹</th>
<th>Sleep latency (mins)¹</th>
<th>Sleep efficiency (%)¹</th>
<th>WASO (mins)¹²</th>
<th>% REM</th>
<th>% NREM</th>
<th>% SWS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-</td>
<td>.05¹¹</td>
<td>-0.9</td>
<td>-</td>
<td>-.52‡</td>
<td>.47‡</td>
<td>-.13</td>
<td>-.22</td>
<td>-.35†</td>
<td>.17</td>
<td>-.28†</td>
<td>-.20</td>
<td>.20</td>
<td>-.27†</td>
<td>.28†</td>
<td>-.12</td>
</tr>
<tr>
<td>Cortisol AUCo</td>
<td>-.22</td>
<td>.21¹¹</td>
<td>.39†</td>
<td>-.19</td>
<td>.01</td>
<td>-.02</td>
<td>-.20</td>
<td>-.25¹Í</td>
<td>-</td>
<td>-.19</td>
<td>.13</td>
<td>-.26</td>
<td>.10</td>
<td>.33†</td>
<td>-.33†</td>
<td>-.06</td>
</tr>
<tr>
<td>Vigilance (speed)</td>
<td>.31†</td>
<td>-.21</td>
<td>-.12†</td>
<td>-.09</td>
<td>.07</td>
<td>.04</td>
<td>.22¹Í</td>
<td>-.19</td>
<td>-.10</td>
<td>.04</td>
<td>-.14</td>
<td>.32†</td>
<td>-.12</td>
<td>.12</td>
<td>.11</td>
<td>.13</td>
</tr>
<tr>
<td>Vigilance (accuracy)²</td>
<td>.06</td>
<td>.17</td>
<td>.06¹Í</td>
<td>-</td>
<td>-.07</td>
<td>.16</td>
<td>-.03Í</td>
<td>.01</td>
<td>.09</td>
<td>-.20</td>
<td>.11</td>
<td>.08</td>
<td>.15</td>
<td>.15</td>
<td>.13</td>
<td>.13</td>
</tr>
<tr>
<td>Long-term memory (speed)¹</td>
<td>.38‡</td>
<td>-.06</td>
<td>.07¹Í</td>
<td>.33†</td>
<td>.04</td>
<td>.25</td>
<td>.26</td>
<td>-.08Í</td>
<td>-.02</td>
<td>-.09</td>
<td>.04</td>
<td>-.04</td>
<td>-.12</td>
<td>.03</td>
<td>.13</td>
<td>.13</td>
</tr>
<tr>
<td>Long-term memory (accuracy)³</td>
<td>.12</td>
<td>.05</td>
<td>-.10Í</td>
<td>-.33†</td>
<td>-.01</td>
<td>.19</td>
<td>-.19</td>
<td>.13Í</td>
<td>.12</td>
<td>0</td>
<td>-.08</td>
<td>.16</td>
<td>.05</td>
<td>.01</td>
<td>-.01</td>
<td>.13</td>
</tr>
<tr>
<td>Delayed recall (speed)¹</td>
<td>.16</td>
<td>-.49‡</td>
<td>.12Í</td>
<td>.33†</td>
<td>-.09</td>
<td>.06</td>
<td>.10</td>
<td>.19Í</td>
<td>-.27</td>
<td>-.02</td>
<td>.25</td>
<td>-.14</td>
<td>.09</td>
<td>-.23</td>
<td>.22</td>
<td>.19</td>
</tr>
<tr>
<td>Delayed recall (accuracy)³</td>
<td>-.11</td>
<td>.36‡</td>
<td>-.27Í</td>
<td>-.38‡</td>
<td>-.02</td>
<td>-.06</td>
<td>-.02</td>
<td>-.08Í</td>
<td>-.03</td>
<td>-.12</td>
<td>-.12</td>
<td>.02</td>
<td>-.26†</td>
<td>.01</td>
<td>.01</td>
<td>-.02</td>
</tr>
<tr>
<td>Working memory (speed)¹</td>
<td>.42‡</td>
<td>-.19</td>
<td>-.26Í</td>
<td>.61‡</td>
<td>-.13</td>
<td>.03</td>
<td>.15</td>
<td>.08Í</td>
<td>-.35†</td>
<td>-.17</td>
<td>.25</td>
<td>-.21</td>
<td>.15</td>
<td>-.14</td>
<td>.14</td>
<td>.03</td>
</tr>
<tr>
<td>Working memory (accuracy)³</td>
<td>-.13</td>
<td>-.02</td>
<td>.04Í</td>
<td>-.11</td>
<td>.20</td>
<td>.01</td>
<td>.10</td>
<td>.04Í</td>
<td>-.13</td>
<td>.30†</td>
<td>-.10</td>
<td>.29†</td>
<td>-.04</td>
<td>.03</td>
<td>-.04</td>
<td>-.03</td>
</tr>
<tr>
<td>Executive functioning (speed)¹</td>
<td>.34‡</td>
<td>-.29†</td>
<td>.15Í</td>
<td>-.38‡</td>
<td>.03</td>
<td>.16</td>
<td>.21</td>
<td>.04Í</td>
<td>-.30†</td>
<td>-.03</td>
<td>.11</td>
<td>-.09</td>
<td>.08</td>
<td>-.17</td>
<td>.16</td>
<td>.09</td>
</tr>
<tr>
<td>Executive functioning (accuracy)³</td>
<td>-.09</td>
<td>.54‡</td>
<td>-.39Í</td>
<td>-.18</td>
<td>-.10</td>
<td>-.02</td>
<td>.09</td>
<td>.02Í</td>
<td>0</td>
<td>.05</td>
<td>-.22</td>
<td>.01</td>
<td>-.11</td>
<td>.01</td>
<td>0</td>
<td>.14</td>
</tr>
</tbody>
</table>

Note. PSQI = Pittsburgh Sleep Quality Index; Cortisol AUCo = Area under the curve with respect to ground; RDI = Respiratory Disturbance Index; ODI-3 = Oxygen desaturation Index, below 3%; MEQ = Morningness-Eveningness Questionnaire; TST = Total sleep time (mins); WASO = Wake after sleep onset (mins); REM = Rapid eye movement sleep; NREM = Non Rapid eye movement sleep; SWS = Slow wave sleep; ¹Higher scores indicate greater use of sleep medication; ²Higher scores indicate slower speed; ³Higher scores indicate greater respiratory disturbance; ⁴Higher scores indicate greater oxygen desaturation; ⁵Higher scores indicate better performance; ⁶Higher scores indicate longer sleep latency; ⁷Higher scores indicate lower sleep efficiency; ⁸Higher scores indicate more time spent awake after sleep onset; ¹²Two tailed: all tests were one-tailed based on theory, except those where no prediction of the direction of expected effect could be made, in which case two-tailed tests are reported; † p < .05; ‡ p < .01.
Morning salivary cortisol differed between currently depressed and not currently depressed groups \(t(33) = 1.80, p = .041\) (one-tailed), \(d = .66\), indicating a moderate effect. This confirms elevated cortisol in the depressed sample, providing a potential biological index of depression.

**Vigilance (speed)**

Standard regression analysis revealed that age \((\beta = .04, p = .005)\) was a significant covariate of vigilance \((R^2 = .09, p = .005)\), indicating 9% of variance explained. After controlling for age, WASO \((\beta = .01, p = .043)\) was a significant predictor of vigilance speed \((R^2 = .17, p = .007)\), explaining an additional 8% of variance. There was no effect of depression and no evidence of moderation effects.

**Vigilance (accuracy)**

Standard regression analysis revealed that there were no significant covariates and none of the sleep variables predicted vigilance accuracy. There was no effect of depression and no evidence of moderation effects.

**Long-term memory (speed)**

Standard regression analysis revealed that age \((\beta = .01, p = .008)\) was a significant covariate of long-term memory speed \((R^2 = .15, p = .008)\), however, none of the sleep variables significantly predicted additional variance. When depression was added as a moderator of the relationship between %SWS and long-term memory speed, it explained an additional 18% of variance in long-term memory speed \((\beta = .16, p = .004\), overall model \(R^2 = .33, p < .001)\). However, depression did not moderate the relationship between sleep and long-term memory speed.

**Long-term memory (accuracy)**

Standard regression analysis revealed that gender \((\beta = 1.20, p < .001)\) and vigilance speed \((\beta = -.36, p = .022)\) were significant covariates of long-term memory accuracy \((R^2 = .45, p < .001)\), indicating 45% of variance explained, however, no additional variance was explained by sleep variables. There were no effects of depression or any interactions between depression and sleep.

**Delayed recall (speed)**

Standard regression analysis revealed that IQ \((\beta = 0, p = .004)\) was a significant predictor of delayed recall speed \((R^2 = .24, p = .004)\). After controlling for IQ, %SWS \((\beta = 0, p = .064)\) was a trend-level predictor of delayed recall speed \((R^2 = .31, p = .002)\), explaining an additional 7% of the variance. No other sleep variables were significant predictors of delayed recall (speed). There were no direct effects of depression or interaction effects of depression and sleep.

**Delayed recall (accuracy)**

Standard regression analysis revealed that IQ \((\beta = .03, p = .029)\) was a significant covariate of delayed recall accuracy \((R^2 = .13, p = .029)\). After controlling for IQ, WASO was a significant predictor of delayed recall accuracy \((\beta = -.01, p = .023, R^2 = .20, p = .016)\), explaining an additional 7% of variance. Although no other sleep variables were significant, there was a significant interaction effect. When depression was added as a moderator of the relationship between sleep latency and delayed recall accuracy, depression was not a significant predictor, but the interaction of sleep latency and depression reached trend-level \((\beta = .05, p = .052)\), explaining an additional 14% of variance (overall model was significant, \(R^2 = .27, p = .011)\). Examination of conditional effects of sleep latency (X) on delayed recall accuracy (Y) at values of the moderator (depression: 0 or 1) revealed that the interaction was being driven by the not currently depressed group,
also at trend-level \((p = .065)\). There were no effects within the depressed group. That is, longer sleep latency predicted poorer delayed recall in the not currently depressed group only, but only at trend levels.

**Working memory (speed)**

Standard regression analysis revealed that age \((\beta = 0, p = .033)\) and vigilance speed \((\beta = .05, p < .001)\) were significant covariates of working memory speed \((R^2 = .44, p < .001)\), explaining 44% of the variance. Sleep variables did not help to explain working memory speed. Depression was a significant independent predictor of working memory speed \((\beta = .25, p = .016)\), and there was a significant interaction of total sleep time and depression \((\beta = 0, p = .007)\), explaining an additional 14% of variance (overall model was significant, \(R^2 = .58, p < .001\)). Inspection of conditional effects of total sleep time \((X)\) on working memory speed \((Y)\) at values of the moderator (depression: 0 or 1) revealed that the interaction effect was being driven by the depressed group \((p = .019)\). This was not significant within the not currently depressed group. That is, shorter total sleep time predicted slower working memory speed in the depressed group only.

**Executive Functioning (speed)**

Standard regression analysis revealed that age \((\beta = .01, p = .014)\) and gender \((\beta = .14, p = .009)\) were significant covariates of executive functioning speed \((R^2 = .27, p = .012)\), indicating 27% of variance explained. No sleep variables were independent predictors of executive functioning speed. Depression was not a significant independent predictor of executive functioning (speed), however, there was a significant interaction effect of WASO and depression \((\beta = .01, p = .037)\), explaining an additional 17% of variance (overall model was significant, \(R^2 = .44, p < .001\)). Inspection of conditional effects of WASO \((X)\) on executive functioning speed \((Y)\) at values of the moderator (depression: 0 or 1), revealed that the interaction effect was being driven by the depressed group at trend- level \((p = .084)\). This was not significant within the not currently depressed group. That is, greater WASO was linked to slower executive functioning speed in the depressed group only, but at trend level.

**Executive Functioning (accuracy)**

Standard regression analysis revealed that IQ \((\beta = .03, p = .014)\) was a significant predictor of executive functioning accuracy \((R^2 = .18, p = .014)\), indicating 18% of variance explained. No sleep variables were independent predictors of executive functioning accuracy.

Subsequent moderation analysis revealed that depression was a significant predictor of executive functioning accuracy at trend-level \((\beta = 2.72, p = .077)\) and there was a significant interaction effect of total sleep time and depression \((\beta = -.01, p = .029)\), explaining an additional 30% of variance in executive functioning accuracy (overall model was significant, \(R^2 = .48, p = .002\)). Inspection of conditional effects of total sleep time \((X)\) on executive functioning...
accuracy (Y) at values of the moderator (depression: 0 or 1) revealed that the interaction was being driven by the not currently depressed group (p = .019). The association of total sleep time and depression was not significant within the depressed group. That is, longer total sleep time was linked to better executive functioning in the not currently depressed group only.

Further moderation analyses revealed that depression was a significant independent predictor of executive functioning accuracy in the context of sleep efficiency (β = 3.89, p = .004), and there was a significant interaction effect of sleep efficiency and depression (β = -.05, p < .001), explaining an additional 25% of variance (overall model was significant, R² = .52, p < .001).

Inspection of conditional effects of sleep efficiency (X) on executive functioning accuracy (Y) at values of the moderator (depression: 0 or 1) revealed that the association of sleep efficiency and executive functioning accuracy was significant in both the depressed group (p < .001) and the not currently depressed group at trend-level (p = .053). That is, higher sleep efficiency was linked to better executive functioning within the not currently depressed group and poorer sleep efficiency was linked to better executive functioning within the depressed group. However, inspection of scatterplots revealed that the association within the depressed group was being driven by an outlier (a participant with very low sleep efficiency and high executive functioning). Once this score was removed, the interaction effect disappeared.

When depression was added as a moderator of sleep latency and executive functioning accuracy, it was a significant independent predictor of executive functioning (β = -1.32, p = .007) at trend-level, and there was a significant interaction effect of sleep latency and depression (β = .05, p = .048), explaining an additional 26% of variance (overall model was significant, R² = .44, p = .019). Inspection of conditional effects of sleep latency (X) on executive functioning accuracy (Y) at values of the moderator (depression: 0 or 1) revealed that the interaction was being driven by the not currently depressed group at trend-level (p = .076). The association of sleep latency and depression was not significant within the depressed group. That is longer sleep latency was linked to poorer executive functioning in the not currently depressed group only.

Therefore, results suggest that there was evidence of a different pattern of association of sleep and cognition in currently depressed versus not currently depressed participants. That is, specific aspects of actigraphic sleep, were linked to performance accuracy decrements in the not currently depressed group, and slower speed of processing in the currently depressed group.

Discussion

This study investigated the effect of sleep on cognition in older adults, and whether depression impacted on this relationship. Results indicate associations between sleep-wake patterns and cognition in the whole sample. By contrast, sleep architecture largely did not impact on cognition. Finally, depression altered the relationship between sleep and cognition, but only for aspects of sleep-wake patterns and not sleep stages. The results are discussed in turn.

Sleep and cognition in the whole sample

Sleep-wake patterns, as indexed by actigraphy, were linked to vigilance, memory and working memory in the entire sample. Specifically, greater WASO was linked to slower vigilance (speed) and poorer memory (delayed recall accuracy). Lower sleep efficiency and shorter sleep
duration (at trend-level) were linked to poorer working memory accuracy.

Findings are consistent with an actigraphic study that found older insomniacs with greater WASO had slower reaction times on a sustained attention task and poorer working memory on a memory span task (Haimov et al., 2008). Results also replicate findings from self-report studies in older adults demonstrating the impact of poor sleep quality on memory (Schmutte et al., 2007) and working memory accuracy (Nebes et al., 2009).

While some studies suggest that sleep quality is more important than sleep quantity (e.g., Blackwell et al., 2011), our results are in line with other studies showing a link between shorter self-reported sleep duration and poorer cognition (e.g., Ohayon & Vecchierini, 2005; Tworoger et al., 2006). Therefore, the current results suggest that sleep-wake patterns are important predictors of cognitive performance in older adults.

With regard to sleep stages, more SWS predicted slower memory performance (delayed recall tasks), albeit only at trend-level, but did not predict delayed recall accuracy. This is not in line with a previous SDMC study showing a positive relationship between SWS and declarative memory in older adults (Backhaus et al., 2007), and might indicate a discontinuation of the SWS-memory link. Results, thus, indicate that the relationship between sleep architecture and cognition might change with age (“functional-dissociation” account).

Overall, the lack of a positive correlation between SWS and REM and cognitive performance, lends support for the “functional-dissociation” account of sleep stages and cognition, rather than the neurodevelopmental theories.

The role of depression

This study revealed medium to large interaction effects of depression and actigraphic sleep variables in predicting cognition. That is, depression had a significant impact on the relationship between sleep-wake patterns (but not sleep architecture) and cognition.

Sleep and cognition in the depressed group. Shorter total sleep time and greater WASO (at trend-level) were linked to slower performance on tasks of working memory and executive functioning, respectively. These findings are in line with a questionnaire study that linked poorer overall sleep quality (Global PSQI) to executive functioning problems in participants with high levels of depressive symptoms (Sutter et al., 2012), and with an actigraphy study that linked greater WASO to impaired executive functioning in older adults with depression (Naismith et al., 2011) – albeit these studies point to impaired accuracy rather than speed, which could be due to the different tasks used in the studies.

The impact of total sleep time on speed of working memory in the depressed group suggests that sleep duration, in addition to sleep quality, is an important factor in cognition in older adults with depression. Thus, although confined to measures of speed, these results suggest sleep-related deficits in working memory and executive functioning in depressed older adults, which is consistent with studies implicating fronto-subcortical networks in depression, cognition and sleep (Naismith et al., 2011).

Sleep and cognition in not currently depressed older adults. Shorter total sleep time and longer sleep latency (at trend-level) were associated with poorer executive functioning performance. This is consistent with actigraphy research in older adults, which shows that longer sleep latency is
linked to poorer executive functioning (Blackwell et al., 2006), and with research in adults demonstrating the impact of sleep loss on executive functioning (e.g., Harrison & Horne, 2000).

Longer sleep latency affected memory (delayed recall accuracy) at trend-level. Results are consistent with one of the few published actigraphy studies that demonstrated a significant association of longer sleep latency and poorer overall cognition (which included measures of delayed recall) in older women (Blackwell et al., 2006). Findings are also consistent with the notion that poor sleep impacts on the medial temporal lobes and hippocampus, causing memory impairment (Drummond et al., 2000). However, the majority of evidence for this association in older adults derives from self-report studies (e.g., Schmutte et al., 2007). Therefore, the current findings provide further evidence of sleep-related memory deficits in older adults using actigraphy.

Taken together, the current findings offer novel evidence of a distinct pattern of sleep-related cognitive impairment in currently depressed versus not currently depressed older adults. In general terms, sleep disturbance was linked to poorer accuracy in the not currently depressed group, but slower processing speed in the depressed group. The finding in the depressed group is consistent with research indicating decreased processing speed in older adults with depression (McDermott & Ebmeier, 2009; Nebes et al., 2000; Sheline et al., 2006). However, it is unclear why we found accuracy deficits in the not currently depressed group, but only deficits in speed in the depressed group.

Overall, our results suggest that the relationship between sleep and cognition is qualitatively different in currently depressed versus not currently depressed older adults. Older adults with current MDD have a specific profile of sleep-related cognitive performance compared to those without MDD, suggesting that depression exerts a specific role on the relationship between sleep and cognition. An attractive explanation, therefore, suggests that the reason for the lack of positive associations found between sleep stages and cognition (“functional-dissociation” account) could be due to the presence of depression in older age, which alters the relationship between sleep and cognitive performance. However, there are problems with this explanation because the relationship between sleep-wake patterns and cognitive performance is in line with findings in the literature. In addition, we found no association of sleep architecture and cognition, in either depressed or non-depressed groups. We are limited in the conclusions we can make, but we can say that depression does impact on the relationship between sleep and cognition, but does not appear to be the reason for the changing relationship of sleep architecture and cognition observed in this study, and other studies. However, future studies would need to directly test a mediating effect of depression on the relationship between sleep architecture and cognition in order to derive firm conclusions.

Future directions

While this study demonstrates exciting and novel relationships between sleep, depression and cognition in ageing, further research is essential. Current findings provide tentative support for the “functional dissociation” account, but the mechanistic processes cannot be identified using the current methodologies.

There were some limitations of this study. First, the moderating effects of depression must be interpreted with caution due to the substantially higher proportion of not currently depressed participants compared
to currently depressed participants. This was due to difficulty recruiting depressed older adults who typically suffer from decreased motivation and loss of interest (Fiske et al., 2009).

Second, Hayes’ moderation method (Hayes et al., 2012) is useful in exploring the nature of any significant interaction effects found. However, while the exploration at the group level provides important information on how to interpret any interactions found, which is not available from conventional moderation analyses, this reduces the power of the analyses. Although limited by sample size and power, a strength of the current study was the size of the interaction effects of sleep and depression, which ranged from medium to large (Cohen, 1988) explaining a significant proportion of variance in cognitive functioning. Nevertheless, results warrant further investigation in a larger sample to see if these findings are replicated.

Third, the neuropsychological tests used in this study as part of the CogState battery, while well-validated (e.g., Falleti et al., 2003; Weaver Cargin et al., 2008), were broad. For example, the Groton Maze Learning task is said to tap into various different cognitive domains, such as problem-solving, decision-making, attention and spatial memory (Pietrzak et al., 2008). Therefore, future studies may wish to investigate the possibility of using more precise cognitive tasks which tap into different types of memory and which assess hippocampal function directly.

Fourth, recent research has highlighted the important role of neurophysiological events associated with specific sleep stages in cognition, such as REM sleep theta and sleep spindles (e.g., Cox, Hofman, & Talamini, 2012; Fogel & Smith, 2006). While it was beyond the scope of this study, future research may wish to consider spectral and spindle analysis of sleep in addition to traditional sleep stageing.

Future studies may also wish to consider an exploration of the impact of daytime sleepiness on cognition. Sleepiness was not assessed in this study given the focus on objective measures of sleep and the fact that the objective alternatives to self-reports of sleepiness (e.g., The Epworth Sleepiness Scale) are demanding in terms of time and effort.

Finally, given that this was a cross-sectional study, the direction of the relationship of sleep and cognition must be interpreted with caution. However, the majority of research in this area supports a uni-directional effect of sleep impacting on cognition, with few exceptions (e.g., see Haimov & Shatil, 2013, for an example of effects of cognition on sleep). In order to derive firm conclusions about the direction of the effects, intervention or sleep-deprivation studies would be needed.

Conclusions

This study investigated the association of sleep-wake patterns and sleep architecture and cognition in older adults, and whether the presence of current MDD impacted on this relationship. Sleep-wake patterns, but not sleep stages, were associated with cognitive performance in our sample of older adults. Furthermore, sleep-wake patterns had dissociable effects on cognition in currently depressed versus not currently depressed older adults, such that poor sleep was associated with working memory and executive functioning speed in the currently depressed group, and with delayed recall and executive functioning accuracy in the not currently depressed group. Because sleep problems were associated with cognitive deficits in both the clinical and not currently depressed group, results highlight the need to assess sleep in all older adults. Early detection of sleep problems is important given that research has shown that
treatments for sleep problems, whether behavioural or pharmacological, can improve cognition in older adults (Pace-Schott & Spencer, 2011).

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