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Objective but not Subjective Sleep Predicts Memory in Community-Dwelling Older Adults

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Summary

Research on the relationship between habitual sleep patterns and memory performance in older adults is limited. No previous study has used objective and subjective memory measures in a large, older-aged sample to examine the association between sleep and various domains of memory. Our aim was to examine the association between objective and subjective measures of sleep with memory performance in older adults, controlling for the effects of potential confounds. 173 community-dwelling older adults aged 65-89 years in Victoria, Australia completed the study. Objective sleep quality and length were ascertained using the Actiwatch 2 Mini-Mitter, while subjective sleep was measured using the Pittsburgh Sleep Quality Index. Memory was indexed by tests of retrospective memory (Hopkins Verbal Learning Test – Revised), working memory (n-back, 2-back accuracy), and prospective memory (a habitual button pressing task). Compared to normative data, overall performance on retrospective memory function was within the average range. Hierarchical regression was used to determine whether objective or subjective measures of sleep, predicted memory performances after controlling for demographics, health, and mood. After controlling for confounds, actigraphic sleep indices (greater wake after sleep onset, longer sleep onset latency, and longer total sleep time) predicted poorer retrospective ($\Delta R^2 = .05$, $p = .016$) and working memory ($\Delta R^2 = .05$, $p = .047$). In contrast, subjective sleep indices did not significantly predict memory performances. In community-based older adults, objectively-measured, habitual sleep indices predict poorer memory performances. It will be important to follow the sample longitudinally to determine trajectories of change over time.

Keywords: actigraphy, aging, neuropsychology, working memory, prospective memory, cognition

Reductions in the length and quality of sleep are common in normal ageing (Ohayon et al., 2004), and while recent research has evaluated the importance of sleep for general cognition (Yaffe et al., 2007), and memory consolidation post-learning (see Scullin et al., 2015 for comprehensive review), the relationship between habitual sleep patterns and memory in older adults has not been sufficiently studied (Cochrane et al., 2012; Naismith et al., 2011; Seelye et al., 2015; Wilckens et al., 2014). Deterioration in various domains of memory is common with age; and, evaluating the relationship between habitual sleep patterns and domains of memory (such as retrospective memory, working memory and prospective memory) will contribute to strategies for improving cognitive health in ageing populations.

In studies using polysomnography, which examines cerebral sleep-wake states in addition to other physiological changes that occur with sleep, and is the gold standard of objective sleep measurement, indices of good sleep quality have been associated with better subsequent memory performance (Lafortune et al., 2014; see Scullin et al., 2015 for review). However, because polysomnography studies are usually conducted in sleep laboratories, both the monitoring equipment and sleeping in a novel environment can lead to disruption to a person's normal sleep, particularly on the first night of recording, known as first night effects (Edinger et al., 2001). Furthermore, in-laboratory studies generally restrict sample sizes, reducing the reliability of any found relationships. Actigraphy, which uses activity-based algorithms to infer sleep/wake states, offers an alternative approach to objective sleep measurement. Whereas it cannot measure sleep stages or microarousals, it involves unobtrusive collection of successive nights of sleep data in naturalistic settings, thereby providing a measure of habitual sleeping patterns, and lends itself to the possibility of much larger sample sizes. Although the existing actigraphy literature suggest no relationship between habitual sleep duration and memory recall or working memory in older adults (Cochrane et al., 2012; Wilckens et al., 2014); consensus is lacking about whether indices of habitual poor sleep quality are associated with poorer memory (Cochrane et al., 2012; Naismith et al., 2011; Seelye et al., 2015; Wilckens et al., 2014). Interpretation of study findings is limited by the use of mixed-age samples, small sample sizes, possible inclusion of people with sleep apnoea, and the use of clinical samples which provide atypical age-related

data. The relationship between habitual sleep patterns and memory in non-clinical, older-aged samples, therefore, requires further evaluation.

Research on habitual sleep patterns and memory in older adults has also been conducted using subjective measures of sleep, however, the area is again marked by significant inconsistencies in outcomes. Self-reported sleep quality, with the exception of sleep onset latency (time taken to fall asleep), has not generally been associated with poor memory in older adults, after controlling for depression (Gamaldo et al., 2008; Jaussent et al., 2012; Nebes et al., 2009; Schmutte et al., 2007; Sutter et al., 2012; Tworoger et al., 2006; Waller et al., 2015), although there are exceptions (Miller et al., 2014), and others have reported self-reported poor sleep quality at age 70 (but not age 50) to be associated with greater lifetime risk of developing dementia, in particular Alzheimer's disease (Benedict et al., 2015).

Shorter subjective sleep duration has been associated with poorer verbal memory performance in a large cohort study of 50-85 year olds (Xu et al., 2011). However, others have found short sleep duration to be associated with poorer memory in middle aged-adults (50-64 year olds), but not in older adults aged 65 and above (Miller et al., 2014). In addition, a decline in sleep duration from a baseline of 7-8 hours per night has not been associated with lower retrospective memory performance (Loerbroks et al., 2010); and while short subjective sleep duration in a sample spanning middle-age to older adulthood was associated with greater beta-amyloid burden (Spira et al., 2013), short sleep in midlife was not associated with subsequent increased risk of Alzheimer's disease in late life in a much larger cohort (Virta et al., 2013).

In contrast, longer sleep duration has more consistently been associated with poorer memory, and the development of dementia of the Alzheimer's type (Benito-León, et al., 2009; Loerbroks et al., 2010; Miller et al., 2014; Schmutte et al., 2007; Virta et al., 2013; Xu et al., 2011), although not all findings have been consistent (Nebes et al., 2009; Tworoger et al., 2006; Waller et al., 2015). A difficulty in explaining these discrepant findings is due to most studies employing subjective measures only and the degree to which subjective report reflects actual sleep is unclear (Buysse et al., 2008). While the mechanisms for the observed relationships between long subjective sleep duration and poorer memory remain contended, a number of possibilities exist; for example, degeneration of cholinergic neurons in the basal forebrain (Stern et al., 2015) or underlying poor health (Fang et al., 2014; Ramos et al., 2014)

could contribute to both. Another possible mechanism by which longer sleep may cause poor memory in older adults is through a process of over-downscaling of synaptic weights (Scullin et al., 2013). Downscaling of synapses that accrue during wakefulness is a process that has been hypothesised to occur during slow-wave sleep to promote neural efficiency (Tononi et al., 2003). It has, however, also been proposed that a relative over-downscaling of synaptic weights can occur in the case of older adults in whom slow-wave sleep is preserved (albeit at lower levels), but the accrual of synaptic weights during the waking day is overly diminished as a result of ageing (Scullin et al., 2013). Finally, poorer memory may lead to longer sleep through a process of compensation, whereby older adults with poorer memory function are cognitively exhausted as a result of increased mental effort required for daily function, and thus sleep longer as a result. Further research, however, is required to substantiate these varied explanations.

Existing research is further limited in only examining memory performance through tests of retrospective memory (the retrieval of explicit, episodic information) (Moscovitch, 1992) and working memory (the temporary storage and manipulation of information) (Baddeley, 1992), even though researchers are now recognising that prospective memory (the capacity to remember to perform an intended action in the future) (Einstein et al., 2012) is an important aspect of everyday memory. In younger adults, sleep, in particular slow-wave sleep, is involved in the consolidation of prospective memory intentions (Diekelmann et al., 2013; Scullin et al., 2010). The relationship between sleep and prospective memory has not been examined in older adults but is important to evaluate as prospective memory has been identified as critical for the maintenance of independence in activities of daily living (Schmitter-Edgecombe et al., 2009).

The objective of this study, therefore, was to investigate whether habitual sleep patterns (using objective and subjective measures) predicted various domains of memory performance in community-living older people. Sleep measures were collected prior to memory assessment, although collection of prospective memory data and actigraphic sleep data were completed in parallel. We mitigated the effects of potential confounds by controlling for mood and health disorders, and excluded people with reported sleep disorders. Although objective and subjective measures of sleep are often not associated, there is evidence that both may predict memory performance. It was, therefore, expected that both objective and subjective measures of good sleep quality would be associated with better

memory, whereas objective and subjective measures indicating long sleep duration would be associated with poorer memory.

Method

Participants

Two hundred older adult volunteers from Victoria, Australia were recruited through experimenter networks and advertisements in community-based organisations. Inclusion criteria were: (i) ≥65 years; (ii) fluent in English; (iii) independent in activities of daily living. Exclusion criteria were: (i) presence of diagnosed dementia or mild cognitive impairment; (ii) history of neurological or psychiatric disorder that may affect cognition; (iii) low cognitive status (Mini Mental State Examination score of <24); (iv) uncorrected impairment of vision, hearing, or communication that would interfere with study participation; (v) post-hoc exclusion of those who self-reported a diagnosed sleep disorder. The research was approved by La Trobe University human ethics committee, and all participants provided written informed consent.

Assessments

As part of a larger study on memory in ageing, participants completed questionnaires prior to two neuropsychological assessment sessions, with measures of objective sleep, prospective memory and a subjective sleep diary collected between the two sessions, spaced two weeks apart.

Objective Sleep. Actigraphy is a widely used instrument that objectively estimates sleep quantity and quality based on wrist movement measured using wristwatch-like devices. It is light-weight, non-intrusive, and validated in older adults (van Hilten et al., 1993). Activity data were sampled in one minute epochs using a medium (default) threshold for sleep/wake determination (Actiwatch 2 Mini-Mitter, Phillips-Respironics, OR, USA), over a fortnight (mean = 13.38 days [*SD* = 1.79]). Bed-times and Rise-times were determined by concordance between light, activity, and sleep diary data. Nocturnal sleep quality was indexed by (i) wake after sleep onset ($WASO_{Acti}$ [mins]), and (ii) sleep onset latency (SOL_{Acti} [mins]). Nocturnal sleep quantity was indexed by total sleep time (TST_{Acti}; minutes in bed scored as sleep). These variables relate to distinct aspects of sleep measurement, have been used in other related research (Cochrane et al., 2012; Wilckens et al., 2014), and were not highly correlated

($WASO_{Acti}$ and SOL_{Acti} r = .46, p <.001; $WASO_{Acti}$ and TST_{Acti} r = -.19, p = .006; and SOL_{Acti} and TST_{Acti} r = -.19, p = .006), thus would not lead to multicollinearity in regression.

Subjective Sleep. The widely used Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989) was completed by participants prior to the first cognitive assessment session (test-retest r = .87) (Backhaus et al., 2002). The PSQI assesses sleep over the month prior using 19 questions which are converted into seven subscale scores, ranging from 0-3 (with higher numbers indicating poorer sleep), the sum of which provides a global score. In order to use subscales that appear to provide a similar construct to the actigraphic sleep variables, and to avoid multicollinearity in regressions, the following variables were extracted for analysis: Sleep quality was indexed by: (i) the subscale score for $Sleep\ Disturbance_{PSQI}$, and (ii) the raw score for SOL_{PSQI}. Sleep quantity was indexed by the raw score for TST_{PSQI}. We also included the subscale score $Daytime\ Dysfunction_{PSQI}$ to indicate the subjective impact of sleep on daily function. Incomplete responses to the questionnaires led to missing data on some of the PSQI variables (resulting in SOL_{PSQI} n = 164; TST_{PSQI} n = 169; $Daytime\ Dysfunction_{PSQI:}$ n = 171). While wearing the Actiwatch, participants completed a sleep diary, with bed-time and rise-time used to aid scoring of the actigraph.

Memory Performance. The Hopkins Verbal Learning Test – Revised (HVLT-R) (Brandt et al., 2001) was administered, using the standard procedure, in the second assessment session. This task is a word-list memory task in which the experimenter orally presented a 12 item word-list from three semantic categories. There are three learning trials, and participants are asked to recall as many words as they can after each trial. . This was followed by a 20-30 minute delay with a subsequent delayed free-recall trial without representing the word-list; and, the total number of correct words in the delayed free-recall trial was used as the index of retrospective memory. In the delay between the learning and delayed recall trials, the n-back task was administered (conditions 0-, 1-, and 2-back). Working memory was assessed by the n-back task, 2-back condition (accuracy). This computer-based task consisted of two blocks of 30 trials of random lower case letters presented for 500 milliseconds, with a fixed inter-stimulus interval of two seconds. Participants indicated, on a numeric keypad, whether or not the currently presented letter was the same as the one presented two previously, which occurred 10 times in each of the 30 trial blocks.

Prospective memory was assessed using the event marker button on the Actiwatch that had been provided in the first assessment session to objectively record sleep patterns.

The event marker button records time-stamped information when pressed. At the end of the first assessment session, in addition to the general instructions about wearing the watch, participants were asked to press the event marker button daily when getting into bed and intending to go to sleep (i.e., at "lights out"), and upon rising from bed in the morning over the two week assessment period. A typed instruction sheet about the use of the actiwatch was also provided to each participant but they were not informed that the instructions contained a memory test. The actiwatch was returned at the second assessment session. Successful prospective memory performance was determined by the proportion of button presses within 10 minutes of Rise-time over the two-week assessment period (Cavuoto et al., in press). Rise-time prospective memory was selected for the study variable as it was relevant to investigating the relationship between sleep and prospective memory performance.

Due to logistic reasons, one participant was unable to complete the second assessment session, and data were lost for four participants following n-back administration, which led to n = 172 for the HVLT-R, and n = 168 for the n-back.

Vigilance. Vigilance was objectively measured using a 5 minute psychomotor vigilance task (PVT; Thorne et al., 2005). This was administered in the same testing session as the HVLT-R and n-back task (approximately 20 minutes prior to these tasks). Due to logistic reasons, data were only collected on 162 participants. Total number of lapses (i.e., response >500 ms) was included as a covariate.

Health. Three indices of health were used - (i) The number of self-reported vascular medical conditions (sum of transient ischaemic attack, heart problem, high cholesterol, pulmonary disease, high blood pressure, and diabetes), an approach similar to that used by others (McKinnon et al., 2014); (ii) the number of self-reported, current medications; and (iii) the PSQI subscale for frequency of sleep medication use (dichotomised into those who reported medication use versus no medication use over the past month). For the latter question there were missing data for two participants.

Mood. The 21-item version of the Depression, Anxiety, and Stress Scale (DASS-21) is a well-validated measure of mood (Lovibond et al., 1993). Items are rated 0-3, with higher numbers indicating greater severity. Each scale is multiplied by two to reflect the full (42-item) version of the scale. Indices of depression, anxiety and stress were derived, with Cronbach's alpha levels of .72, .62, and .80, respectively.

Statistical Method

Outliers were replaced with a value just outside of the median ± 3 x interquartile range ("^{O}"). Skewed or kurtotic variables were square-root ("^{SQ}") or natural-log transformed ("^{LN}"), and, where necessary, were reflected before transformation ("^{RSQ}"/"^{RLN}"). Analyses were conducted using transformed variables. The assumption of linearity was considered upheld. In order to determine the contribution of sleep variables to memory performance, over and above that accounted for by potentially confounding variables (i.e., demographics, health, and mood), the following analyses were conducted. Two hierarchical regressions were performed to determine whether i) $WASO_{Acti}$, SOL_{Acti}, and TST_{Acti}; and ii) Sleep $Disturbance_{PSQI,}$ $SOL_{PSQI,}$ $TST_{PSQI,}$ Daytime $Dysfunction_{PSQI}$ predicted HVLT-R delayed recall, 2-back accuracy, and Rise-time prospective memory after controlling for age, gender, vascular conditions, number of medications, use of sleep medications, depression, anxiety, and stress. Additional analyses were conducted to determine whether vigilance accounted for any observed relationships between sleep measures and retrospective and working memory performances. We also present associations between each variable in a correlation matrix.

Results

Participant Characteristics

From 200 participants who completed the study, actigraphy data on seven were lost due to recording malfunction, one participant chose not to wear the Actiwatch, and 19 participants were excluded due to sleep disorders (sleep apnoea: 13; restless legs syndrome: 4; insomnia:1; delayed sleep phase syndrome: 1). The mean age of the final sample (n = 173) was 73.78 (SD = 5.73 years), 65.3% were female, 55.5% had more than 12 years of education, and the average estimated intelligence (Test of Premorbid Function; Pearson Assessments, 2009) was in the average range at 109.37 (SD = 11.26).

Retrospective memory performance was average (see Table 1), and levels of depression, anxiety, and stress were comparable to normative data (Crawford et al., 2003).The average actigraphically-derived bedtime was 10:55pm (SD = 54.14 minutes), and the average Rise-time was 7:22am (SD = 48.07 minutes). Normal levels of sleep complaint were reported by 58% of the sample (i.e., Global PSQI ≤5). Other sleep parameters are listed in Table 1. Because the dependent variables (HVLT-R delayed recall, 2-back accuracy, and

Rise-time PM) were not correlated more than .7, (i.e., HVLT-R delayed recall and 2-back accuracy r = .27; HVLT-R delayed recall and Rise-time PM r = .22; and 2-back accuracy and Rise-time PM r = .16) the following analyses were not corrected for multiple comparisons.

(insert Table 1)

Prediction of Memory Performances

Retrospective memory. For HVLT-R Delayed Recall, in Step 1, age, gender, vascular conditions, number of medications, use of sleep medications, depression, anxiety, and stress explained 22.8% of the variance, R^2 = .23, F(8, 161) = 5.95, p<.001, with better performance associated with younger age (sr = -.21, p = .002), female gender (sr = .18, p = .010), and lower anxiety (sr = -.16, p = .019). In Step 2, $WASO_{Acti}$, SOL_{Acti} and TST_{Acti} explained an additional 4.8% of the variance, ΔR^2 = .05, ΔF(3, 158) = 3.52, p = .016, with longer TST_{Acti} and SOL_{Acti} being associated with poorer performance, sr = -.16, p = .020 and sr = -.13, p = .056, respectively (see Table 2). In contrast, using subjective sleep measures (Sleep $Disturbance_{PSQI}$, SOL_{PSQI}, TST_{PSQI}, $Daytime\ Dysfunction_{PSQI}$) in Step 2 did not explain additional variance, ΔR^2 = .02, ΔF(4, 149) = 0.72, p = .581 (see Table 3). We performed a further hierarchical regression to determine whether vigilance explained the relationship between actigraphic sleep and HVLT-R Delayed Recall. In Step 1, we added PVT lapses to the demographics, which in total explained 24.2% of the variance, R^2 = .24, F(9, 150) = 5.32, p<.001; however, PVT lapses did not contribute significant unique variance to the model, (sr = -.12, p = .108), and the amount of variance explained by the subsequent actigraphic variables in Step 2 remained much the same, 4.6% of the variance, ΔR^2 = .05, ΔF(3, 147) = 3.14, p = .027, with similar unique contributions from TST and SOL, sr = -.16, p = .025 and sr = -.12, p = .080, respectively.

Working memory. For 2-back accuracy, in Step 1, age, gender, vascular conditions, number of medications, use of sleep medications, depression, anxiety, and stress did not explain significant variance, R^2 = .09, F(8, 157) = 1.84, p = .074. In Step 2, $WASO_{Acti}$, SOL_{Acti} and TST_{Acti} explained an additional 4.6% of the variance, ΔR^2 = .05, F(3, 154) = 2.71, p = .047, although no variable contributed significant unique variance (Table 2). In contrast, subjective sleep ($Sleep\ Disturbance_{PSQI}$, SOL_{PSQI}, TST_{PSQI}, $Daytime\ Dysfunction_{PSQI}$) only accounted for an additional 2.4% of the variance in Step 2, ΔR^2 = .02, ΔF(4, 146) = 1.00, p = .411 (Table 3). We also performed another hierarchical regression to determine whether vigilance explained

the relationship between actigraphic sleep and 2-back accuracy. In Step 1, we added PVT total lapses to the demographics, which still only explained 8.6% of the variance, R^2 = .09, $F(9, 149) = 1.56$, p = .133, and the actigraphic variables in Step 2 explained the same amount of variance as previously (i.e., 4.6%), although the overall step became marginally non-significant probably due to loss of power from having fewer participants, ΔR^2 = .05, $\Delta F(3, 146) = 2.55$, p = .058.

Habitual prospective memory. For prospective memory accuracy, in Step 1, age, gender, vascular conditions, number of medications, use of sleep medications, depression, anxiety, and stress did not explain significant variance, R^2 = .05, $F(8, 161) = 1.08$, p = .378. In Step 2, $WASO_{Acti}$, SOL_{Acti} and TST_{Acti} did not explain significant additional variance, ΔR^2 = .03, $\Delta F(3, 158) = 1.78$, p = .152 (Table 2). Similarly, using Sleep $Disturbance_{PSQI,}$ $SOL_{PSQI,}$ $TST_{PSQI,}$ $Daytime\ Dysfunction_{PSQI}$ in Step 2 did not account for significant additional variance, ΔR^2 = .03, $\Delta F(4, 149) = 1.28$, p = .280 (Table 3). Although neither objective nor subjective sleep predicted prospective memory performance, significant zero-order correlations were observed between poorer prospective memory, and longer objective $WASO_{Acti}$ (r = -.16, p = .018) and longer subjective SOL_{PSQI}, , and (r = -.19, p = .007), (see Table 4 for correlations). Four of the 173 participants made no button presses upon rising from bed throughout the entire study period, which may indicate that they had forgotten or did not understand the instructions. After re-running the analyses without these 4 participants, prospective memory results were essentially unchanged.

(insert Table 2)

(insert Table 3)

(insert Table 4)

Discussion

The primary findings indicate that objective, but not subjective, sleep predicted memory performance. Specifically, after controlling for demographics, health, and mood, the collective contribution of actigraphically-measured longer WASO, SOL, and TST were predictive of poorer retrospective memory (HVLT-R delayed recall) and poorer working memory (2-back accuracy), with greater unique contributions coming from TST and SOL.

The inclusion of both objective and subjective sleep measures in a large sample of community-living older adults who were screened for self-reported sleep disorders, were the study's major strengths.

In demonstrating that longer objectively measured TST was negatively associated with memory performance, the present study extended previous research in older adults which has relied on *self-report* to associate sleep quantity with poorer memory (Miller et al., 2014). One possible explanation relating memory decline and age-related changes in sleep-wake control would be if they shared a common underlying aetiology; for example, age-related reductions in wake-promoting neurons, such as the cholinergic neurons of the nucleus basalis of Meynert, are implicated in both memory performance, and sleep-wake control (see Stern et al., 2015 for review). Furthermore, in older adults, self-reported longer sleep duration has been associated with other negative health outcomes, such as higher prevalence of stroke (Fang et al., 2014), and increased brain white-matter hyperintensity volumes (Ramos et al., 2014), which are associated with decreased memory performance (Silbert et al., 2008). The possibility that objectively measured, long TST in the current study indirectly reflected poor sleep quality was not supported by the current data, as the correlation between actigraphic time in bed and sleep efficiency (the proportion of time spent asleep compared to time spent in bed) was low (r = -.07, p = .361). However, such an association may be more likely to be observed in individuals with insomnia who, in an attempt to compensate for perceived lack of sleep, may stay in bed for longer periods, thus leading to lower sleep efficiency (Friedman et al., 1991). Alternatively, long sleep duration could reflect less restorative sleep in a way that is not detected by actigraphy, or the effect of long sleep may be mediated by sleep stage or other neurophysiological markers that occur during sleep, for example REM sleep or sleep spindles (Lafortune et al., 2014). A further theory suggests that in older adults, slow-wave sleep may in fact be deleterious for memory (Scullin, 2013). Synaptic downscaling has been proposed as a process that occurs during slow-wave sleep in which there is a pruning of synaptic weights that accrue over the waking day as a result of experiences (Tononi et al., 2003). It has been suggested that slow-wave sleep could have a negative impact on memory in older adults through a process of synaptic over-downscaling when the accrual of synaptic weights has decreased as a process of ageing but downscaling during slow-wave sleep is maintained (Scullin, 2013). However, support for this theory is difficult to gauge as sleep stages were not measured in the current study.

The current study also demonstrated a relationship between longer objective SOL and poorer memory, adding to the growing body of evidence suggesting that longer SOL is associated with lower performance in other domains, such as attention allocation (Yaffe et al., 2007). The current finding was not accounted for by mood or age, which has previously been associated with SOL (Naismith et al., 2011; Ohayon et al., 2004). It has been speculated that difficulties with sleep onset and maintenance with age may reflect, as is observed with other aspects of sleep, broader changes in cortical volume and structure (Buysse et al., 2005), and for this reason may be associated with reduced memory performance. Alternatively, preliminary findings have indicated an association between longer subjective SOL and greater beta-amyloid burden in healthy adults (Branger et al., 2015 [conference abstract]; Spira et al., 2013). However, whether underlying Alzheimer's disease pathology could explain the relationship between longer objective SOL and poorer memory in the current study are speculative, and further research is required to understand the potential mechanism.

Lack of a strong relationship between prospective memory and sleep may be due to the nature of the prospective memory task, in a naturalistic setting. Because older adults often outperform younger adults in naturalistic settings (Rendell et al., 2000), this may indicate that older people with subtle memory decline may be able to modify their behaviour in naturalistic settings, thereby mitigating cognitive impacts that sleep may have on performance. The prospective memory task (i.e., pressing the Actiwatch Event Marker button within 10 minutes of rising from bed) may, however, have been affected by sleep inertia, which has been shown to occur in older adults over the first 30 minutes following waking (Silva et al., 2008), and could explain the reason sleep did not predict prospective memory performance.

The current study found no association between memory and subjective sleep quality or quantity, which is consistent with some (Sutter et al., 2012), but not other research (Miller et al., 2014; Schmutte et al., 2007). Previous studies are likely to have included people with sleep disorders (Miller et al., 2014; Schmutte et al., 2007; Waller et al., 2015), such as sleep apnoea, which is independently associated with memory impairment (Wallace et al., 2013), and may account for some of the associations previously reported between subjective sleep measures and memory performance. Furthermore, a difficulty with interpreting subjective measures of sleep is that they do not always correlate highly with objective measures of sleep, particularly in older adults (Buysse et al., 2008); and this was also observed in the current study.

The current findings are important because of the significant inconsistencies and lack of consensus in previous research about how sleep predicts memory in older adults, particularly in non-clinical populations. Although we attempted to control for a variety of potential confounds, limitations include the reliance on self-reported presence of sleep disorders, number of medications, and vascular medical conditions. It is therefore possible that the sample contained people with undiagnosed sleep disorders. However, sleep disorders are commonly, although not always associated with daytime dysfunction (as indicated by the PSQI), of which there were not high levels in this sample, nor did daytime dysfunction predict memory performances. Both of these observations would indicate that the association between actigraphic sleep indices and memory performances are less likely to be explained by the presence of sleep disorders. A further limitation is that cognitive testing occurred at different times of day across participants and should be controlled in further studies even though as noted in our statistical method section, controlling for vigilance, which may change at different times of the day, did not change the study results. Due to the cross-sectional design, the present study cannot confidently address the causal directions on reported associations. This suggests the importance of further longitudinal research to determine trajectories of change over time, as understanding factors which affect memory decline will have implications for the early detection and treatment of people at risk of developing memory impairment with advancing age. The relevance of the findings is highlighted when considering the body of research demonstrating a relationship between sleep disturbance and Alzheimer's disease (Peter-Derex et al., 2015). Further development of behavioural and neurobiological models will be useful in explaining the mechanism behind the current findings. For example, future studies may be able to test whether there is a causal association between longer sleep and poorer memory by restricting sleep (minimally) in older adults with long sleep duration to see whether memory performances improve.

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Tables

Table 1

Descriptive Statistics on Memory, Sleep, Health, and Mood

	Mean/Median (SD/IQR) [a]	
HVLT-R Total Trials 1-3	24.83	(5.28)
HVLT-R Total T-score	51.47	(10.38)
HVLT-R Delay ^{RSQ}	9.00	(4.00) a
HVLT-R Delay T-score	52.00	(18.00) a
2-back accuracy $^{O.RLN}$ (Total)	48	(9.00) a
PM % accuracy	57.16	(26.20)
$WASO_{Acti}$ $^{O.SQ}$ (mins)	47.86	(22.04) a
SOL_{Acti} $^{O.LN}$ (mins)	6.14	(7.93) a
TST_{Acti} ^{O} (hrs: mins)	7:16.79	(1:12.36) a
$Sleep\ Disturbance_{PSQI}$ (subscale score)	1.23	(0.50)
SOL_{PSQI} $^{O.SQ}$ (mins)	15.00	(20.00) a
TST_{PSQI} (mins)	418.58	(65.98)
$Daytime\ Dysfunction_{PSQI}$ (subscale score)	0.59	(0.58)
Global PSQI	5	(5) a
Number of Vascular conditions	1.29	(1.19)
Number of Medications ^{SQ}	3.00	(4.00) a
Sleep medications (% Y)	(20.8%)	
Depression $^{O.LN}$	2.00	(4.00) a
Anxiety $^{O.LN}$	0.00	(4.00) a
Stress ^{LN}	4.00	(8.00) a

[a] Medians and Interquartile ranges reported for skewed variables. HVLT-R: Hopkins verbal Learning Test – Revised; WASO = wake after sleep onset; SOL = sleep onset latency, TST = total sleep time. Sleep medications (% Y) = proportion of sample who endorsed use of sleep medication over the past month; Acti = actigraphy; PSQI = Pittsburgh Sleep Quality Index; Note. The following transformations were applied to variables used in analysis: ^{O} = outlier transformed; ^{R} = reflected prior to transformation; ^{SQ} = square root transformed; ^{LN} = natural log transformed.

Table 2

Summary of Regression and Hierarchical Regression Analyses for Objective Measures of Sleep, Demographic, Health, and Mood Variables Predicting Retrospective Memory, Working Memory, and Prospective Memory

Dependent Variable	Predictor Block [a]	Change Statistics		Overall model						
		ΔR^2	ΔF	Predictors	β	sr	r	R^2	df	F
HVLT-R Delayed Recall [RSQ]	1. Demographics, Health, and Mood	.23	5.95***	Age	-.23**	-.21	-.30	.23	(8, 161)	5.95
				Gender (F)	.19*	.18	.23			
				Vascular	-.17	-.14	-.27			
				Meds [SQ]	-.08	-.06	-.28			
				Sleep Meds	.03	.03	.02			
				Depression [O.LN]	.09	.08	-.07			
				Anxiety [O.LN]	-.18*	-.16	-.21			
				Stress [LN]	-.13	-.12	-.13			
	2. Objective Sleep	.05	3.52*	WASO [O.SQ]	-.07	-.06	-.16	.28	(3, 158)	5.50
				SOL [O.LN]	-.15	-.13	-.19			
				TST [O]	-.17*	-.16	-.12			
2-back accuracy [O.RLN]	1. Demographics, Health, and Mood	.09	1.84	Age	-.15	-.14	-.14	.09	(8, 157)	1.84
				Gender (F)	.08	.08	.09			
				Vascular	-.10	-.08	-.08			
				Meds [SQ]	.07	.05	-.03			
				Sleep Meds	.13	.12	.12			
				Depression [O.LN]	.19*	.17	.09			
				Anxiety [O.LN]	-.14	-.12	-.10			
				Stress [LN]	-.10	-.09	-.06			
	2. Objective Sleep	.05	2.71*	WASO [O.SQ]	-.10	-.09	-.12	.13	(3, 154)	2.12
				SOL [O.LN]	-.13	-.12	-.16			
				TST [O]	-.15	-.14	-.09			
Rise-time PM accuracy	1. Demographics, Health, and Mood	.05	1.08	Age	.06	.05	<.01	.05	(8, 162)	1.08
				Gender (F)	.02	.02	.02			
				Vascular	-.04	-.03	-.11			
				Meds [SQ]	-.17	-.13	-.16			
				Sleep Meds	.05	.05	.02			
				Depression [O.LN]	.16	.14	.10			
				Anxiety [O.LN]	-.05	-.05	-.03			
				Stress [LN]	-.06	-.06	-.04			
	2. Objective Sleep	.03	1.78	WASO [O.SQ]	-.11	-.10	-.16	.08	(3, 159)	1.29
				SOL [O.LN]	-.05	-.04	-.11			

			TST [O]	.09	.08	.09		

*p < .05, **p < .01, ***p < .001. [a] For each dependent variable, the first block shows the contribution of demographics, medical factors, and mood, and the second block shows the additional contribution of objective sleep. Note. The degrees of freedom reported in the table vary because of missing data on some variables. Vascular = number of vascular conditions; Meds = number of medications, Sleep Meds = use of sleep medication over the past month; WASO = wake after sleep onset; SOL = sleep onset latency; TST = total sleep time. The following transformations were applied to variables used in analysis: [O] = outlier transformed; [R] = reflected prior to transformation; [SQ] = square root transformed; [LN] = natural log transformed. To aid interpretation a negative sign for coefficients has only been used when the relationship is negative, regardless of whether the variable was reflected in transformation.

Table 3

Summary of Regression and Hierarchical Regression Analyses for Subjective Measures of Sleep, Demographic, Health, and Mood Variables Predicting Retrospective Memory, Working Memory, and Prospective Memory

Dependent Variable	Predictor Block [a]	Change Statistics		Overall model						
		ΔR^2	ΔF	Predictors	β	sr	r	R^2	df	F
HVLT-R Delayed Recall [RSQ]	1. Demographics, Health, and Mood	.23	5.65***	Age	-.23**	-.21	-.30	.23	(8, 153)	5.65
				Gender (F)	.19*	.18	.23			
				Vascular	-.17	-.14	-.27			
				Meds [SQ]	-.08	-.06	-.28			
				Sleep Meds	.03	.03	.02			
				Depression [O.LN]	.09	.08	-.07			
				Anxiety [O.LN]	-.18*	-.16	-.21			
				Stress [LN]	-.13	-.12	-.13			
	2. Subjective Sleep	.02	0.72	Disturbance	-.03	-.03	-.05	.24	(4, 149)	3.98
				SOL [O.SQ]	-.08	-.07	-.07			
				TST	-.02	-.02	.03			
				Day-Dysf.	-.11	-.09	-.14			
2-back accuracy [O.RLN]	1. Demographics, Health, and Mood	.09	1.75	Age	-.15	-.14	-.14	.09	(8, 150)	1.75
				Gender (F)	.08	.08	.09			
				Vascular	-.10	-.08	-.08			
				Meds [SQ]	.07	.05	-.03			
				Sleep Meds	.13	.12	.12			
				Depression [O.LN]	.19*	.17	.09			

				Anxiety [O.LN]	-.14	-.12	-.10			
				Stress [LN]	-.10	-.09	-.06			
	2. Subjective Sleep	.02	1.00	Disturbance	-.06	-.05	-.01	.11	(4, 146)	1.50
				SOL [O.SQ]	<.01	<.01	.02			
				TST	-.12	-.11	-.11			
				Day-Dysf.	-.09	-.07	-.03			
Rise-time PM accuracy	1. Demographics, Health, and Mood	.05	1.03	Age	.06	.05	<.01	.05	(8, 153)	1.03
				Gender (F)	.02	.02	.02			
				Vascular	-.04	-.03	-.11			
				Meds [SQ]	-.17	-.13	-.16			
				Sleep Meds	.05	.05	.02			
				Depression [O.LN]	.16	.14	.10			
				Anxiety [O.LN]	-.05	-.05	-.03			
				Stress [LN]	-.06	-.06	-.04			
	2. Subjective Sleep	.03	1.28	Disturbance	<.01	<.01	-.02	.08	(4, 149)	1.12
				SOL [O.SQ]	-.18*	-.17	-.19			
				TST	.03	.03	.04			
				Day-Dysf.	-.01	-.01	.05			

*p < .05, **p < .01, ***p < .001. [a] For each dependent variable, the first block shows the contribution of demographics, medical factors, and mood, and the second block shows the additional contribution of subjective sleep. Note. The degrees of freedom reported in the table vary because of missing data on some variables. Vascular = number of vascular conditions; Meds = number of medications, Sleep Meds = use of sleep medication over the past month; Disturbance = Sleep disturbance; SOL = sleep onset latency; TST = total sleep time; Day-Dysf. = Daytime dysfunction. The following transformations were applied to variables used in analysis: [O] = outlier transformed; [R] = reflected prior to transformation; [SQ] = square root transformed; [LN] = natural log transformed. To aid interpretation a negative sign for coefficients has only been used when the relationship is negative, regardless of whether the variable was reflected in transformation.

Table 4

Correlations between Objective Memory, Sleep, Demographics, Mood and Health in Participants

	HVLT RSQ	2back O.RLN	PM	WASO Acti O.SQ	SOL Acti O.LN	TST Acti O	Age	Gend.	Vasc.	Meds SQ	Sleep Meds	Dep O.LN	Anx O.LN	Stress LN	Dist. PSQI	SOL PSQI O.SQ	TST PSQI	Dysf. PSQI	PVT O.LN
HVLT RSQ	--																		
2back O.RLN	.27***	--																	
PM	.22**	.16*	--																
WASO Acti O.SQ	-.16*	-.12	-.16*	--															
SOL Acti O.LN	-.19**	-.16*	-.11	.46***	--														
TST Acti O	-.12	-.09	.09	-.19**	-.19**	--													
Age	-.30***	-.14*	<.01	-.02	-.04	.12	--												
Gend. (F)	.23**	.09	.02	-.05	-.13*	.23**	-.11	--											
Vasc	-.27***	-.08	-.11	.10	.07	.03	.17*	-.16*	--										
Meds SQ	-.28***	-.03	-.16*	.12	-.01	.11	.36***	-.11	.57***	--									

Sleep Meds	.02	.12	.02	.11	.04	.20**	.05	.20**	<.01	.14*	--							
Dep O.LN	-.07	.09	.10	.13	.02	-.13*	.01	-.18**	.09	.07	-.04	--						
Anx O.LN	-.21**	-.10	-.03	.20**	.05	.03	.06	.04	-.02	.09	.07	.33***	--					
Stress LN	-.13	-.06	-.04	.09	.05	<.01	-.13	.04	-.02	-.02	.07	.28***	.34***	--				
Dist. PSQI	-.05	-.01	-.02	-.18**	.05	.09	-.12	-.01	.04	.10	.25***	.11	.13*	.13	--			
SOL PSQI O.SQ	-.07	.02	-.19**	.16*	.24**	.05	-.02	.17*	.05	.07	.21**	-.07	.07	.22**	.11	--		
TST PSQI	.03	-.11	.04	.02	.01	.18*	-.15*	.07	-.04	-.04	-.23**	.03	.04	-.07	-.03	-.18*	--	
Dysf. PSQI	.14*	-.03	.05	-.01	.01	-.05	.10	-.09	.11	.15*	.14*	.45***	.11	.16*	.14*	-.10	.09	--
PVT O.LN	-.23**	-.06	-.10	.03	.06	.04	.28***	-.02	.20**	.19**	.01	.10	-.02	.16*	-.03	.03	.02	.15*	--

*p < .05, **p < .01, ***p < .001. HVLT: HVLT-R delayed recall; 2back: n-back, 2-back accuracy; $Dist._{PSQI}$ = PSQI Sleep Disturbance subscale; $Dysf._{PSQI}$ = PSQI Daytime Dysfunction subscale; Dep = Depression; Anx = Anxiety; Vasc = number of vascular conditions; Meds = number of medications; Sleep Meds = use of sleep medications over the past month (positive association = association with use of sleep medications, negative association = association without use of medications; PVT (number of PVT lapses > 500ms). For gender, positive associations = greater

relationship with women, negative associations = greater relationship with men. The following transformations were applied to variables used in analysis: ^{O} = outlier transformed; ^{R} = reflected prior to transformation; ^{SQ} = square root transformed; ^{LN} = natural log transformed. To aid interpretation a negative sign for coefficients has only been used when the relationship is negative, regardless of whether the variable was reflected in transformation.