

# Effects of sleep deprivation on component processes of working memory in younger and older adults <sup>FREE</sup>

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## Abstract

### Study Objectives

Working memory (WM) has been described as a multicomponent process, comprised of the following: attention-driven encoding, maintenance and rehearsal of information, and encoding to and retrieval from episodic memory. Impairments can affect higher-order cognitive processes and many everyday functions. The impact of sleep changes on these cognitive processes across the life span needs to be investigated. The aim of the current study is to examine the effects of sleep deprivation on component processes of WM, comparing younger and older adults across verbal and visuospatial modalities.

### Methods

Thirty-one younger adults (19–38 years) and 33 older adults (59–82 years) attended two counterbalanced sleep protocols: a regular night of sleep followed by testing the next day (normally rested condition), and 36 hr of total sleep deprivation (TSD), followed by testing (TSD condition). Participants completed matched versions of verbal and visuospatial WM tasks across conditions.

### Results

Younger adults significantly outperformed older adults on encoding and displacement component processes, for both verbal and visuospatial WM. Following TSD, younger adults showed a significantly larger drop compared with older adults in verbal encoding and in visuospatial displacement. A main effect of condition was observed for verbal displacement.

### Conclusions

Differences were observed in the performance of younger and older adults on component processes of WM following TSD. This suggests that TSD can have differential effects on each component process when younger and older adults are compared, in both verbal and visuospatial tasks. Understanding this profile of changes is important for the development of possible compensatory strategies or interventions and the differentiation of clinical and healthy populations.

## Statement of Significance

There is increasing recognition that sleep plays a vital role in cognitive aging. Given decreases in sleep quantity with aging, we explored this relationship experimentally with sleep deprivation. The sleep–cognition interaction is particularly underexplored in working memory. Despite being a complex, multicomponent process underpinning many everyday activities (e.g., reading and driving), prior studies examine only global performance. In contrast, this is the first study to examine component processes of both verbal and spatial working memory in both older and younger adults with a sleep deprivation challenge. Younger adults were more vulnerable to sleep deprivation, but only on specific working memory components. By measuring working memory at a more nuanced level, we can further enrich our understanding of the sleep–cognition interaction in aging.

## Introduction

Across the lifespan, the cognitive consequences of sleep loss have provided a complex context for research, particularly in the domain of working memory (WM) [1]. WM enables an individual to temporarily maintain a limited amount of information in an active state to support various cognitive processes such as reasoning and problem solving [2]. A number of theoretical models describe WM as a process comprised of multiple components within a complex system, including attention-driven encoding, the rehearsal and maintenance of information, and the encoding to and retrieval from episodic memory [3–5]. Impairments in WM can have significant effects on higher-order cognitive processes such as decision-making, planning, and organization, but also on functional abilities including reading, performing arithmetic, and even keeping track of ideas in a conversation [6, 7]. Therefore, WM plays a vital role in everyday functioning.

It is well documented that WM declines throughout ageing [2, 8]. Clinically, older adults frequently report difficulty keeping track of relevant information and maintaining attention on specific events for periods of time, particularly in the face of distraction [9]. WM performance is heavily reliant on these skills. However, studies comparing younger and older adults have traditionally used global measures of WM, providing a composite score for overall performance. Although these studies consistently show poorer WM performance in older adults [1, 10], how each of the component processes of WM is affected by age remains to be explored. Age-related impairments in global scores could be the result of individual or multiple component deficits, whereas there may be sparing of other cognitive processes. Understanding the profile of changes in WM components is important for the development of possible compensatory strategies or interventions, and the differentiation of clinical and healthy populations. This becomes particularly relevant when considering age-related declines in older populations and their impact on the quality of life.

One possible contributing factor to declines in cognition with aging may be changes to sleep. Sleep becomes more shallow and fragmented, even in healthy aging [11–14], and these changes have been associated with poorer cognitive performance [15–17]. In younger adults, sleep loss has been shown to impair WM performance [18]. The general consensus amongst the sleep deprivation literature is that impairments in WM can be seen from 20 hr or more of total sleep deprivation (TSD) [6]; however, some studies have shown no such effects [19]. As in the aging literature, the predominant reliance on global WM measures in the majority of studies could be contributing to this inconsistency in findings. Results from an investigation using a verbal task modeling, three distinct processes of WM, showed that the limited capacity updating and maintenance parameter, perhaps reflecting rehearsal span, i.e., the amount of information simultaneously kept in WM, was significantly affected by 42 hr of sleep deprivation in younger adults [20]. The attention–driven stimulus encoding component was also affected, although to a smaller degree. The study of Turner et al. utilized verbal information. To the best of our knowledge, no other sleep studies investigating WM for visuospatial stimuli have been published, in either young or older adults. The absence of such studies is significant in that the relative impact of disrupted brain function on stimulus encoding and WM updating and maintenance processes may differ by stimulus modality [21, 22].

Whether these effects of sleep loss on specific component processes of WM are generalizable to older adults is yet to be explored. This is of interest because several studies have shown that older adults are able to maintain a more constant level of attention during sleep deprivation compared with younger adults, whose performance significantly decreases following TSD [23]. This age-by-sleep deprivation interaction has not been examined beyond sustained attention and thus remains unexplored in the context of component processes of WM. Nor has the age-by-sleep deprivation interaction been compared across spatial and verbal modalities. The work of Turner et al. [20] highlights the need for the inclusion of a visuospatial task to provide a more complete picture of WM performance and individual variability. Disentangling these processes could help us to improve consensus around the impact of sleep deprivation on age-related WM impairment.

Therefore, three key gaps in the literature are evident as follows: (1) almost all studies examining the effects of sleep deprivation on WM have reported global measures of performance, with very few attempting to dissect WM into its component parts; (2) only a handful of studies have

examined sleep loss–related impacts on WM in older adults and none have examined an age  $\times$  sleep deprivation interaction in WM; and (3) no studies, in any age group, have concurrently examined both verbal and visuospatial WM in the context of sleep deprivation. We aimed to address these gaps by providing a better understanding of the interaction of age and sleep deprivation on component processes of WM, comparing younger and older adults across both verbal and visuospatial WM modalities. To achieve this, we administered a computer-based task enabling computational modeling of individual performance data to provide a measure of three component processes of WM: stimulus encoding, updating and maintenance, and efficiency of encoding into episodic memory [21]. We utilized a TSD model of sleep loss, rather than a more subtle sleep restriction or fragmentation model, since this was the first attempt to examine sleep loss effects on component processes of WM in older adults. We hypothesized as follows: (1) older adults would have a poorer global WM performance following TSD compared with younger adults in both verbal and visuospatial WM domains and (2) of the three component processes, attention–driven stimulus encoding would be most affected in younger adults and the updating and maintenance component most affected in older adults, and this would be the case on both the verbal and visuospatial versions of the task.

## Methods

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### Ethics statement

The data presented in the current study were collected as part of a larger study conducted at the University of California, San Diego [8, 24], which was approved by the local Human Research Protections Program and the Veterans Affairs San Diego Healthcare System R&D Committee.

### Participants

Sixty-four individuals were included in the final analysis for this study, including 31 younger adults (age range: 19–38 years,  $M = 28.00$ ,  $SD = 4.75$ , 61.3% female) and 33 older adults (age range: 59–82 years,  $M = 68.24$ ,  $SD = 5.86$ , 75.8% female). All participants provided written informed consent and underwent extensive medical and psychiatric screening. Participants were included in the study on meeting the following criteria: (1) were right handed, (2) were at least 59 years of age or between 18–39 years of age, (3) had at least 12 years of education, (4) had a consistent sleep–wake schedule that consisted of 6–9 hr total sleep time each night and no more than one nap per week under 1 hr in length (7–9 hr total sleep time was required for younger adults and no naps), (5) had a nonpolarized chronotype, defined as having a Horne–Ostberg Morningness–Eveningness Questionnaire score between 31–69 [25], and (6) had no cognitive impairment (Dementia Rating Scale score  $<130$  with a memory subscale score  $\geq 22$  for the older adult group). All participants were screened for personal or immediate family history of Axis I psychopathology as determined by the SCID-I [26], medical conditions, primary sleep disorders, current consumption of nicotine, psychotropic medications or illicit substances, and/or regular consumption of more than two alcoholic beverages or 400 mg of caffeine per day. Participants were asked to abstain from alcohol and caffeine consumption commencing 3 days prior to the start of their laboratory stay and for the duration of their stay in the laboratory. Habitual sleep schedule needed to include a bedtime between 10:00 pm and 12:00 am and wake time between 6:00 am and 8:00 am. This was consistent across both age groups, in order to minimize confounding effects of varying habitual sleep parameters on baseline WM performance, and to reflect current population-level bedtime averages. For 1 week prior to the commencement of the study, participants were asked to comply with a sleep–wake schedule based on their habitual sleep schedule. To monitor this, each person completed a daily sleep diary to document their sleep and wake times and wore an actigraph as an objective measure of sleep obtained [24].

In the original data collection study, a total of 110 participants were consented. Of those who were enrolled, but did not complete the study, 18 withdrew voluntarily for personal reasons (e.g., inability to keep the time commitment of the study) and 28 were excluded (15 for health or psychiatric exclusions; four related to unreported sleep disorders found during screening: three older adults with obstructive sleep apnea and one older adult with periodic limb movement with arousal index [PLMA]).

### The working memory task

The Continuous Recognition Memory Test (CRMT) is based on the Continuous Paired Associates Test developed for the computational modeling of WM [27, 28]. Previous studies employing this model have been published and detailed manipulation and calculation of model parameters can be found in other articles [29, 30]. These studies consistently demonstrate the utility of the model in differentiating the three component processes of WM described in the Introduction (attention–driven stimulus encoding, updating and maintenance, and efficiency of encoding into episodic memory). The CRMT is composed of multiple test–study trials. A target stimulus is presented for 3 s, which the participant is required to remember. This target is later presented along with three foils in a test trial, and the person is required to recognize the item and respond accordingly. Test trials remained on

the screen until the participant responded. The number of test-study trials between the presentation of a stimulus and its test is an item's lag length. Recognition of a stimulus may be tested immediately after its presentation (lag 0) or up to four lags after the stimulus was presented, and this varies throughout the task. Eight trials of each of the five lag lengths (lag 0–lag 4) were pseudorandomly presented. Each version (versions 1 and 2) of both the verbal and visuospatial tasks of the CRMT usually takes 5 to 6 min to complete.

## Verbal CRMT

The verbal CRMT (vbCMRT) used for this study followed the CRMT format described above and was identical to that used by Turner et al. [20]. Participants are presented with a single, pronounceable, five-letter nonsense word, which they are instructed to remember. During the test conditions, this target word is presented alongside three similar nonsense word foils. The vbCMRT requires the participants to identify which word they previously studied. For the verbal task, the following instructions appear on the screen for the participant to read: “This is a working memory task. In this experiment, you will see some words that don't mean anything. When a nonword is presented individually, look at it carefully, and try to remember it. Later in the test you will be shown the same nonword mixed in with some similar nonwords. You will be asked to press the button that goes with the nonword you have seen before. Sometimes you will see a star in the center of the screen. Please look at the star and wait for the test to continue. Press any key to begin the practice test.” The participant is provided with automated feedback on whether they responded correctly or incorrectly in the practice trials. Once this is complete, the following message appears: “Very good! Remember each nonword very carefully, because sometimes you won't be asked to identify it until much later in the test. Press any key to begin.” The test trials then begin.

## Visuospatial CRMT

The visuospatial CRMT (vsCMRT) assesses the ability to recognize the orientation of a line drawing across intervening lags [31]. The vsCMRT has the same trial and lag structure as the vbCMRT task, except that in the vsCMRT participants are provided feedback to maintain motivation [31]. The visual stimuli for the spatial task are nonsense drawings and other visual figures sensitive to right hemispheric lesions [22, 28, 32, 33]. The nonsense drawings, which do not resemble any objects, shapes, or familiar forms, help us to control for the use of language cues and minimizes the reliance on other verbal memory processes that could assist with task performance [32]. The figural drawings are more familiar shapes, which can be associated with one another through Gestalt principles to form more complex shapes [34]. The drawings and figures were digitally scanned in a paper format, then modified using Adobe Photoshop 7.0 to include a rotational–spatial component through the following process [31]. Foils for each target design were created by rotating the targets intervals of 30° over a complete circle, skipping the 180 rotation, which might produce foils identical to the target [31]. See Figure 1 for an example of a Spatial CRMT test trial. For the visuospatial task, the following instructions appear: “This is a working memory task. In this experiment you will see some shapes inside a black box. When a shape is presented individually, please look at it carefully, and try to remember the position of the shape. Later in the test you will see the same shape in four positions. You will be asked to press the button that goes with the shape that is in the same position as the one you saw before. Sometimes you will see a star in the center of the screen. Please look at the star and wait for the test to continue. Press any key to begin the practice test.” As with the verbal task, the participant is provided with automated feedback on whether they responded correctly or incorrectly in the practice trials. Once this is complete, the following message appears: “Very good! Remember the position of each shape very carefully, because sometimes you won't be asked to identify it until much later in the test. Press any key to begin.” The test trials then begin.

### Figure 1.



The visuospatial WM task. The participant is asked to remember each single line drawing presented. The line drawing is then presented with three foils at intervening lags.

Forty items in total are presented in both the verbal and visuospatial CMRTs, each item is presented only once, and there are eight items in each lag condition. Alternate versions of both tasks were used to allow for the test–retest design between sleep deprived and normally rested (NR) conditions. The verbal task has previously been reported to have a parallel forms reliability coefficient of  $r = .79$ , with equivalent difficulty and variance [31].

The computational model employed in this study, as described by Brown and colleagues [22], estimates three distinct parameters of WM:

- (1) ENCoding (ENC): the probability that a newly presented item will be encoded into WM. This parameter encompasses all attentional, sensory, perceptual, and recoding processes that transform the stimulus into a WM representation. This parameter thus models the gating function of attention.
- (2) DISplacement (DIS): the probability that an item being rehearsed will be knocked out or displaced from short-term WM when a new item is encoded. The DIS parameter represents the limited capacity of WM. Not all items presented are able to be maintained in WM. Although there is no consensus about the cause of WM's limited capacity, common theories include resource, interference, and slot buffer models [27, 35–38]. If the slot buffer model of WM is assumed, the inverse of displacement represents the span capacity of WM (i.e., when WM capacity is full, a new item must displace an older item before the new item can be placed into WM).
- (3) Episodic (E): the amount of episodic information that is encoded for each second an item is rehearsed. It models memory processes involved in the correct recall of an item, even though its memory representation is not activated in WM at the time it is tested [22].

Each WM parameter value contributes to the performance curve across increasing task difficulty (Lag 0–Lag 4) [20]. Poor encoding negatively affects performance at Lag 0; displacement (or inversely, WM span) has its greatest impact on short lags beyond Lag 0, whereby higher displacement equates to worse performance; and a lowered episodic parameter value represents reduced performance at the longer lags.

We used a derivative-free, multidimensional, direction set method to estimate parameter values that best fit the model to each participant's total correct score at each lag [39]. The estimation method minimized a log-likelihood dispersion score, which was used to assess adequacy of fit [22, 40].

## Procedure

The study included two sets of counterbalanced appointments for each of the participants, and these were scheduled approximately 2 weeks apart. One comprised a regular night of sleep and testing the following day (NR condition). The other consisted of 32 hr of TSD, followed by testing (TSD condition) at the same time as the NR condition.

Participants underwent a full polysomnogram to screen out possible sleep disorders prior to their first experimental condition in the laboratory. Given the increased prevalence of sleep disorders in older adults, we excluded anyone with an apnea–hypopnea index  $\geq 15$ , or a PLMA  $\geq 15$ . The NR

condition comprised two consecutive nights in the laboratory. The first provided an opportunity for readaptation to the laboratory environment and equipment. On waking (day 2), they could go home and return to the lab 2 hr prior to their habitual bedtime for the baseline night of sleep (night 2). Bedtimes and rise times were consistent with participants' habitual sleep schedules. For the TSD condition, participants arrived at the laboratory at 20:00 hr. Research staff monitored participants throughout the protocol. Strenuous exercise, napping, and alcohol or caffeine consumption were not permitted and time cues were removed. Participants could interact with researchers to remain awake, and they were free to move around and engage in activities that were provided. Participants completed matched versions of the verbal and visuospatial WM tasks when NR and during TSD. Testing occurred at the same time of day on both occasions (6 hours post habitual wake time) and test order was counterbalanced across subjects.

## Data analysis

Statistical analyses were conducted using SPSS version 20.0 software. Baseline sleep parameters were compared across the groups. These parameters were then evaluated in the context of all significant results to determine whether they influenced any of the observed effects. We correlated each significant sleep measure with each of the verbal and visuospatial task parameters, separately for each group. In addition, a chi-square analysis was conducted to examine whether the ratio of males to females was significantly different between the older and younger people. Rank transformation of behavioral data (performance at lag 0–lag 4) and model parameters (ENC, DIS, and E) was used to overcome assumption violations and is consistent with other group analyses of WM parameters [21, 22, 41]. Parametric analyses were performed on these rank transformations [41]. To examine the behavioral data and assess the effects of TSD across lag conditions in each group, we ran a  $2 \times 2 \times 5$  mixed model ANOVA on rank-ordered data (between subject factor: Group—older adults vs younger adults; within subject factors: Night—NR vs TSD, Lag condition—lag0 to lag4). We ran a  $2 \times 2$  ANOVA to look at the effects of Group and TSD on each of the WM model parameters (between subject factor: Group—older adults vs younger adults; within subject factor: Night—NR vs TSD). *t*-Tests were used to follow-up significant interactions of main effects with more than two levels. Type I error was protected for each analysis at  $p = .05$ . Effect sizes are reported either as partial  $\eta^2$  or as Cohen's *d* values.

## Results

Descriptive statistics are reported in Table 1. Mean SE% based on actigraphy for the week prior to baseline and night prior measures based on PSG (TST, SE%, %REM, and %Delta) were significantly different between the two groups (all  $p < .05$ , Cohen's *d* ranged from 0.61 to 1.09) There was no significant difference between the proportion of males:females between the younger and older adult groups,  $\chi^2(1, N = 64) = 1.56, p = .212$ .

**Table 1.**

Baseline sleep parameter values for younger and older adults, prior to study commencement

	Younger adults		Older adults	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
PSG—night prior				
TST <sup>†</sup>	429.98	45.03	394.25	50.28
SE% <sup>†</sup>	90.52	7.92	85.03	8.25
%REM <sup>†</sup>	24.76	5.16	20.75	6.52
%Delta (N3 + N4) <sup>†</sup>	17.95	7.64	8.90	8.92
Actigraphy—week prior				
TST	441.77	40.70	433.56	42.79
SE% <sup>†</sup>	91.33	5.18	93.93	3.17

PSG = polysomnography; TST = total sleep time; SE% = sleep efficiency percentage; %REM = rapid-eye movement sleep percentage; CI = confidence interval;

<sup>†</sup>Significant difference between groups, after correction for multiple comparisons.

## Verbal working memory task

No statistically significant interactions were observed in the behavioral data for verbal WM. The main effects for group ( $F(1, 53) = 13.32, p = .001, \eta_p^2 = .20$ ), condition ( $F(1, 53) = 8.73, p = .005, \eta_p^2 = .14$ ), and lag ( $F(4, 50) = 79.10, p < .001, \eta_p^2 = .86$ ) were all significant. The lag effect was driven by a significant performance drop between lag0–lag1 overall ( $p < .001$ ). Descriptive statistics are shown in Table 2, and group performance at each lag for both conditions is shown in Figure 2.

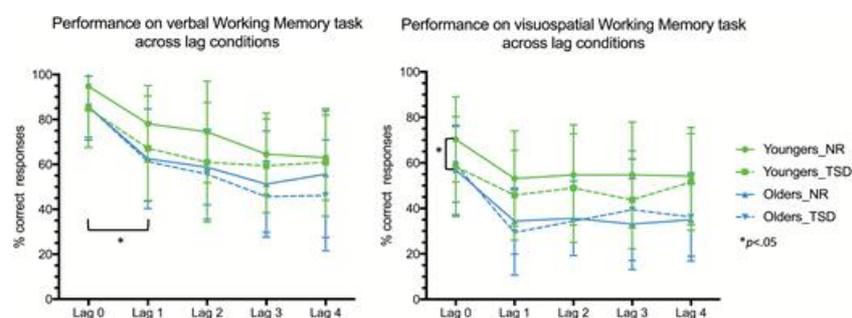
**Table 2.**

Verbal task mean performance scores at each lag condition while normally rested and following total sleep deprivation, by group

	Younger adults				Older adults			
	NR		TSD		NR		TSD	
	<i>M</i> ( <i>SD</i> )	95% CI						
Lag0	95.42 (9.56)	[91.85, 98.99]	84.58 (18.48)	[77.68, 91.48]	84.13 (13.95)	[78.50, 89.77]	78.75 (24.82)	[69.48, 88.02]
Lag1	78.75 (16.46)	[72.60, 84.90]	67.50 (25.55)	[57.96, 77.04]	61.54 (20.59)	[53.22, 69.86]	55.83 (21.46)	[47.82, 63.85]
Lag2	70.83 (22.34)	[62.49, 79.18]	61.25 (26.33)	[51.42, 71.08]	53.37 (18.89)	[45.73, 61.00]	47.92 (23.23)	[39.24, 56.59]
Lag3	64.17 (19.35)	[56.94, 71.39]	57.92 (20.63)	[50.21, 65.62]	50.48 (21.65)	[41.74, 59.22]	43.33 (19.62)	[36.01, 50.66]
Lag4	63.33 (19.68)	[55.99, 70.68]	59.58 (23.83)	[50.69, 68.48]	51.92 (27.32)	[40.89, 62.96]	39.58 (24.80)	[30.32, 48.84]

NR = normally rested; TSD = total sleep deprivation; CI = confidence interval.

**Figure 2.**



Group performance scores for younger and older adults on the verbal working memory task (left) and the visuospatial working memory task (right), at each lag, in both NR and sleep-deprived (TSD) conditions. Error bars represent the standard deviation. Chance level performance was defined as being 25% correct responses or less.

For the Encoding parameter, a significant interaction between condition and group was found ( $F(1, 53) = 7.15, p = .01, \eta_p^2 = .12$ ), as was a significant main effect for group ( $F(1, 53) = 7.40, p = .009, \eta_p^2 = .12$ ). Post hoc analyses for each group showed the interaction was driven by a significantly larger drop in attention in the TSD condition for the younger adults ( $t(28) = 2.95, p = .006, d = .61$ ) than the older adults ( $t(25) = -.73, p$

= .47,  $d = -.14$ ). For the Displacement parameter, main effects for condition ( $F(1, 53) = 11.71, p = .001, \eta_p^2 = .18$ ) and group ( $F(1, 53) = 9.31, p = .004, \eta_p^2 = .15$ ) were found. For the Episodic parameter, no significant interaction or main effects were found. Data are shown in Table 3 and Figure 3.

**Table 3.**

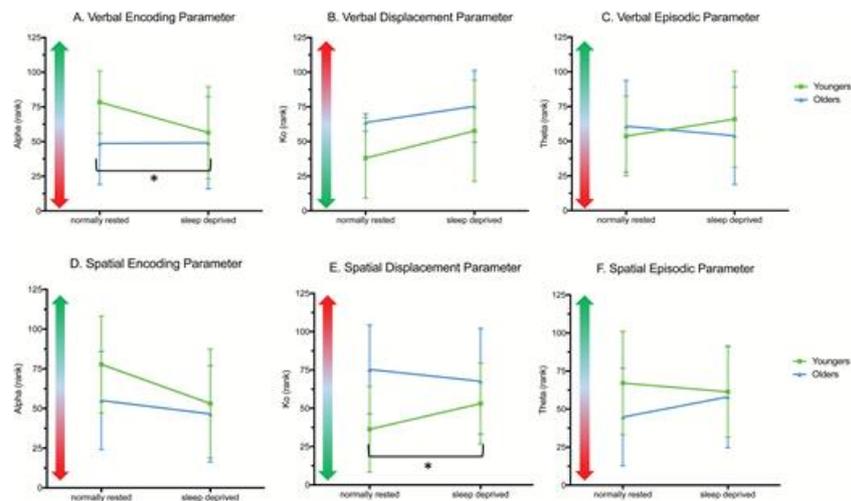
Verbal task mean parameter estimates while normally rested and following total sleep deprivation, by group

	Younger adults				Older adults			
	NR		TSD		NR		TSD	
	<i>M (SD)</i>	95% CI	<i>M (SD)</i>	95% CI	<i>M (SD)</i>	95% CI	<i>M (SD)</i>	95% CI
Attention	.95 (.10)	[.91, .98]	.81 (.26)	[.71, .91]	.80 (.17)	[.73, .87]	.75 (.30)	[.63, .86]
Displacement	.28 (.20)	[.21, .35]	.47 (.34)	[.34, .59]	.48 (.26)	[.38, .59]	.58 (.27)	[.49, .68]
Episodic	.52 (.61)	[.29, .75]	.84 (.90)	[.51, 1.18]	.80 (1.20)	[.32, 1.28]	.54 (.63)	[.30, .77]

NR = normally rested; TSD = total sleep deprivation; CI = confidence interval.

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**Figure 3.**



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Group performance based on ranked data on verbal (A–C) and visuospatial (D–F) working memory parameters of Attention, Displacement, and Episodic Encoding in normally rested vs total sleep-deprived conditions. Higher ranking on displacement indicates worse performance. Error bars represent the standard deviation. \*Significant Group  $\times$  Condition interaction ( $p < .05$ ).

## Visuospatial working memory task

For behavioral data (Table 4, Figure 2), a statistically significant interaction was observed between condition and group,  $F(1, 46) = 5.85, p = .02, \eta_p^2 = .11$ . Post hoc analyses showed this was driven by a larger drop in performance by the younger adults ( $F(1, 24) = 12.74, p = .002, \eta_p^2 = .35$ ) in the TSD condition compared with the older adults ( $F(1, 22) = .02, p = .88, \eta_p^2 = .001$ ). In addition, the main effects for group ( $F(1, 46) = 17.56, p < .001, \eta_p^2 = .28$ ), condition, ( $F(1, 46) = 4.77, p = .03, \eta_p^2 = .09$ ) and lag ( $F(4, 43) = 15.36, p < .001, \eta_p^2 = .59$ ) were all significant.

**Table 4.**

Visuospatial task mean performance scores at each lag condition while normally rested and following total sleep deprivation, by group

	Younger adults				Older adults			
	NR		TSD		NR		TSD	
	<i>M (SD)</i>	95% CI						
Lag0	72.41 (18.41)	[65.41, 79.42]	56.94 (21.74)	[48.34, 65.55]	59.26 (18.21)	[52.06, 66.46]	53.70 (19.25)	[46.09, 61.32]
Lag1	54.31 (20.67)	[46.44, 62.17]	47.22 (19.09)	[39.67, 54.78]	37.96 (18.17)	[30.77, 45.15]	31.48 (19.72)	[23.68, 39.28]
Lag2	56.90 (22.31)	[48.41, 65.38]	46.76 (24.41)	[37.10, 56.42]	34.26 (17.19)	[27.46, 41.06]	33.80 (15.43)	[27.69, 39.90]
Lag3	55.60 (21.80)	[47.31, 63.90]	41.67 (23.00)	[32.57, 50.76]	36.11 (19.71)	[28.31, 43.91]	38.89 (19.71)	[31.09, 46.69]
Lag4	56.03 (20.22)	[48.34, 63.72]	50.46 (20.94)	[42.18, 58.75]	33.80 (15.82)	[27.54, 40.05]	37.04 (20.36)	[28.98, 45.09]

NR = normally rested; TSD = total sleep deprivation; CI = confidence interval.

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When examining visuospatial WM model parameters (Table 5, Figure 3), the Encoding parameter showed a main effect for group ( $F(1, 49) = 5.22, p = .03, \eta_p^2 = .10$ ). For the Displacement parameter, a significant interaction was found between group and condition ( $F(1, 49) = 5.23, p = .03, \eta_p^2 = .10$ ), and this was driven by a larger increase in displacement (worse performance) during TSD for the younger group ( $t(25) = -2.45, p = .02, d = -.72$ ), relative to the older group ( $t(24) = .93, p = .36, d = .28$ ). There was also a main effect for group (observed,  $F(1, 49) = 21.17, p < .001, \eta_p^2 = .30$ ). For the Episodic parameter, no significant interaction or main effects were found.

**Table 5.**

Visuospatial task mean parameter estimates while normally rested and following total sleep deprivation, by group

	Younger adults				Older adults			
	NR		TSD		NR		TSD	
	<i>M (SD)</i>	95% CI	<i>M (SD)</i>	95% CI	<i>M (SD)</i>	95% CI	<i>M (SD)</i>	95% CI
Attention	.67 (.25)	[.57, .76]	.47 (.27)	[.37, .58]	.50 (.23)	[.41, .59]	.42 (.25)	[.33, .52]
Displacement	.41 (.30)	[.30, .53]	.62 (.28)	[.52, .73]	.81 (.27)	[.70, .92]	.75 (.34)	[.63, .88]
Episodic	.91 (.87)	[.58, 1.24]	.83 (1.26)	[.34, 1.31]	.47 (.83)	[.14, .80]	.84 (1.30)	[.36, 1.31]

NR = normally rested; TSD = total sleep deprivation; CI = confidence interval.

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## Potential effects of baseline sleep group differences

In examining the possible effects of group baseline sleep differences on WM parameter results, the only significant correlation among those 60 correlations (5 sleep parameters  $\times$  2 tasks  $\times$  3 parameters/task  $\times$  2 groups) was between actigraphy SE% and the Episodic parameter of the verbal WM task in the younger adult group ( $p = .044$ , uncorrected). However, since there was neither main effect nor interaction observed for this Episodic parameter and that  $p$ -value would clearly not survive correction, we find it highly unlikely that any of the sleep parameters showing a baseline group difference played a confounding role in the results.

# Discussion

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The current study employed a computational model to examine the effect of TSD on three component processes of WM in younger adults and older adults, in both verbal and visuospatial modalities. The findings for younger adults on verbal WM largely replicate those of Turner et al. [20], despite a shorter TSD, here. Specifically, in both studies, younger adults showed impairments in WM encoding and in displacement, but not episodic memory. In the work of Turner et al., WM displacement showed a stronger effect than encoding, whereas we found the opposite. The difference may be related to the longer TSD (42 hr) in the study of Turner et al., or the difference in time of day (18 hr after habitual wake versus 6 hr in the current study), as participants in our study completed the task at a different time of day with respect to circadian timing and attention.

We extended prior work on the differential impact of TSD on component processes of WM by examining changes in an older adult sample. Although older adults also experienced verbal WM displacement deficits during TSD, they did not show encoding deficits. Relative to younger adults, older adults had worse encoding and WM displacement performance when NR. However, following TSD, younger adults' performance declined to a point similar to that observed in NR older adults in each parameter. This is consistent with earlier findings showing that TSD in younger adults elicited cognitive performance similar to the cognitive decline observed with increasing age [42].

The visuospatial WM task showed a similar pattern of results to the verbal WM task. Younger adults had reduced WM displacement following TSD, as well as a decrement in encoding, although the latter did not reach statistical significance. Older adults, on the other hand, did not show any statistically significant changes in visuospatial WM parameters following TSD.

Limited studies have looked at both visuospatial and verbal domains in the context of sleep loss, particularly in aging populations [43, 44]. Our study found younger adults, relative to older adults, showed greater reductions in attention for verbal stimuli and rehearsal span for visuospatial stimuli. These findings are in keeping with a well-documented larger effect of TSD on sustained attention in younger adults, compared with a relatively preserved performance in older adults [45–47]. Moreover, a number of imaging studies also support the findings from both tasks, particularly when considered alongside known neuroanatomical correlates of WM. The prefrontal areas of the cortex are key in the function of WM, and particularly in attentional control [48, 49]. Furthermore, frontoparietal networks are highly susceptible to sleep loss [50]. When compromised by sleep loss, less reliable performance in the attention component of WM would therefore be expected, and this is reflected in the performance of our younger group on the verbal task. Thus, although we did not measure brain structure or function in this study, the commonly reported reduction in frontoparietal activity and/or connectivity following sleep loss would be expected to compromise performance on the attentional component of verbal WM.

On the other hand, older adults generally show deficits in frontoparietal networks and are consistently found to have shrinkage of the prefrontal areas, reduced white matter microstructure, and decreased parietal volume throughout aging [51, 52]. Previous studies have reported a TSD-induced compensatory response in cerebral activation during WM performance in older adults [24, 53, 54]. At the group level, then, older adults appear more resilient to the effects of TSD on component processes of verbal and visuospatial WM, though the cerebral mechanisms of such resilience require future studies to investigate.

The findings reported in this study further elucidate the inconsistency in the literature regarding differences in cognitive performance declines following TSD between younger and older adults [46]. WM in particular has been a contentious area, and by focusing on each component process of WM individually, the current study has identified that WM processes are not affected equally across these populations.

A recent study by Fenn and Hambrick looked at how individual differences in WM capacity affected overall verbal declarative memory performance following TSD [55]. They reported a significant positive correlation between WM capacity and the increase in verbal memory performance following a period of sleep. Although this particular study was conducted in younger adults, it raises the question as to how component processes of WM might affect other cognitive domains, particularly in the context of typical sleep changes which occur in later life (i.e., depleted slow wave activity and increased awakenings).

Although our study has provided the first systematic evaluation of both visuospatial and verbal WM performance at the component level in younger and older adults in the context of sleep deprivation, there are limitations to its scope. WM performance was measured once at the point of 32 hr TSD in the current study. The work of Chee et al. [43] showed differences in frontoparietal activation following normal sleep, and after 24 and 35 hr of TSD in healthy younger adults performing a WM task. Their findings showed greater interindividual variability in performance at 35 hr TSD. In contrast to this, Turner et al. [20] found that a lower percentage of younger adults showed selective vulnerability or resilience to 42 hr TSD on the verbal WM task compared with our findings following 32 hr TSD. Due to clear differences in outcomes in these two studies, elucidating the contribution of both homeostatic and circadian influences on component processes of WM is important. Furthermore, although we made every effort to recruit a sample with a generalizable habitual sleep schedule (e.g., excluding extreme chronotypes and requiring bed or wake times approximating the population averages), given that we did not assess dim light melatonin onset, it is possible that one or more of the participants were either phase

delayed or phase advanced. Future research may consider assessing circadian phase to better ensure that individuals are sleeping at a biologically appropriate time both leading into the study and during the study itself.

Examining the individual variability in performance following TSD on each of the component processes of WM would also be an important extension to the current study and may provide further elucidation of the inconsistencies in previous findings. Repeating the WM task at multiple time points may reveal differential changes in the different components of WM as homeostatic sleep need increases and elucidate how this interacts with circadian influences on performance. In a similar vein, functional imaging used in future studies could further increase the utility of this component process WM model in identifying individuals who are vulnerable or resilient to sleep loss at a component level. For example, McKenna et al. [56] identified the neural correlates of the model parameters from the verbal WM task employed here. Future studies could examine how functional changes in those brain regions (or, likely, their related networks) as a result of sleep disruption affect performance. Finally, while identifying individual variability in response to TSD, possible predictive factors for these differences were not explored in this study.

This study investigated the effects of TSD on component processes of both verbal and visuospatial WM in younger and older adults. Current findings advance both the sleep deprivation and WM literature in the context of aging in several ways: (1) the component model of WM is theoretically driven and more meaningfully depicts cognitive performance; (2) the effect of TSD can be quantified on each component; and (3) some of the inconsistency in the literature can be clarified, particularly in relation to the impact of TSD on WM. These findings provide further elucidation of differences between clinically healthy younger and older adult groups and help us to increase understanding of the cognitive profile of healthy aging.

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## Notes

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*Conflict of interest statement.* None declared.

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