Memory encoding is impaired after multiple nights of partial sleep restriction

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Keywords
declarative memory, episodic memory, learning

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Accepted in revised form 29 May 2017; received 28 March 2017

DOI: 10.1111/jsr.12578

SUMMARY
Sleep is important for normative cognitive functioning. A single night of total sleep deprivation can reduce the capacity to encode new memories. However, it is unclear how sleep restriction during several consecutive nights affects memory encoding. To explore this, we employed a parallel-group design with 59 adolescents randomized into sleep-restricted (SR) and control groups. Both groups were afforded 9 h time in bed (TIB) for 2 baseline nights, followed by 5 consecutive nights of 5 h TIB for the SR group (n = 29) and 9 h TIB for the control group (n = 30). Participants then performed a picture-encoding task. Encoding ability was measured with a recognition test after 3 nights of 9 h TIB recovery sleep for both groups, allowing the assessment of encoding ability without the confounding effects of fatigue at retrieval. Memory was significantly worse in the sleep-restricted group (P = 0.001), and this impairment was not correlated with decline in vigilance. We conclude that memory-encoding deteriorates after several nights of partial sleep restriction, and this typical pattern of sleep negatively affects adolescents' ability to learn declarative information.

INTRODUCTION

Sleep is important in learning and memory processing (Kopasz *et al.*, 2010; Rasch and Born, 2013). While sleep after learning has garnered considerable attention because of its contributions to memory consolidation, relatively fewer studies have explored the influence of sleep before learning on the brain's ability to encode new information (Antonenko *et al.*, 2013; Drummond *et al.*, 2000; Mander *et al.*, 2011; Van Der Werf *et al.*, 2009; Yoo *et al.*, 2007).

Many people obtain insufficient sleep throughout the working week (Watson *et al.*, 2015), and this may be particularly important in adolescents (Eaton *et al.*, 2010; Owens, 2014), whose brains are actively developing and constantly learning. To date, the impact of inadequate sleep on cognition has been explored primarily by observing behaviour after a night of total sleep deprivation (TSD). In adults this has been associated with picture-encoding deficits attributed to impaired hippocampal function (Yoo *et al.*, 2007). Impaired learning after sleep deprivation (Hagewoud *et al.*, 2010; Li *et al.*, 2009) has been linked to a reduced capacity for long-term potentiation in animal studies (Campbell *et al.*, 2002; Vyazovskiy *et al.*, 2008). To account for reduced learning capacity, the synaptic homeostasis hypothesis (SHY) proposes that sustained wakefulness potentiates synapses to a point of saturation that requires slow wave sleep (SWS) to down-scale synapses for the restoration of encoding ability (Kuhn *et al.*, 2016; Tononi and Cirelli, 2014).

While a single night of sleep deprivation is well suited for laboratory studies, exposure to multiple nights of reduced sleep is the more common form of sleep loss. Several aspects of sleep physiology differ between these two forms of sleep loss. For example, unlike the case of total sleep deprivation, where slow wave sleep (SWS) is lost completely on the experimental night, SWS duration remains relatively intact following multiple nights of sleep restriction, even though other sleep stages are reduced (Ong *et al.*, 2016 Voderholzer *et al.*, 2011). It is currently unclear how multiple nights of partial sleep restriction impact on memory-encoding processes.

This is a question that is particularly pertinent to adolescents, who must encode and retain declarative information regularly to succeed academically. Some studies show remarkable resilience in adolescents' cognitive abilities after 1 night of sleep restriction (4 h) (Carskadon *et al.*, 1981; Fallone *et al.*, 2001). However, other studies have found that several consecutive nights of sleep restriction result in progressive deterioration of subjective alertness (Anderson *et al.*, 2009; Wolfson and Carskadon, 1998) and mood (Lo *et al.*, 2015), as well as objective measures of sustained

attention, working memory and speed of processing (Lo *et al.*, 2015).

In this study we asked whether the encoding deficit observed in young adults after a night of TSD (Yoo *et al.*, 2007) appears in adolescents after several consecutive nights of partial sleep restriction. Our sleep-restricted group consisted of 15 to 18-year-olds who were permitted only 5 h time in bed (TIB) for 5 consecutive nights prior to a picture-encoding task, simulating a demanding school week. A control group with a 9-h sleep opportunity each night followed the same protocol. Encoding success was measured via a delayed recognition test conducted after 3 nights of 9 h TIB recovery sleep. We predicted that picture recognition would be impaired in the sleep-restricted group, indicating a deficit in prior encoding, and this would be independent of a more general deterioration in subjective alertness and vigilance.

METHODS

Participants

Sixty adolescents aged between 15 and 18 years were selected from volunteers who reported no history of chronic medical conditions, psychiatric illness or sleep disorders, were not habitual short sleepers (<6 h actigraphically assessed average TIB), consumed <5 caffeinated beverages a day and had not travelled across more than two time zones 1 month prior to the study. Participants and parents provided written informed consent, in compliance with a protocol approved by the National University of Singapore Institutional Review Board, and received monetary compensation after completion of all conditions.

Participants were randomized into sleep restriction (SR) and control groups. One participant withdrew during the study for personal reasons, leaving a final sample comprised of 59 participants [30 males, 16.1 ± 0.6 years, mean ± standard deviation (SD)]. The SR ($n = 29$) and control ($n = 30$) groups did not differ in age, gender, consumption of caffeinated beverages or on tests of non-verbal intelligence, morningness–eveningness preference, levels of daytime sleepiness, symptoms of chronic sleep reduction, subjective sleep quality, self-reported and actigraphically assessed sleep habits, or levels of anxiety and depression ($P > 0.198$).

Design

The experiment was part of the Need for Sleep 3 study, an 11-day study protocol including several cognitive tests (Fig. 1a). The control group had 9 h TIB (23:00–08:00 hours) for all 10 nights of the protocol. The SR group had 2 baseline nights (B1–B2) of the same 9-h TIB, followed by a 5-day manipulation period (M1–M5) with only 5 h TIB each night (01:00–06:00 hours), simulating a school week containing insufficient sleep. This was followed by 3 recovery nights of 9 h TIB (R1–R3). Participants were prevented from napping and were monitored constantly.

To explore the effects of sleep restriction on encoding, participants performed a picture-encoding task on day M5 (i.e. after 5 nights of sleep restriction for the SR group), and were then given a surprise recognition test after 3 nights of recovery sleep on day R3.

Stimuli

Picture-encoding task

The task included 240 images containing buildings (120) or no buildings (120), selected from Takashima *et al.* (2006). Images were of a wide variety of landscapes and types of building displayed centrally on a computer screen in colour. They were split into three groups of 80 (40 building, 40 non-building). Two of these groups were presented to participants in both the encoding and retrieval session (old images), while the remaining 80 served as 'new' distractor images to be presented only at retrieval. Image groups used during encoding and retrieval were counterbalanced across participants.

Encoding took place in a single block lasting approximately 15 min, where participants viewed 80 building and 80 non-building images in a randomized order. Each image was displayed for 2500 ms, followed by a response screen: '(1) Building, (2) No building'. Participants responded with the appropriate keyboard keys, at which point the trial ended and was followed by a 1000 ms intertrial interval (ITI) (Fig. 1b).

The retrieval session tested participants' recognition of the 160 'old' images from encoding intermixed randomly with the 80 'new' images. Each image was displayed in the same screen position as encoding, now with a five-point confidence scale displayed below: '(1) Definitely did not see, (2) Probably did not see, (3) Unsure, (4) Probably saw, (5) Definitely saw'. The trial ended after a keyboard response was made, or a 5000-ms limit was reached, and this was followed by a 1000-ms ITI (Fig. 1c).

Responses and response time (RT) were collected for encoding and retrieval sessions. Analysis of retrieval excluded any images that were judged incorrectly to contain buildings or not during encoding, as these trials indicate a lack of attention rather than an encoding deficit *per se*. Responses were split into four outcome measures: (1) confidence ratings of 4 (probably saw) and 5 (definitely saw) to old images were classed as 'hits', (2) ratings of 4 and 5 to new images were 'false positives', (3) ratings of 1 (definitely did not see), 2 (probably did not see) to old images were classed as 'misses' and (4) 1 and 2 ratings to new images were 'correct rejections'. To account for participants' individual response bias toward old/new responses, the signal detection measure A′ was calculated, which is the non-parametric version of the more widely used d′ (Stanislaw and Todorov, 1999). For this measure, 0.5 indicates chance performance.

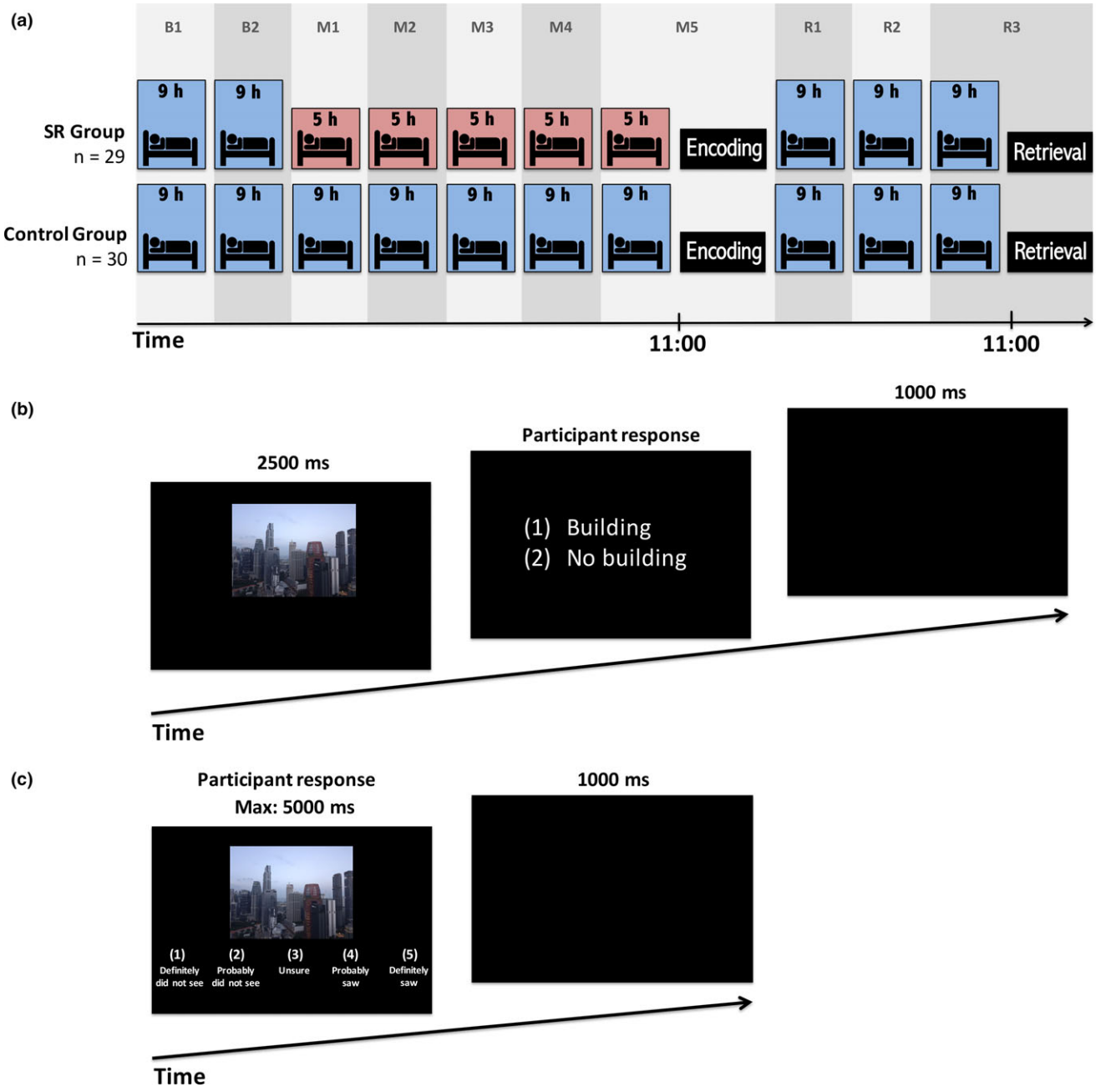

Figure 1. Study protocol and stimuli. (a) Participants in the sleep-restricted group (SR) underwent 2 nights of 9 h time in bed (TIB), followed by 5 h TIB for 5 nights prior to the encoding session on manipulation day 5 (M5). The control group had 9 h TIB for the same period. Both groups had 9 h TIB for the retention interval before the retrieval session on recovery day 3 (R3). (b) Encoding involved the viewing of 160 images each followed by a building/no-building judgement. (c) Retrieval consisted of a recognition test where all 160 old images were presented with an additional 80 new images, and participants indicated their confidence that it was an old or new image on a five-point scale.

N-back task

Participants performed separate blocks of 1-back and 3-back tasks as part of a test battery performed three times daily (at 10:00, 15:45, 20:00 hours). The last of these for the baseline period (at 20:00 hours on B2) was analysed to establish that our two experimental groups were matched for working memory and executive function. In this task, a letter appeared centrally for 1000 ms, followed by a 3000-ms blank screen ITI before the next letter was presented. For the 1-back task, participants were required to hold the previous trial stimulus in memory and respond with a button press to indicate if the current stimulus matched (Y) or did not match (N) the previous trial. For the 3-back task, participants must judge whether the stimulus presented three trials previously matched the current stimulus. The match to mismatch ratio

was 8:24. We included two performance indicators: A′ indicated the participant's ability to discriminate between matches and mismatches (range: 0–1; chance performance = 0.5), while B″ indicated the participant's tendency towards liberal ($B''_D < 0$) or conservative (B″ > 0) response behaviour (neutrality: B″ = 0).

Subjective sleepiness and vigilance

The Karolinska Sleepiness Scale (KSS) was used to provide a subjective indication of alertness, with participants rating their alertness on a nine-point scale (Åkerstedt and Gillberg, 1990). The psychomotor vigilance task (PVT) (Dinges and Powell, 1985) provided an objective indication of sustained attention. Participants responded as quickly as possible with the space bar when a counter appeared on screen, appearing at random intervals between 2000 and 10 000 ms. Participants heard a beep through headphones if no response was detected within 10 000 ms. This was performed in a 10-min continuous block. Response speed (1/RT) and lapses (responses slower than 500 ms) were measured (Fig. 1).

Procedure

Participants' habitual term-time sleep was assessed actigraphically for a 1-week period 1–3 months prior to commencement of the study. This showed a typical pattern of sleep for Singaporean adolescents, with shortened sleep on weekdays [TIB = 6.75 ± 0.92 h, total sleep time (TST) = 5.38 ± 0.87)] that is far below the recommended 8–9 h for that age group (Watson *et al.*, 2015), and sleep extension on weekends (TIB = 8.33 ± 0.92 h, TST = 6.67 ± 0.86 h). All participants adhered to a 9-h TIB sleep schedule for the week prior to the study (23:00–08:00 hours), and groups did not differ for actigraphically assessed TIB (control: 8.79 ± 0.27 h, SR: 8.79 ± 0.43 h, $t_{(57)} = -0.067$, $P = 0.947$) or TST (control: 7.49 ± 0.53 h, SR: 7.41 ± 0.65 h, $t_{(57)} = 0.51$, $P = 0.612$) during this period, confirming that participants were well rested prior to the study.

All cognitive tasks took place in a classroom with participants performing simultaneously on individual laptops. Prior to the manipulation on baseline day 2 (B2), participants performed the n-back task at 20:00. On day M5, participants first rated their sleepiness on the KSS at 10:00 hours, and had vigilance assessed using PVT at 10:15 hours. Picture-encoding took place at 11:30 hours. Participants were instructed to look carefully at each image and indicate whether they contained a building or not. They were not told that it was a memory test or that they would be tested again.

The retrieval session took place at 11:00 hours on day R3, again preceded by a KSS (10:00 hours) and PVT (10:15 hours). Participants were first asked whether they were expecting to have a test related to the day M5 encoding session. Participants were then asked to indicate whether they remembered each image from the previous session.

Statistical analysis

Analysis of variance (ANOVA), *t*-tests or Mann–Whitney *U*-tests were used for group comparisons. The latter was used when the Shapiro–Wilks test indicated a non-normal distribution. Spearman's rho correlations explored the relationship between fatigue effects and memory.

RESULTS

Picture-encoding and recognition

During the encoding session (M5), although the proportion of correct responses for the picture judgement task was high in both groups (SR = 0.95 ± 0.03; control = 0.97 ± 0.03), accuracy was significantly lower in the SR group ($U = 224.5$, $P = 0.001$) (Fig. 2a). The SR group (573 ± 352 ms) was also significantly slower than controls (353 ± 104 ms, $U = 211$, $P = 0.001$) (Fig. 2b).

At retrieval (R3), all participants reported they did not expect the memory test. Recognition was poorer in the SR group, as evidenced by significantly lower A′ scores [$t_{(57)} = 3.459$, $P = 0.001$; Cohen's $d = 0.9$] (Fig. 2c). Response times during retrieval did not differ significantly between groups ($t_{(57)} = 0.91$, $P = 0.366$), suggesting that poorer retrieval was not a result of residual sleepiness (Table 1 for full results).

Subjective sleepiness and vigilance

To examine the relationship between encoding capacity and attention, participants performed a KSS and PVT approximately 1 h prior to the encoding task. As expected, vigilance was impaired significantly for the SR group compared to controls, with a greater number of lapses [$U = 145$, $P < 0.001$ (Fig. 3a)] and slower response speed ($t_{(57)} = 4.636$, $P < 0.001$) (Fig. 3b). The KSS also showed significantly greater sleepiness in the SR group ($U = 225$, $P = 0.001$).

The same measures taken approximately 1 h prior to memory retrieval (R3) showed no significant group differences for PVT lapses ($U = 343$, $P = 0.157$), response speed ($t_{(57)} = 1.672$, $P = 0.1$) or subjective sleepiness ($U = 371$, $P = 0.319$), indicating that the SR group had recovered from the negative effects of sleep restriction.

Next, we correlated impaired alertness (KSS, encoding task RT and both PVT measures) on the day of encoding (M5) with memory retrieval performance (A′) on R3, separately for each group (Fig. 3c,d). In the control group there were no significant correlations between memory and KSS [$r = 0.033$, not significant (NS)], encoding RT ($r = 0.056$, NS), PVT lapses ($r = -0.02$, NS) or PVT response speed ($r = 0.11$, NS). Similarly for the SR group, memory did not correlate significantly with KSS ($r = -0.086$, NS), encoding RT ($r = 0.139$, NS), PVT lapses ($r = 0.047$, NS) or PVT response speed ($r = -0.068$, NS).

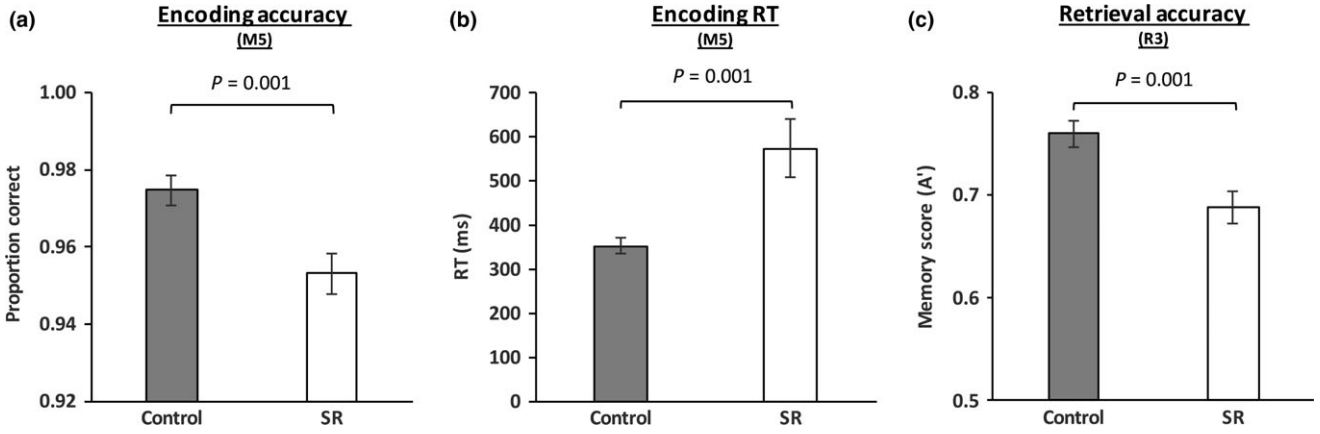

Figure 2. Encoding and retrieval performance. (a) The proportion of correct responses to building/no-building judgements during encoding (M5) differed significantly between sleep-restricted (SR) and control groups, as did (b) mean response time (RT) during the encoding session. (c) During retrieval testing (R3), picture recognition was impaired in the SR group, as shown by significantly lower A′ relative to controls. Mean ± standard error of the mean (SEM).

Table 1 Performance for picture encoding and retrieval, psychomotor vigilance, subjective alertness and n-back tasks

	Control (mean ± SD) n = *30*	*SR (mean ± SD)* n = *29*
N-back (B2)		
1-back A′	0.97 ± 0.03	0.98 ± 0.03
1-back B″	0.12 ± 0.63	0.3 ± 0.7
3-back A′	0.93 ± 0.05	0.91 ± 0.08
3-back B''_D	0.27 ± 0.68	0.32 ± 0.75
Encoding (M5)		
Accuracy (proportion)	0.97 ± 0.02	0.95 ± 0.03**
RT (ms)	353 ± 104	573 ± 352**
PVT lapses	2.7 ± 3.88	15.34 ± 12.49**
PVT response speed (1/RT)	3.62 ± 0.49	2.9 ± 0.72**
KSS	4.67 ± 1.35	6.07 ± 1.51**
Retrieval (R3)		
A′	0.76 ± 0.07	0.69 ± 0.09**
Hits (proportion)	0.36 ± 0.13	0.31 ± 0.12
False alarms (proportion)	0.09 ± 0.08	0.15 ± 0.12*
Misses (proportion)	0.38 ± 0.2	0.42 ± 0.18
Correct rejections (proportion)	0.62 ± 0.25	0.57 ± 0.21
RT (ms)	1470 ± 337	1385 ± 381
PVT lapses	3.4 ± 4.75	4.52 ± 4.99
PVT response speed (1/RT)	3.67 ± 0.58	3.44 ± 0.49
KSS	3.9 ± 1.4	4.31 ± 1.31

SD: standard deviation; RT: response time; PVT: psychomotor vigilance task; KSS: Karolinska Sleepiness Scale.
*$P < 0.05$; **$P < 0.01$.

Last, we assessed whether fluctuations in attention on a trial-by-trial basis could have influenced encoding. If this were the case, we would expect RTs during encoding to be significantly faster for items that were 'later-remembered' when compared to items that were 'later-forgotten'. A 2 × 2 mixed ANOVA with factors of group (SR/control) and memory (later-remembered/later-forgotten) showed a marginal main effect of memory ($F_{(1,57)} = 3.65$, $P = 0.061$; main effect of group, $F_{(1,57)} = 9.985$, $P = 0.003$; interaction, $F_{(1,57)} = 0.245$, $P = 0.622$). The marginal main effect of memory (later-remembered = 414 ± 172 ms; later-forgotten = 464 ± 287 ms) was driven by the control group, where later-remembered items (339 ± 91 ms) were significantly faster than later-forgotten items (376 ± 138 ms, $t_{(29)} = 2.424$, $P = 0.022$). This was not the case for the SR group, where encoding RT for later-remembered (491 ± 201 ms) and later-forgotten items (554 ± 367 ms) did not differ significantly ($t_{(28)} = 1.238$, $P = 0.226$).

In sum, this suggests that while levels of sustained attention measured by the PVT could not account for encoding deficits, there was an association between alertness and encoding success on a trial-by-trial basis reflected in response times to the encoding task itself, although this only reached significance in the control group.

N-back task

Prior to the sleep manipulation, A′ and B''_D did not differ significantly between the SR and control groups ($P > 0.261$; Table 1), indicating that the two groups had similar baseline working memory and executive function. Group differences in picture recognition were therefore unlikely to be due to prior group differences in working memory or executive function.

Actigraphy

There were no differences in TIB or TST between the two groups during the baseline nights (means of B1 and B2) ($P > 0.747$; Table 2). Sleep restriction during the 5-night manipulation period was effective, with the SR group experiencing significantly lower mean TIB and TST relative to the control group ($P < 0.001$). As expected, there was a significant increase in TST for the SR group on the first recovery

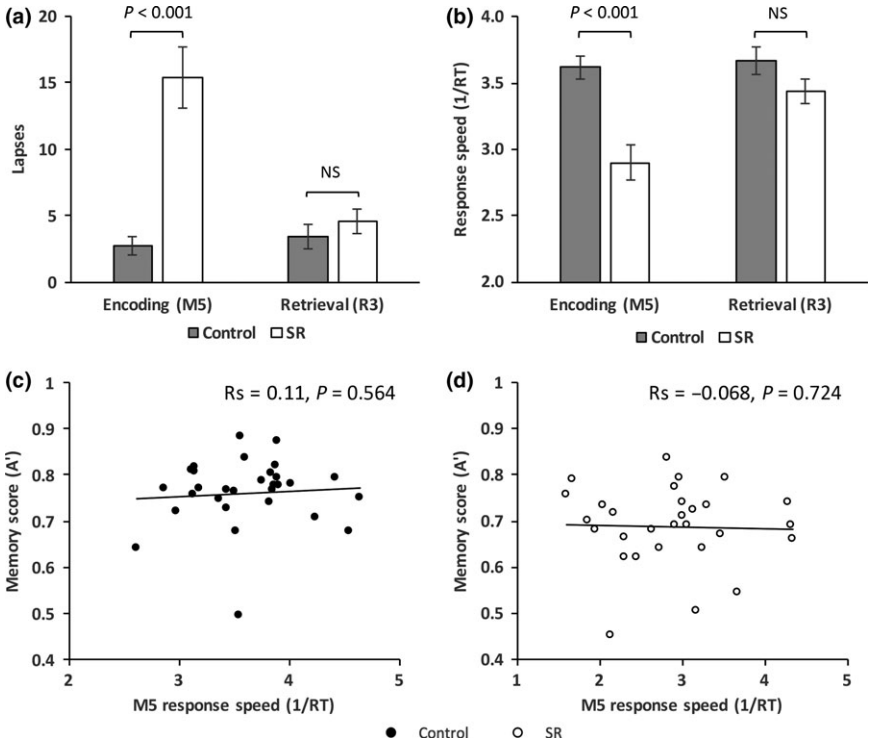

Figure 3. Psychomotor vigilance and its association with encoding. (a) A psychomotor vigilance task (PVT) performed shortly before the encoding task on day M5 (10:15 hours) showed significantly more attentional lapses after 5 nights of sleep restriction relative to controls, while groups did not differ for the PVT performed after 3 nights of recovery sleep on day R3 (10:15 hours). (b) Similarly, our measure of response speed (1/RT) was significantly worse for the sleep-restricted group (SR) relative to the control group on day M5 but not R3. (c) Encoding ability, measured by recognition success after recovery sleep (A′) was not correlated with PVT response speed in either the control group or (d) the SR group. Mean ± standard error of the mean (SEM).

night ($P < 0.001$), while subsequent recovery nights were comparable between the SR and control groups ($P > 0.29$).

DISCUSSION

We found that long-term memory for pictures encoded after 5 nights of sleep restriction was impaired significantly relative to adolescents who had the opportunity to sleep for the recommended amount during the same period (Watson *et al.*, 2015). This impairment was not correlated with decline in psychomotor vigilance, suggesting a specific deficit in the ability to effectively encode new information following successive nights of sleep restriction.

Our findings indicate that memory-encoding deficits after TSD (Campbell *et al.*, 2002; Vyazovskiy *et al.*, 2008; Yoo *et al.*, 2007) are also observed in the more common scenario of cumulative sleep loss during multiple weekday nights. The deficit we observed could be the result of degraded cortical representations during encoding (Poh and Chee, 2017), impaired hippocampal function (Yoo *et al.*, 2007) and/or reduced capacity for LTP (Campbell *et al.*, 2002; Vyazovskiy *et al.*, 2008) following sleep loss.

Encoding deficits following sleep loss are consonant with the synaptic homeostasis hypothesis (SHY) (Tononi and Cirelli, 2014), whereby down-scaling of synaptic connections during SWS is thought to reverse the saturation of memory networks that follows sustained wakefulness. This SWS-dependent down-scaling is key for the restoration of encoding

Table 2 Sleep characteristics across baseline, manipulation and recovery nights (assessed with actigraphy)

	Control (mean ± SD) n = *30*	*SR (mean ± SD)* n = *29*	P-*value*
Baseline (B1–B2)			
TIB (min)	8.99 ± 0.04	8.99 ± 0.03	0.748
TST (min)	7.56 ± 0.54	7.59 ± 0.50	0.829
Manipulation (M1–M5)			
TIB (min)	9.00 ± 0.02	5.01 ± 0.01	<0.001
TST (min)	7.46 ± 0.47	4.38 ± 0.26	<0.001
Recovery (R1)			
TIB (min)	9.01 ± 0.04	8.99 ± 0.04	0.108
TST (min)	7.34 ± 0.41	7.96 ± 0.41	<0.001
Recovery (R2)			
TIB (min)	9.01 ± 0.02	9.01 ± 0.02	0.71
TST (min)	7.62 ± 0.53	7.58 ± 0.53	0.844
Recovery (R3)			
TIB (min)	9.00 ± 0.01	9.00 ± 0.01	0.188
TST (min)	7.31 ± 0.52	7.50 ± 0.52	0.291

P-values correspond to independent-samples *t*-tests.
TIB: time in bed; TST: total sleep time; SD: standard dviation.

ability. In support of this framework, artificially increasing slow wave activity (SWA) can increase encoding capacity (Antonenko *et al*., 2013), while interrupting SWA reduces it (Van Der Werf *et al*., 2009).

Although we did not obtain PSG in the present study our previous Need for Sleep (NFS) studies used a similar protocol of sleep restriction, and inform of the probable changes to sleep architecture in the present study. In the two studies, during 5 and 7 nights of sleep restriction, SWS duration was preserved, while stage 1 (N1), stage 2 (N2) and rapid eye movement (REM) sleep duration were reduced significantly (Ong *et al*., 2016; Ong *et al*., unpublished). Critically, it appears that despite the relative preservation of SWS after severe sleep restriction, encoding capacity was diminished, suggesting that other sleep stages are also important in restoring memory function.

Decline in vigilance is an extremely robust finding in sleep deprived individuals (Lim and Dinges, 2010), and it would be no surprise that encoding would be compromised during PVT lapses, which often represent microsleeps. To minimize the effect of this potential confound, encoding trials where participants made incorrect responses were removed from subsequent analyses.

Response times during encoding trials provide some indication of alertness, and congruent with prior work we found that these did not correlate with subsequent memory impairment (Yoo *et al*., 2007). Adding to this, however, we showed that items that were remembered later were responded to significantly faster during encoding than later forgotten items, although only in the control group. In addition, we found no significant correlation between memory and subjective alertness or psychomotor vigilance (lapses and response speed) in tests performed 45 min prior to encoding. Taken together, it appears that the impairment to subjective alertness and psychomotor vigilance associated with several nights of partial sleep restriction did not account for deficits to long-term memory encoding. However, trial-by-trial fluctuations in attention were associated with RT during encoding in the control group, therefore we cannot rule out attention as a factor influencing encoding ability. The lapses in attention that go hand-in-hand with insufficient sleep are very likely to impact upon a student's ability to learn, but here we show that even when stimuli appear to be attended to by sleep-restricted individuals, they are not encoded effectively.

There has been a great deal of interest in the role of sleep in memory consolidation (Rasch and Born, 2013), but recent evidence suggests that partial sleep restriction as opposed to TSD after learning has little impact upon retention of declarative memories (Lo *et al*., 2016b; Voderholzer *et al*., 2011), unlike the deficit in encoding observed here. Moreover, the few studies that have examined the consequences of TSD after learning have shown no long-term impact on declarative memory (Gais *et al*., 2007). A recent study showed that initial impairments to word-pair consolidation were no longer present 3–6 days later, presumably because subsequent recovery sleep allowed memory processes to 'catch up' (Schönauer *et al*., 2015). This relative resilience, or recovery, of memory consolidation stands in contrast to the deficit we observed to encoding, that is not remedied with recovery sleep. It seems there is little that can be done if a memory is not encoded effectively in the first place. Therefore, to understand the practical implications of sleep loss on learning, future work should focus more attention upon encoding capacity.

These findings add to prior investigations performed by our laboratory exploring the cognitive consequences of sleep restriction. These have identified deficits associated with mood, sustained attention, working memory, executive function, speed of processing (Lo *et al*., 2015, 2016a), vocabulary learning (Huang *et al*., 2016) and an increased tendency to form false memories (Lo *et al*., 2016c). Conversely, memory consolidation seems unaffected (Lo *et al*., 2016b).

To conclude, many adolescents are known to function on a schedule of accumulated sleep debt throughout the school week, and here we show that this leads to an inability to form new memories effectively. More emphasis must be placed upon proper sleep habits, and possible countermeasures to poor sleep should be explored, for children to learn and retain information more effectively.

ACKNOWLEDGEMENTS

This work was supported by the National Medical Research Council Singapore (STaR/0015/2013) and the Far East Organization. The authors thank Su Mei Lee, June Lo, Jesisca Tandi, Lee Xuan Kai, Cher Wei Shan, Lydia Teo Manling, Chong Shin Wee, Nicholas Chee, Shirley Koh, Teo Teck Boon and Vaisakh Puthusseryppady for contribution to data collection. Nanyang Girls Boarding School provided excellent support for the study.

AUTHOR CONTRIBUTIONS

JNC and MWLC designed the study, JNC and KS collected data, JNC conducted data analysis and JNC, KS and MWLC wrote the manuscript.

CONFLICT OF INTEREST

The authors declare no competing financial interests.

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