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Consolidation of vocabulary is associated with sleep in typically developing children, but not in children with dyslexia

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Research Highlights

- We used overnight polysomnography to investigate whether sleep promotes vocabulary learning via the same active mechanisms in children as are known to be important in adults.
- We found that, in typically developing children, slow wave activity was a key predictor of overnight improvements in recall of new vocabulary, similar to findings in adults.
- In children with dyslexia, who typically have word learning difficulties, slow wave activity was not related to consolidation of new vocabulary despite overnight improvements in recall, which advocates examining both typical and atypical development in order to inform models of sleep-associated memory consolidation.
- One interpretation is that sleep plays a more passive, protective role in children with dyslexia, with potential long-term implications for learning.

Abstract

Sleep is known to play an active role in consolidating new vocabulary in adults; however, the mechanisms by which sleep promotes vocabulary consolidation in childhood are less well understood. Furthermore, there has been no investigation into whether previously reported differences in sleep architecture might account for variability in vocabulary consolidation in children with dyslexia. Twenty-three children with dyslexia and 29 age-matched typically developing peers were exposed to 16 novel spoken words. Typically developing children showed overnight improvements in novel word recall; the size of the improvement correlated positively with slow wave activity, similar to previous findings with adults. Children with dyslexia showed poorer recall of the novel words overall, but nevertheless showed overnight improvements similar to age-matched peers. However, comparisons with younger children matched on initial levels of novel word recall pointed to reduced consolidation in dyslexics after 1 week. Crucially, there were no significant correlations between overnight consolidation and sleep parameters in the dyslexic group. This suggests a reduced role of sleep in vocabulary consolidation in dyslexia, possibly as a consequence of lower levels of learning prior to sleep, and highlights how models of sleep-associated memory consolidation can be usefully informed by data from typical and atypical development.

Keywords: vocabulary acquisition, sleep, dyslexia, word learning, language development, memory consolidation

Sleep has a well-documented role in memory consolidation: the process by which new and initially weak memories become strengthened and resistant to interference (Born, 2010; Diekelmann & Born, 2010; Kurdziel et al., 2013; Rasch & Born, 2013; Wilhelm et al., 2008, 2013). Sleep difficulties are present in an array of neurodevelopmental disorders, with researchers beginning to address whether sleep-dependent memory consolidation may be implicated (Gruber & Wise, 2016): e.g., autism spectrum disorder (Henderson, Powell, Gaskell, & Norbury, 2014; Maski et al., 2015) attention deficit/hyperactivity disorder (ADHD; Prehn-Kristensen et al., 2013). Whilst preliminary evidence suggests that dyslexia is associated with aberrant sleep architecture, to our knowledge no research has investigated whether there are differences in the extent to which newly learned material is consolidated during sleep in these individuals. Understanding the ways in which sleep is affected in developmental disorders not only has important clinical ramifications, but can also advance theories of how sleep supports cognition during development (Smith & Henderson, 2016).

A complementary learning systems (CLS) view of word learning (Davis & Gaskell, 2009; McClelland, McNaughton, & O'Reilly, 1995; McClelland, 2013) posits that new word forms can be acquired rapidly via the hippocampal system. However, for a new word form to become a robust long-term lexical representation (and behave like a real word), integration into existing

neocortical networks is required. The consolidation process required for strengthening and integrating lexical representations has been associated with sleep. In adults, Dumay and Gaskell (2007) found that explicit recall of novel spoken words (e.g., “dolpheg”) significantly increased after a period of sleep but not after an equivalent period of wake. Providing clear support for the CLS account, the novel words only engaged in lexical competition with existing words after sleep. Lexical competition is a hallmark of an existing lexical representation (Gaskell & Dumay, 2003; Henderson, Weighall, Brown, & Gaskell, 2013; Mattys & Clark, 2002), proposed by many models of spoken word recognition as crucial for automatic language comprehension (Gaskell & Marslen-Wilson, 2002; Grainger & Jacobs, 1996; Luce & Pisoni, 1998; McClelland & Elman, 1986; Norris & Norris, 1994). Thus, once a novel word engages in lexical competition it can be said to be integrated within neocortical memory (Davis & Gaskell, 2009). Lexical competition can be measured via the pause detection task (Mattys & Clark, 2002), in which a 200ms pause is inserted into basewords (e.g., “dolph_in”) for which new competitors have been taught (e.g., “dolpheg”) and control words for which no new competitors have been taught. Dumay and Gaskell (2007) observed slower pause detection latencies for basewords than control words, but only after sleep. Pause detection latency is argued to signal the amount of lexical activity that is present at the pause, with greater levels of lexical activity reducing the resources available for pause detection (Mattys & Clark, 2002).

A similar sleep-associated time-course of lexical integration and strengthening of explicit memory for novel words has been reported in school-aged children (Henderson, Weighall, Brown & Gaskell, 2012). Furthermore, other studies have reported sleep-associated gains in explicit memory for second

language (i.e., German) translations of familiar words in school-aged children (Gais, Lucas, & Born, 2006), as well as greater forgetting of newly learned words under sleep-restricted conditions in adolescents (Huang et al., 2016).

Two key EEG events that occur during nonrapid eye movement (NREM) sleep have been implicated in the consolidation of declarative memory (Diekelmann & Born, 2010): EEG slow oscillations (<1 Hz) and sleep spindles (short bursts of 12-15hz EEG activity, generated in thalamocortical circuits, that are temporally synchronised with the up-state of slow oscillations). These EEG events are synchronized with hippocampal ripples (Staresina et al., 2015) and together have been proposed to coordinate the reactivation of newly learnt information stored in hippocampal networks and its subsequent integration into neocortical systems (Rasch & Born, 2013). In a study of adult novel word learning, Tamminen, Payne, Stickgold, Wamsley, and Gaskell (2010) demonstrated that overnight increases in lexical competition strongly correlated with spindle density, and increases in recognition speed strongly correlated with slow wave sleep (SWS) duration. This suggests an active role for sleep in vocabulary consolidation. More recently, Weighall, Henderson, Barr, Cairney, and Gaskell (2016) reported a strong positive correlation between the magnitude of a cued-recall advantage for novel words learned the previous day (as compared to novel words learned on the day of the test) and fast spindle density (13.5 – 15 Hz).

Since children display proportionally more SWS than adults, and have stronger slow-wave activity (SWA, that is, EEG power in the frequency range of 0.5 – 4 Hz), peaking at 10-12 years (Campbell & Feinberg, 2009; Kurth et al., 2010; Ohayon, Carskadon, Guilleminault, & Vitiello, 2004), the benefits of sleep

for memory consolidation may be enhanced during childhood (James, Gaskell, Weighall, & Henderson, 2017; Weighall et al., 2016; Wilhelm et al., 2013). However, relatively few studies have examined sleep-associated memory consolidation of declarative memory in children (Backhaus et al., 2008; Kurdziel et al., 2013; Prehn-Kristensen et al., 2011; Wilhelm et al., 2013, 2008). In one exception, Urbain et al. (2016) found that hippocampal activity (measured via magnetoencephalography) during the successful immediate recall of new objects positively correlated with percentage of SWS in a subsequent nap in 8-12-year-olds. After sleep however, successful recall was negatively correlated with hippocampal activity, and positively associated with activity in the prefrontal cortex. This study suggests that – as in adults – sleep plays an active role in strengthening neocortical representations. However, studies that record overnight sleep parameters associated with memory consolidation (e.g., SWS and sleep spindles) and examine the neurobiological sleep correlates of vocabulary consolidation are completely lacking.

An effective developmental model of sleep-dependent consolidation must account for individual differences. Hence, to advance our understanding of how new vocabulary is consolidated it is vital to understand how the consolidation process might be disrupted. Developmental dyslexia affects around 10% of children (Lewis, Hitch, & Walkert, 1994; Shaywitz, Shaywitz, Fletcher, & Escobar, 1990) and is characterized by difficulties with learning to read (Vellutino, Scanlon, & Spearing, 1995). Preliminary evidence suggests that the sleep architecture of children with dyslexia is atypical (Bruni, Ferri, Novelli, Finotti, et al., 2009; Bruni, Ferri, Novelli, Terribili, et al., 2009; Mercier, Pivik, & Busby, 1993). Bruni, Ferri, Novelli, Terribili, et al. (2009) found that children with

dyslexia showed increased spindle density that was positively associated with their reading difficulties. However, whether sleep difficulties are associated with language learning difficulties in dyslexia remains unknown.

Children and adults with dyslexia show impairments in learning new phonological forms when tested immediately after exposure (Di Betta & Romani, 2006; Mayringer & Wimmer, 2000; Messbauer & de Jong, 2003), but very few studies have examined consolidation. Two studies reported that children with dyslexia showed equivalent retention rates to typical peers after one week (Li, Shu, McBride-Chang, Liu, & Xue, 2009; Messbauer & de Jong, 2003). However, in both cases words were learnt with visual referents and enhanced memory for the visual stimuli may have masked consolidation deficits. Warmington (2008) also examined spoken word learning in adults with dyslexia and obtained data suggestive of a long-term lexical integration problem. Both typical and dyslexic adults showed good levels of recognition of novel words immediately after exposure. Whilst typical adults showed lexical competition after one week (i.e., slowed responses to “biscuit” having learned “biscal”), for dyslexic adults, lexical competition only emerged after additional training.

The main aim of this study was to examine whether individual differences in sleep parameters relate to how well new phonological representations are strengthened and integrated in children with and without dyslexia. Based on previous adult research, we predicted that SWA and sleep spindle power (EEG power density in the slow-wave and spindle frequency ranges, respectively) would correlate with overnight improvements in cued recall and overnight increases in lexical competition (Tamminen et al., 2010; Weighall et al., 2016). For dyslexics, we predicted poorer novel word learning relative to age-matched

peers, owing to their phonological difficulties (Mayringer & Wimmer, 2000). We also predicted that correlations between sleep features and vocabulary consolidation may differ in this group, based on previous reports of sleep spindle abnormalities in dyslexia (e.g., (Bruni, Ferri, Novelli, Finotti, et al., 2009; Bruni, Ferri, Novelli, Terribili, et al., 2009; Mercier, Pivik, & Busby, 1993). Dyslexia often co-occurs with disorders such as ADHD (Willcutt & Pennington, 2000) and specific language impairment (Bishop & Snowling, 2004) where sleep disturbances have also been reported (Gruber & Wise, 2016; Owens, Maxim, Nobile, Mcguinn, & Msall, 2000). Therefore we investigated whether any potential group differences in sleep architecture and/or consolidation were related to co-occurring cognitive features (i.e., attention and broader language skills) rather than reading difficulties *per se*.

Method

Participants

Fifty-two monolingual children (8-13-years-old) were recruited: 23 children with dyslexia (DY; 14 males) and 29 chronological-age matched typical peers (CA; 16 males). Children with dyslexia were initially recruited on the basis of parentally reported concerns about reading. They were then retained in this group if they met one/both of: i) standard scores <90 on at least two out of three literacy measures (word reading, nonword reading and spelling) or ii) diagnosis of dyslexia from an educational psychologist. There were 25 initial referrals and two were reclassified as typically developing as they did not meet either of these criteria and parental concerns were mild. No children initially referred as typically developing were reclassified as dyslexic on the basis of these criteria.

All children were attending mainstream schools and had normal, or corrected to normal vision and hearing. Children with dyslexia who had additional difficulties were included to permit examination of associations with co-occurring features (one participant had diagnoses of dyspraxia and ADHD, one had a diagnosis of ADHD and another had a diagnosis of dyscalculia). No learning difficulties were reported in the CA group. Table 1 shows the group means and standard deviations for the background measures. The two groups did not differ significantly in age ($t=.04$; $p=.972$) or nonverbal ability ($t=1.86$; $p=.069$). However, as expected, the group with dyslexia performed significantly worse on the literacy measures (word reading ($t=5.35$, $p<.001$), nonword reading ($t=9.39$, $p<.001$), spelling ($t=6.67$, $p<.001$)). None of the children in either group had been diagnosed with a sleep disorder and there was no group difference in total score on the sleep screening questionnaire (CSHQ (see *Sleep Measures* below); $t=.54$, $p=.594$).

Twenty-four younger children (C2; 12 males) were recruited post hoc to control for Day 1 differences between the CA and DY groups on the cued recall task. This control group is vital as baseline differences in word learning between the CA and DY group make interpretation of improvements between sessions i.e. consolidation, more difficult to interpret. Including a younger, ability-matched control group alongside a group matched on chronological age is very common in studies of children with reading and language difficulties (e.g. Georgiou, Papadopoulos, Zarouna, & Parrila, 2012; Talcott, Witton, & Stein, 2013). This enables both age and ability to be taken into account in a way that cannot be achieved with a single control group of either type, allowing the most comprehensive understanding of consolidation patterns in the current study,

The C2 group had significantly weaker nonverbal ability ($t=2.61, p=.012$) and significantly stronger reading ($t=2.43, p=.019$) and nonword reading skills ($t=5.34, p<.001$) than the DY group. However, crucially, the C2 group did not differ from the DY group on Day 1 cued recall scores ($t=.34; p=.739$). Due to time constraints, this group only completed key literacy and nonverbal background measures (see Table 1). Furthermore, this group did not participate in the overnight sleep EEG part of the study due to logistical constraints. Whilst these data would undoubtedly have added value, the key comparison group for the sleep measures are the CA controls as sleep architecture changes across development and, as such, data from controls of a different chronological age are difficult to interpret.

Stimuli

Thirty-two stimulus pairs consisting of a baseword (e.g. “dolphin”) and a novel competitor word (e.g. “dolpheg”) were used. The overlap between the words was essential for eliciting competition effects in the pause detection task. For details on how these stimuli were constructed see Henderson, Weighall, Brown and Gaskell (2012). During training participants were exposed to 16 novel competitors (List 1 or List 2, counterbalanced across participants). In the pause detection task, assessing lexical integration, all participants heard both lists of existing words (n 32); half of these words had a trained novel competitor (competitor condition) and half did not (control condition). Stimuli were recorded on a Pioneer PDR 509 system by a female native English speaker.

Procedure

Participants completed the *training phase*, where they were introduced to the 16 novel words. Knowledge of the new words was assessed via *cued recall*

(to assess explicit phonological memory) and *pause detection* (to assess lexical integration) immediately after learning (Day 1), approximately 24 hours later (Day 2) and approximately 1 week later (see Figure 1). At the end of the 1 week session, children's knowledge of the known base words was assessed using a *picture-word matching* task. The DY and CA groups underwent overnight *polysomnography* between the Day 1 and Day 2 sessions. The DY and CA groups also completed standardized tests of language and attention, administered at the end of the Day 2 and 1-week sessions. The parents of these groups completed questionnaires assessing sleep and attention skills.

For the DY and CA groups, the mean time of testing on Day 1 was 17:40 (SD= 01:23), on Day 2 was 09:21 (SD= 01:35) and at 1-week was 14:30 (SD= 03:40). There were no significant differences between the DY and CA groups in time of testing at any session. As the C2 group did not undertake the overnight polysomnography and were seen at school for all sessions, they were seen at different times on average to the other two groups. The mean time of testing on Day 1 was 11:38 (SD= 01:43), on Day 2 was 12:11 (SD= 01:14) and at 1-week was 11:10 (SD= 01:00).

Training Phase. Children were exposed to each novel word 18 times across two training tasks. Stimuli were presented on laptops via headphones using DMDX (Forster & Forster, 2003). The phonologically-based training tasks comprised: (i) phoneme monitoring (i.e., children indicated whether a pre-specified phoneme was present in each word; total of 12 exposures to each novel word), (ii) phoneme segmentation (i.e., children listened to each novel word and were asked to repeat it aloud and say the first (Block 1) or last (Block 2) sound; total of 6 exposures to each novel word). This procedure has been used with

typically developing children (Henderson et al., 2012; Henderson et al., 2013) and children with autism (Henderson et al., 2014).

Testing Phase. In a *cued recall* task, children heard the first syllable (e.g. “dol-“) of each of the novel words and were asked to recall the whole string. Accuracy was recorded with no feedback. A *pause detection* task (measuring lexical integration) was used as in Henderson et al. (2012). Participants indicated with a button press whether or not there was a 200ms pause present in the 32 existing base words, 16 of which had a trained novel competitor (competitor condition) and 16 of which did not have a trained novel competitor (control condition), and 32 bisyllabic filler words. Pauses were present in half of the competitor words, half of the control words and half of the filler words (with pause present/absent versions counterbalanced across participants). RT was measured from the onset of the pause and accuracy was recorded.

Baseword familiarity. Participants completed a picture-word matching test at the end of the 1-week session (see Henderson et al., 2012) in which they heard each base word via headphones and selected one of four pictures on-screen.

Sleep Measures (DY and CA)

Questionnaires. Parents completed the *Children’s Sleep Habits Questionnaire* (CSHQ; Owens, Spirito, & McGuinn, 2000) (return rates 96% (n=22) for DY and 97% (n=28) for CA). This is a 45-item scale, from which eight sub-scores and a total score can be derived (bedtime resistance, sleep onset delay, sleep duration, sleep anxiety, night waking, parasomnias, sleep disordered breathing and daytime sleepiness). Respondents indicate how frequently a behavior occurs in a typical week. Higher scores indicate greater difficulties.

Children and parents completed bespoke sleep diaries for eight nights, with the first night being the night before their sleep recording. Parents were asked to note children's bedtime and wake time on each day and note anything atypical. Each morning children rated (on a 10-point scale, with 10 representing the best outcome) how easily they got to sleep, how well they slept, how easy it was to get up and how awake they felt in the morning, as well as recording the number of night awakenings. The sleep diaries served to establish whether children's sleep on the night of the sleep recording was typical. Sleep diaries were returned by 26 of the CA families (90%) and 21 of the DY families (91%).

Overnight sleep recording. Sleep recordings took place in participants' homes, minimizing sleep disruption by ensuring familiar surroundings and routines. Lab-based recordings can result in atypical sleep on the first night (Scholle et al., 2003), an effect which can be mitigated by home recordings (Edinger et al., 1997). Home recordings have been validated (Zou, Grote, Peker, Lindblad, & Hedner, 2006) and are often preferred in developmental studies (Gruber et al., 2009). Sleep EEG was recorded using the portable Embla Titanium amplifier (Embla Systems Titanium, Broomfield, CO, USA) with RemLogic version 1.1 software. After the scalp locations were cleaned with NuPrep exfoliating agent (Weave and Company, Aurora, CO, USA), silver/silver chloride electrodes were attached according to the international 10-20 system at six standard sites: frontal (F3, F4), central (C3, C4) and occipital (O1, O2). Each was referenced to an electrode on the contralateral mastoid (A1 or A2) and a ground electrode was attached to the forehead. Electrooculography (EOG) was recorded using two electrodes placed diagonally next to the eyes and electromyography (EMG) was recorded from two electrodes placed underneath the chin. Data were recorded

on online and sampled at 256 Hz. The EEG and EOG channels were filtered between 0.3-35 Hz and the EMG channel was filtered between 10-100Hz.

Sleep stages were visually scored offline by an expert scorer, using Embla REMLogic software, according to the standard criteria of the American Academy of Sleep Medicine (AASM; Iber, Ancoli-Israel, Chesson & Quan, 2007). A random 30% of the recordings were double scored with a second expert scorer and 100% agreement was reached. Time spent in each sleep stage (N1, N2, SWS and REM) was calculated, in addition to percent time (relative to total time asleep). Three key parameters were calculated as markers of declarative memory consolidation: spindle density i.e. number of spindles per minute (Tamminen et al., 2010; Weighall et al., 2016), spindle power i.e. power density in spindle frequency range (Wilhelm et al., 2013) and slow wave activity (SWA; Wilhelm et al., 2013, 2014).

To detect sleep spindles, epochs scored as either N2 or SWS were extracted from all six EEG channels. Artefacts were rejected visually from the data using EEGLAB version 10.0 (Swartz Center for Computational Neuroscience, University of California San Diego) before a linear finite impulse response filter was used to bandpass filter each channel at 12-15 Hz. An automated detection algorithm (Ferrarelli et al., 2007) counted discrete spindle events within the filtered time series that exceeded a threshold of eight times the mean channel amplitude (following Cairney, Durrant, Jackson, & Lewis, 2014; Tamminen et al., 2010; Tamminen, Ralph, & Lewis, 2013). Spindle density (counts per minute) was then calculated for central (C3, C4) and frontal (F3, F4) EEG channels for each participant and averaged across these channels. Spindle density was

averaged across the 12-15 Hz range (following the same procedure as Cairney, Lindsay, Sobczak, Paller, & Gaskell, 2016; Weighall et al., 2016).

Power spectral analysis of the EEG signal was performed using Fast Fourier Transformation on all recording sites (following previous studies e.g., Kurth et al., 2010; Ohayon et al., 2004; Wilhelm et al., 2013). Absolute power (measured as $\mu\text{V}^2/\text{Hz}$) was computed for two frequency bands of interest: 0.5 – 4 Hz (SWA, averaged across all electrode sites) and 12-15Hz (spindle power, averaged across frontal and central electrode sites).

Results

Training

Performance on the training tasks was high (above 80%) for all groups, with the exception of final phoneme segmentation for the dyslexia group (Table 2), suggesting children were able to engage with the training procedure. Unsurprisingly, the group with dyslexia performed significantly worse than the CA controls on the phoneme monitoring task ($p=.005$) and the segmentation tasks ($p=.009$), and worse than the younger (C2) controls on the segmentation tasks ($p=.005$). On the repetition trials, which do not rely on manipulating phonological information, the scores for all groups were near ceiling; whilst the two control groups did not significantly differ from each other ($p=.547$), only the younger C2 controls performed significantly better than the children with dyslexia ($p=.002$). There were no group differences on the control picture-word matching task assessing knowledge of the base words, with all groups scoring at ceiling (all mean scores >15.8 out of 16; $F(2, 73)= .28, p=.760$).

Word learning outcomes

Explicit memory. The Shapiro-Wilk test was used to explore the normality of the distributions on the cued recall task for all three groups. There were no significant deviations from normality at any time point in either the CA or DY group. There was a trend towards a bimodal distribution on Day 1 cued recall scores in the C2 group, but the distributions on Day 2 and 1 week did not deviate from normality. The mean number of correct responses on the *cued recall* task in each session (Figure 2) was entered into a mixed ANOVA with Session as a within-subjects factor (Day 1, Day 2, 1 week) and Group as a between-subjects factor (CA, DY, C2).

Cued recall improved across sessions (Session, $F(2, 146) = 273.06, p < .001, \eta_p^2 = .789$). The CA controls recalled significantly more novel words than both the children with dyslexia and the younger C2 controls (Group, $F(2, 73) = 13.65, p < .001, \eta_p^2 = .272$). There was also a Session x Group interaction ($F(4, 146) = 2.57, p = .040, \eta_p^2 = .066$). Bonferroni-corrected pairwise t-tests revealed that all three groups showed a significant improvement between Day 1 and Day 2 (CA, $t(28) = 13.00, p < .001$; DY, $t(22) = 8.72, p < .001$; C2, $t(23) = 8.86, p < .001$). The magnitude of this improvement did not differ between any of the groups ($F(1, 50) = .008, p = .930$). However, there was no further improvement in either the group with dyslexia or the CA controls between Day 2 and 1 week (CA, $t(28) = 1.09, p = .284$; DY, $t(22) = .71, p = .487$), whereas there was a further significant improvement for the C2 controls ($t(23) = 3.92, p = .001$), which accounts for the Session x Group interaction. In sum, while the patterns of consolidation were similar across sessions for the group with dyslexia and the CA controls, the C2 controls showed an extra performance boost at 1 week.

Pause Detection. The mean percentage of correct responses across experimental items (both competitors and controls) in the pause detection task was calculated for each group: CA controls mean accuracy 92.67% (SD= 4.91%), DY mean 84.51% (SD=11.01%), C2 controls mean 87.02% (SD= 8.50%). A one-way ANOVA revealed a significant main effect of Group ($F(2, 73)= 6.73, p= .002, \eta_p^2=.156$). Post-hoc Tukey's HSD tests were conducted, with Bonferroni correction applied for multiple comparisons, giving an adjusted alpha level of $p=.017$. These revealed that the CA controls were significantly more accurate than the group with dyslexia ($p=.002$). There was also a trend for CA controls to outperform the younger C2 controls ($p=.041$), although this did not survive Bonferroni correction. Accuracy in the group with dyslexia and the younger C2 controls did not differ ($p=.554$).

Pause detection RT <250ms and >2.5 SDs from the condition mean were removed for each participant separately. The groups did not significantly differ on the number of outliers removed ($F(2, 73)=.96, p=.387, \eta_p^2=.026$). Participants were excluded from analyses if they had a mean accuracy score of <70% (i.e., two participants with dyslexia and two C2 controls). Therefore, the final analyses were based on 21 children with dyslexia (DY), 29 CA controls and 22 C2 controls. RT's were analysed for correct responses only.

The RT data (see Table 3) were entered into a 3 (Group: DY, CA, C2) x 2 (Condition: competitor, control) x 3 (Session: Day 1, Day 2, 1-week) mixed-design ANOVA. In general, RTs lengthened across sessions (Session, $F(2, 138)= 9.43, p<.001, \eta_p^2=.120$). Although the group with dyslexia had the slowest RTs and the CA controls had the fastest RTs, the main effect of Group did not reach significance ($F(2, 69)= 3.05, p=.054, \eta_p^2=.081$). The main effect of Condition also

did not reach significance ($F(1, 69) = 2.41, p = .125, \eta_p^2 = .034$), suggesting no overall difference in RT between competitor and control conditions.

The Shapiro-Wilk test was used to explore the normality of the distributions of the lexical competition effects (the difference between RT for competitors and controls). There were no significant deviations from normality in any group at any timepoint. Figure 3 shows the lexical competition effects for each group in each session. While the C2 controls showed a significant competition effect on Day 2 ($t(21) = 2.37, p = .027$) but not on Day 1, in line with previous research, surprisingly neither the CA controls nor the group with dyslexia showed a significant competition effect at any time point. While, numerically, the group with dyslexia consistently showed smaller competition effects than the CA controls, there was enormous variability in RT's and the three-way (Session x Condition x Group) interaction did not reach significance ($F(4, 138) = .96, p = .429, \eta_p^2 = .027$). None of the other interactions or main effects were significant (all p 's $> .05$).

Sleep Measures

Do children with dyslexia show sleep architecture differences? Sleep recordings for two of the CA controls were lost due to technical difficulties. A further three recordings from the CA controls and four from the group with dyslexia were not of sufficient quality for sleep scoring, most often due to scalp electrodes falling off during the night. As such, the data from overnight polysomnography are based on 24 CA controls and 19 children with dyslexia. Table 4 shows that, on average, the dyslexic group showed longer mean total sleep time ($p < .05$), but this did not survive Bonferroni correction (corrected alpha $p < .006$). There were no significant group differences for percent time

spent in any sleep stage. There were no group differences in sleep spindle power or in SWA. The group difference in spindle density failed to reach significance (in contrast with Bruni, Ferri, Novelli, Terribili, et al., 2009), although there was a trend for the group with dyslexia to show increased spindle density.

The results from the parent- and self-report measures of sleep also suggest that the two groups experience similar sleep quality. There were no significant group differences on any of the CSHQ sub-scales or the overall score (overall score: $t(47)=.27, p=.787$). Furthermore, there were no differences in average ratings of sleep quality ($t(44)=1.46, p=.152$) or number of night-time awakenings ($t(44)=.51, p=.610$) reported across the eight nights of the sleep diary, completed by the participant. Paired t-tests also revealed that sleep quality on the night of the EEG recording, as reported in the sleep diaries, was not significantly different to the average of the other seven nights in either the typically developing ($t= -1.63, p= .116$) or the dyslexic group ($t= -.018, p=.986$). This suggests that the overnight EEG recordings reflect a typical night of sleep.

Are sleep parameters related to overnight vocabulary

consolidation? Pearson's r correlations between key sleep parameters and the two measures of vocabulary consolidation (overnight change in cued recall and lexical competition) were calculated. Lexical competition was calculated from the pause detection data as [Competitor RT – Control RT]; overnight change in lexical competition was calculated as [Lexical Competition Day 2 – Lexical Competition Day 1]. To minimize the number of correlations computed, we focused on the sleep features most relevant to consolidation, and therefore of most interest, namely spindle power, spindle density and SWA (following Tamminen et al., 2010; Wilhelm et al., 2014; Wilhelm et al., 2013). In addition,

we included total sleep time to determine whether vocabulary consolidation relates more generally to time asleep, as opposed to specific markers of consolidation. Bonferroni correction was applied, giving an adjusted alpha level of $p < .0125$. The distributions of both spindle power and SWA deviated significantly from normality. As is standard for EEG power data (John et al., 1980) a log-10 transformation was used for both measures to improve normality properties for parametric analyses, although some minor skewness remained.

Log SWA ($p < .001$) was significantly correlated with overnight improvements in cued recall for CA controls (see Table 5). There was also a moderate correlation between log spindle power and cued recall for the CA control group, although this did not reach significance at the adjusted alpha level ($p = .020$). A significant correlation was also found between overnight changes in lexical competition and log spindle power (i.e., children with larger increases in lexical competition showed greater activity in the spindle frequency band) for the CA controls. In stark contrast, there were no significant correlations between sleep measures and overnight changes in cued recall or lexical competition in children with dyslexia. These correlations are plotted in Figure 4. Of particular note, the scatterplot in Figure 4(A) shows the difference in the strength of correlations between slow wave activity and overnight improvement in cued recall in the CA controls and the children with dyslexia.

Given that the magnitude of overnight change can depend on baseline performance, which differed between groups for cued recall, regression analyses were conducted predicting Day 2 cued recall and lexical competition effects while controlling for Day 1 performance. This approach was taken rather than using a relative score (e.g., (cued recall Day 2 – cued recall Day 1) / cued recall

Day 1) because such scores can add noise (given the baseline condition is not free from noise) and reduce power. Analyses were conducted for each group separately. Based on the correlation analyses, the key sleep features of interest were SWA and spindle power for cued recall, and spindle power for lexical competition. The correlation between SWA and spindle power was not significant for either group (CA controls, $r=.382$, $p=.065$; DY, $r=.123$, $p=.615$).

Table 6 shows that for CA controls SWA is a significant predictor of cued recall performance on Day 2 when controlling for recall on Day 1. Although spindle power was a moderate correlate of cued recall performance, including both in the regression model together indicates that it is SWA that largely accounts for variance in cued recall outcomes. However, for children with dyslexia, neither sleep spindle power nor SWA predicted cued recall performance after sleep. Similarly, the size of the lexical competition effect on Day 2 was predicted by spindle power when controlling for lexical competition on Day 1 in typically developing children; this was not the case for children with dyslexia. To assess whether these group differences were meaningful and reliable, two further factorial regression models were tested including Group (CA vs DY) and interaction variables (see Table 7).

Table 7 shows that, for the cued recall measure there was a significant interaction between group and SWA. This reflects the stronger relationship between SWA and Day 2 cued recall in the typically developing children compared to the children with dyslexia. For the lexical competition task, the interaction between group and spindle power was not significant, despite the apparently large differences in the correlations between spindle power and Day

2 lexical competition. This is most likely due to the large variability in the lexical competition effects in both groups.

Are differences in sleep-dependent consolidation related to continuous co-occurring features of dyslexia? We examined correlations between the key sleep-dependent consolidation measures (cued recall overnight change, lexical competition overnight change, SWA, spindle density and spindle power) and composite measures of different cognitive domains (i.e., literacy, phonology, nonverbal ability, language ability and attention). To devise the composites, we calculated z-scores for each of the standardized and questionnaire measures used, grouped them into their relevant cognitive domains and calculated an average z-score for each domain, for each child. The tests used in each composite are listed under Table 1.

There were no significant correlations between any of the cognitive composites and the sleep measures in either group (see Table 8). In the study by Bruni, Ferri, Novelli, Terribili, et al. (2009), the relationship between reading and sleep spindles was only seen on some measures. However, when we correlated the sleep measures with the individual literacy tests in each group separately, there were still no significant relationships.

Discussion

This study examined whether sleep plays an active role in vocabulary consolidation in childhood. The data suggest that whilst the overnight consolidation of newly learned spoken words is strongly associated with sleep parameters in typical development, this same association was not observed in children with dyslexia.

Previous behavioural evidence suggests that sleep is key to consolidating newly learned words in children, just as in adults (Henderson et al., 2012, 2013, 2015). Consistent with this, the typically developing children in this study showed significant overnight improvements in novel word recall after a night of sleep, which was maintained one week later. In a crucial extension to previous work, we also found that SWA and sleep spindle power on the night after training were associated with overnight gains in cued recall. Regression analyses, controlling for baseline cued recall performance, indicated that the key driver of this relationship was SWA as opposed to spindle power. However, larger overnight increases in lexical competition (as measured by slower responses to existing words e.g., “dolphin” once novel competitors have been integrated) were associated with greater spindle power on the night after learning. These data are consistent with findings from adults (Tamminen et al., 2010; Weighall et al., 2016) and suggest a degree of constancy in the underlying neurological mechanisms of vocabulary consolidation across development.

Children with dyslexia had more difficulty learning the new spoken words, as evidenced by generally lower cued recall compared to age-matched peers. This is unsurprising given the well documented phonological learning difficulties in dyslexia (Di Betta & Romani, 2006; Mayringer & Wimmer, 2000), reflected here by weaknesses on the phonological training tasks. Despite this, they showed similar overnight gains in their ability to recall the novel words to both chronological age and younger typical peers. On the surface, this suggests that children with dyslexia have intact vocabulary consolidation processes, consistent with findings from cross-modal word learning studies (Li et al., 2009; Messbauer & de Jong, 2003). However, interpretation of the consolidation patterns is

complicated by differences between the dyslexic and their chronological-age matched peers in baseline cued recall performance (prior to sleep), which allow for different degrees of consolidation in the two groups. Namely, both the age-matched controls and children with dyslexia showed a significant overnight improvement in cued recall with no further improvement at 1 week but the age-matched controls produced near-ceiling levels of performance after 24 hours, unlike the children with dyslexia. Consequently, the similar trajectories might be due to task insensitivity as performance approached ceiling in the control group, rather than true parallels in consolidation. Indeed, in comparison to a younger group of typically developing children matched on immediate recall of the novel words, the children with dyslexia showed significantly smaller gains after one week. This could suggest subtle impairments in longer-term consolidation in dyslexia. Restricted long-term consolidation of new vocabulary could compound difficulties with phonological encoding during the initial stages of vocabulary acquisition and consequently play a part in the oral language weaknesses often seen in children with dyslexia (Vellutino et al., 1995). However, it is important to replicate these differences between the children with dyslexia and the younger controls given the different training and testing environments (e.g. home vs. school) that might have led to attention and vigilance differences.

Measures of sleep architecture were similar between children with and without dyslexia. Numerically, the group with dyslexia showed higher spindle density (consistent with Bruni, Ferri, Novelli, Terribili, et al., 2009); however, group differences were not statistically significant despite a larger sample size in the present study. It should be noted that we did not incorporate a control night of sleep EEG recording into this study, which makes the lack of significant group

differences in spindle density somewhat difficult to interpret. Previous studies have shown that the coherence of slow oscillations and spindle density increases in response to word-pair learning prior to sleep (Gais, Mölle, Helms, & Born, 2002; Molle, Marshall, Gais, & Born, 2004). Thus, it is plausible that different learning experiences may have had differential effects on sleep in dyslexics and typical peers which may account for the failure to replicate Bruni et al (2009).

Despite the largely similar sleep architecture in each group, different patterns of correlation were found between sleep parameters and overnight changes in new word knowledge. In contrast to the strong association between SWA and overnight change in cued recall for the typical peers ($r = .69$), there was no correlation between SWA and overnight changes in recall in children with dyslexia ($r = .01$). This was confirmed by a regression analysis, in which we observed an interaction between group and the predictive value of SWA on cued recall performance after sleep. Similarly, the correlation data also suggest a somewhat reduced association between spindle power and lexical competition in children with dyslexia ($r = .14$) as compared to typical peers ($r = .56$). However, this was not supported by a significant interaction between group and spindle power as a predictor of lexical competition after sleep.

Whilst group differences in the magnitude of association between sleep variables and behavioural measures should be interpreted cautiously (e.g., as a consequence of small sample sizes), the view that sleep may support the consolidation of vocabulary learning to a lesser extent in children with dyslexia is consistent with previous findings from other populations with learning difficulties (e.g. Adi-Japha, Strulovich-Schwartz, & Julius, 2011; Henderson et al., 2014). For example, a recent study with adults who have accelerated forgetting

due to epilepsy demonstrated that similar patterns of consolidation on behavioural measures can mask differences in neural processes (Atherton et al., 2016): Both patient and control groups showed similar patterns of recall across all testing sessions, but whereas consolidation was positively related to slow-wave sleep duration in the controls, there was a negative relationship for patients. The authors proposed that, in patients, sleep might provide passive protection from interference, leading to overnight recall improvements, but active consolidation processes are likely disrupted. Such an explanation could account for the pattern of results observed here: Similar overnight recall improvements to controls could reflect sleep's role in protecting new vocabulary from interference but disruption to active consolidation processes could lead to restricted longer-term memory improvements, as supported by the present data. Of course, this hypothesis is only speculative at present and needs testing directly in future studies.

Well-established models of sleep and memory consolidation posit that sleep spindles and slow oscillations represent reactivations of newly learnt information (Rasch & Born, 2013). However, these reactivations are thought to be dependent upon initial encoding levels, with the association between initial encoding and the potential for sleep consolidation being argued to follow a U-shaped curve (Stickgold, 2009). Therefore, one tentative explanation for the lack of correlations between SWA and behavioural changes overnight could be that for children with dyslexia, difficulties with the encoding of new word forms could lead to inefficient tagging of memories for reactivation during sleep. The increased sleep spindle density that has been reported in dyslexia may reflect inefficiencies such that an increased amount of irrelevant information is

reactivated alongside relevant information during sleep. Therefore, for children with dyslexia, one hypothesis could be that slow oscillations and sleep spindles may not consistently reflect reactivations of learned material. Recent evidence suggests that temporal coherence between slow oscillations and sleep spindles is important for faithful reactivations arising from the hippocampus (Staresina et al., 2015), which are proposed as essential for active sleep-associated memory consolidation (Clemens et al., 2007). Future studies could directly test whether, in dyslexia, this temporal coherence is disrupted, minimizing the relationships between SWA, spindles and consolidation.

Unlike Bruni, Ferri, Novelli, Terribili, et al., (2009), we did not find that the severity of the reading impairment in dyslexia was related to increased spindle density, or any other sleep measure. In fact, there were no significant correlations between any of the cognitive measures and the sleep metrics. It is important to reiterate that we only recorded a single night of sleep that followed an intensive learning episode, and thus this night of sleep may not be representative of sleep architecture more generally. Notwithstanding this, coupled with the largely similar sleep architecture between groups, these data suggest that dyslexia is not characterized by significant global sleep differences that are responsible for the severity of the reading deficit. Rather, there appear to be more subtle differences in how sleep relates to learning and consolidation in children with dyslexia, which warrants further exploration.

Whilst there were correlations between sleep spindle power and overnight increases in lexical competition (signaling that better lexical integration is associated with sleep architecture), the expected time course of lexical competition (i.e., no effect immediately after learning, but a competition effect

after sleep; Dumay & Gaskell, 2007; Gaskell & Dumay, 2003; Henderson et al., 2015, 2014, 2013, 2012) was only observed for the young control group, and not for the dyslexic or age-matched controls. Although, it is important to note that the means for each condition and group fell within the confidence intervals of previous studies using the same stimuli and a similar design (see Henderson et al., 2012). Unfortunately therefore, these data do not allow us to test hypotheses about how children with dyslexia may differ in the time course by which a novel word is integrated with the lexicon. Previous studies have suggested that lexical competition effects are prone to variability. For example, Brown, Weighall, Henderson and Gaskell, (2012) reported lexical competition effects both immediately after learning and after a delay with children of a similar age to the CA controls; Tamminen et al., (2010) reported lexical competition effects after both wake and sleep in adults. . It is possible that there was additional noise in the lexical competition data in this study as a consequence of the wider age range of the CA controls and the dyslexic group than in comparison to the younger controls and previous studies (Henderson et al., 2015; Henderson et al., 2012). This may have introduced greater variability in RT (Kail, 1991), which could plausibly impact a task that relies on relatively small global RT differences between conditions. Perhaps more importantly, the younger control group were tested in their school, as in previous studies (Henderson et al., 2015; Henderson et al., 2012), whereas the older CA controls and the dyslexic group were tested in their homes. For a sensitive RT task vulnerable to distractions, home testing may have introduced unexpected noise into the data. Nevertheless, it is important to reiterate that these potential sources of additional variability in the present data did not prevent the anticipated correlations emerging between sleep spindle

power and overnight changes in lexical competition for the typically developing children.

This study makes seminal steps to address sleep-associated memory consolidation in dyslexia, and as such, there are a number of ways in which the methodology could be improved. For example, the lack of overnight sleep recordings for the younger control group means that we cannot rule out that the absence of an association between sleep and vocabulary consolidation in dyslexia is not due to differences in initial encoding. It is highly likely that the younger control group would have displayed similar correlations with the sleep measures to the older control group, since sleep spindles even in infants are related to overnight improvements on declarative memory tasks (e.g., Kurdziel et al., 2013). Nevertheless, it is possible that relationships between sleep and consolidation are linked to the quality of encoding in relation to developmental stage (Stickgold, 2009; Wilhelm, Metzkw-Mészáros, Knapp, & Born, 2012). Furthermore, the present study would benefit from a larger sample that allows for a sleep-wake design (e.g., see Henderson et al., 2012), in which participants are either trained in the morning or evening and retested twelve hours later, permitting the investigation of whether it is sleep (or the simple passing of time) that accounts for the group differences in vocabulary consolidation. Future, larger studies may also benefit from incorporating adaptation nights (to increase the reliability and validity of sleep recordings) and control nights (that do not follow an intense/novel learning period). Finally, while group matched designs allow specific hypotheses about developmental disorders to be tested, it is not possible to measure and control for all possible relevant variables that may differ between groups. Larger studies that examine a broad range of predictors (e.g.

puberty score, chronotype) of individual differences in memory consolidation in children may yield important insights.

In conclusion, consistent with an accumulation of previous findings, the present data provide novel evidence that sleep (namely SWA and spindle power) plays an active role in the strengthening and integration of new vocabulary in typically developing children. These data suggest that neural models of word learning and memory consolidation in adults (Davis & Gaskell, 2009; Rasch & Born, 2013), can be extended to children. However, this strong association between sleep and strengthening of new vocabulary was not observed in children with dyslexia, despite similar overnight improvements in word recall. Furthermore, consolidation over the course of a week was restricted in children with dyslexia in comparison to younger typically developing children who achieved similar levels of initial recall. Together, these data point to potential differences in the way that sleep works to support vocabulary consolidation in dyslexia, and open up exciting avenues for future research. These data also highlight the need to examine the neural processes underlying learning and memory phenomena, as similar behavioural patterns of consolidation may mask differences in the underlying mechanisms. Understanding these differences could be key to informing remediation strategies and improving language abilities in affected individuals. The present findings have broader implications for incorporating an individual differences perspective into models of sleep-associated memory consolidation, including understanding the influence of variability in initial learning on sleep-associated consolidation.

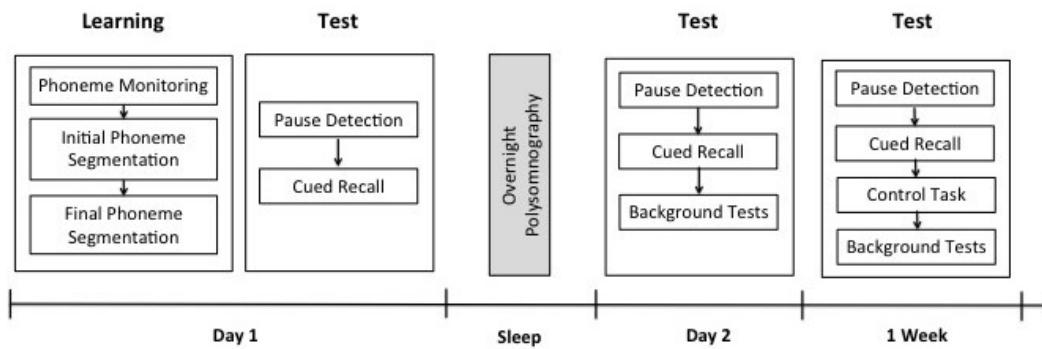


Figure 1. Outline of the study procedure. All three groups partook in the learning phase and all instances of the pause detection, cued recall and control tasks. The CA and DY groups partook in the overnight polysomnography, but the C2 group did not, and the C2 group completed a more limited set of background tests than both the CA and DY groups.

Table 1. Mean (and SD) and range scores for background measures for the age-matched peers (CA controls), children with dyslexia (DY) and the younger peers matched on immediate cued recall performance. Group differences were calculated using a one-way ANOVA and post-hoc Tukey's HSD tests, where main effects were significant. Raw scores were used for these analyses and analogous standard scores are for sample description only.

	CA-controls (N=29)		DY group (N=23)		Younger controls (C2) (N=24)		<i>F</i>
	Mean (<i>SD</i>)	Range	Mean (<i>SD</i>)	Range	Mean (<i>SD</i>)	Range	
Age (years; months)	10;09 (01;09)	07;09–13;11	10;09 (01;07)	08;03–13;09	09;01 (00;07)	07;10–09;09	11.27, <i>p</i> <.001 (CA=DY) > C2
Nonverbal IQ (raw)	24.07 (4.54)	13 - 30	21.91 (3.62)	15 - 28	17.79 (6.69)	8 - 27	10.11, <i>p</i> <.001 (CA=DY) > C2
Nonverbal IQ (T-score)	54.83 (7.23)	42 - 73	51.52 (6.24)	43 - 67	50.29 (11.91)	32 - 68	
Word reading (raw)	73.59 (11.30)	47 - 99	50.57 (19.44)	5 - 81	61.88 (11.72)	35 - 81	16.59, <i>p</i> <.001 CA > C2 > DY
Word reading (standard score)	107.45 (10.59)	92 - 127	85.57 (13.21)	53 - 107	106.42 (12.33)	85 - 127	
Nonword reading (raw)	44.00 (10.46)	15 - 60	16.70 (10.37)	4 - 41	33.21 (10.83)	18 - 54	43.08, <i>p</i> <.001 CA > C2 > DY
Nonword reading (standard score)	113.00 (14.10)	90 - 136	81.70 (8.00)	71 - 104	108.42 (13.33)	89 - 135	
Spelling (raw)	36.28 (7.09)	19 - 46	23.17 (6.97)	14 - 36	-	-	44.52, <i>p</i> <.001 CA>DY
Spelling (standard score)	105.79 (13.88)	83 - 140	77.70 (12.31)	53 - 100	-	-	
Day 1 cued recall (/16; used for matching DY and C2 groups)	7.03 (3.35)	1 - 13	3.22 (2.94)	0 - 10	3.50 (2.84)	0 - 8	12.83, <i>p</i> <.001 CA > (DY=C2)

Note. The following measures were administered: Nonverbal ability (*Matrix Reasoning*, Wechsler Abbreviated Scale of Intelligence, Wechsler, 1999). Literacy: word and nonword reading (Test of Word Reading Efficiency, Torgesen, Wagner, & Rashotte, 2012), spelling (*Spelling*, Wechsler Individual Achievement Test, Wechsler, 2005). Phonological skills: phonological awareness (*Phoneme Deletion*, Comprehensive Test of Phonological Processing (CTOPP), Wagner, Torgesen, & Rashotte, 1999), rapid automatized naming (*RAN digits* test, Warmington, Stothard, & Snowling, 2013), nonword repetition (CTOPP, Wagner et al., 1999), Language: expressive vocabulary (Expressive One Word Picture Vocabulary Test (Brownell, 2000) and sentence repetition (Clinical Evaluation of Language Fundamentals, Semel, Wiig, & Secord, 2003), Attention: stop signal reaction time (SSRT) task ('STOP-IT', Verbruggen, Logan and Stevens, 2008). Parents completed the Strengths

and Weaknesses of ADHD Symptoms and Normal Behaviour Rating Scales (SWAN; Swanson et al., 2006; return rates were 91% (n=21) for the DY group and 97% (n=28) for the CA group). Group comparisons were performed on raw scores (not standardized scores).

Table 2. Mean accuracy (% correct) on the word learning training tasks in each of the groups (standard deviation given in brackets). Group differences were calculated using a one-way ANOVA with post-hoc Tukey's HSD tests applied to significant main effects.

	CA controls	DY group	C2 controls	Group differences <i>F</i> (2,73)
Monitoring	90.70 (4.66)	82.68 (9.86)	78.69 (11.56)	12.54, <i>p</i> <.001 CA>(DY=C2)
Repetition	93.32 (4.79)	91.85 (6.70)	97.09 (2.95)	6.95, <i>p</i> =.002 (CA=)C2>DY(=CA)
Initial segmentation	96.05 (4.70)	85.69 (20.95)	97.22 (4.79)	6.49, <i>p</i> =.003 (CA=C2)>DY
Final segmentation	84.99 (10.61)	62.59 (25.19)	86.20 (13.56)	14.47, <i>p</i> <.001 (CA=C2)>DY

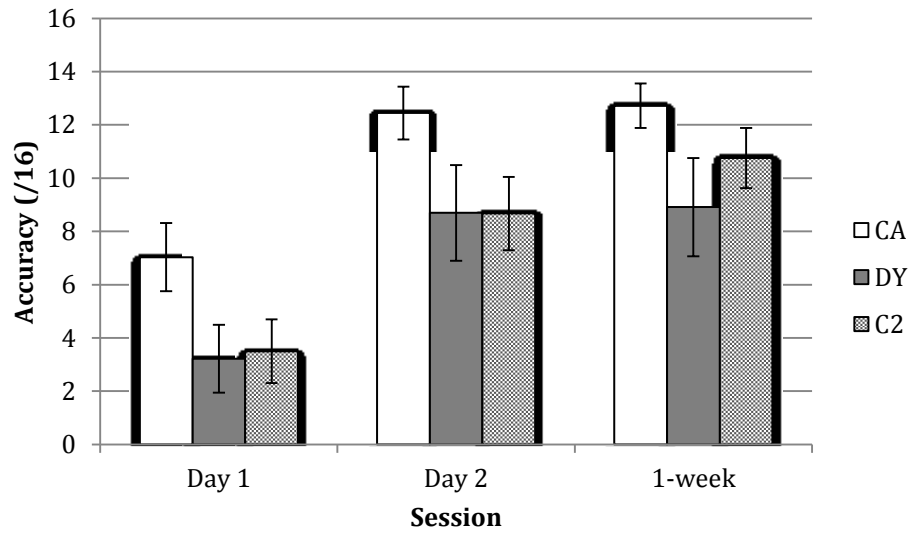


Figure 2. The mean number of correct answers in the cued recall task in each session, for each group. Error bars represent 95% confidence intervals.

Table 3. Mean reaction times in the pause detection task to competitor and control words in all groups at all time points.

Group	Session	Competitor RT (ms)	Control RT (ms)
CA controls	Day 1	954 (296)	912 (270)
	Day 2	1000 (299)	961 (301)
	1-week	1128 (386)	1062 (342)
DY group	Day 1	1169 (415)	1174 (444)
	Day 2	1197 (410)	1194 (395)
	1-week	1295 (486)	1274 (498)
C2 controls	Day 1	1081 (295)	1120 (261)
	Day 2	1052 (185)	968 (233)
	1-week	1152 (388)	1184 (378)

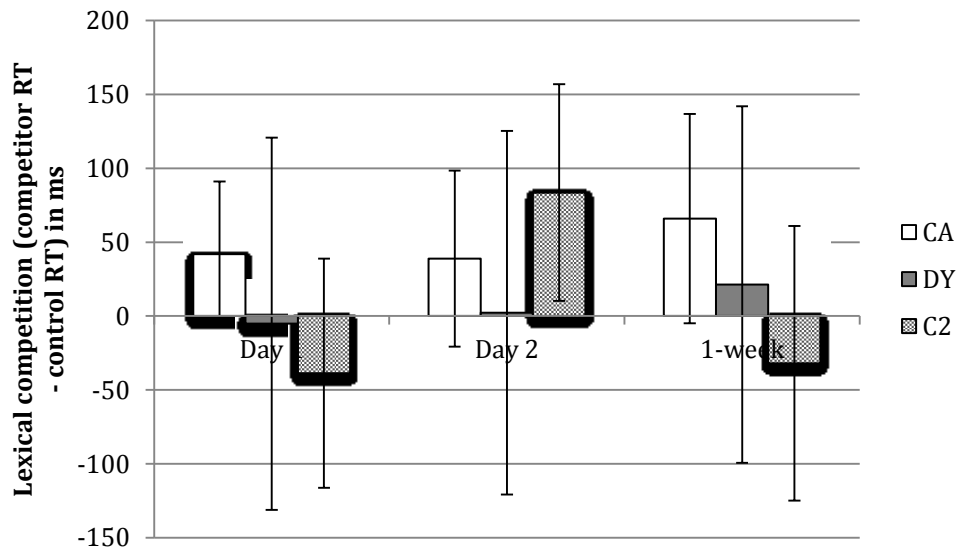


Figure 3. Lexical competition effects (difference between mean RT for competitor and control items) in all groups at all time points. Error bars represent 95% confidence intervals. The only significant competition effect, where RT's for competitor items were longer than for control items, was on Day 2 for the C2 group.

Table 4. Summary of sleep parameters with group comparisons. Bonferroni corrections applied for multiple comparisons (corrected alpha $p < .006$).

	CA controls Mean (<i>SD</i>)	DY group Mean (<i>SD</i>)	<i>F</i>	<i>p</i>
Total sleep time (mins)	513.00 (68.44)	550.84 (40.93)	4.52	.04
% N1	7.82 (3.93)	8.97 (3.92)	.92	.34
% N2	45.90 (8.10)	43.77 (5.86)	.93	.34
% SWS	31.48 (9.15)	31.48 (7.11)	.00	.99
% REM	14.80 (5.50)	15.78 (4.81)	.38	.54
Spindle density	.94 (.29)	1.22 (.69)	3.38	.07
Spindle power	7.29 (3.24)	7.57 (2.42)	.10	.75
SWA	841.05 (531.20)	949.42 (609.24)	.39	.54

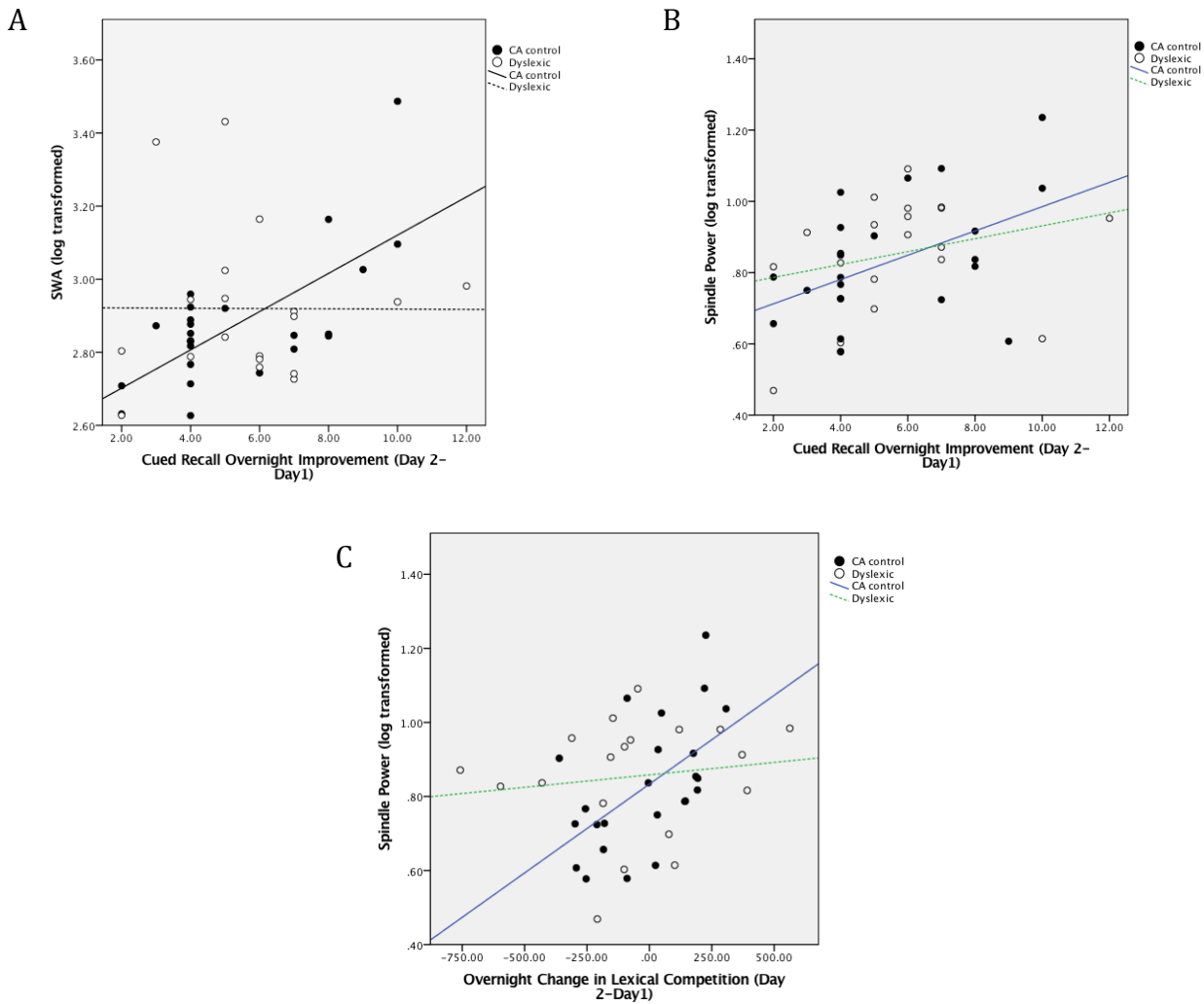


Figure 4. Scatterplots showing the relationship between slow wave activity and overnight improvements in cued recall for both the CA controls and the children with dyslexia (A), the relationship between spindle power and overnight improvements in cued recall (B) and the relationship between spindle power and overnight changes in lexical competition (C). CA controls are represented with the black dots and solid line, and the children with dyslexia and represented by the white dots and dashed line.

Table 5. Pearson's r correlations between sleep parameters and the overnight change in cued recall scores (Day 2 – Day 1) and lexical competition (Day 2 – Day 1) for each group separately.

	Total sleep time	Spindle density	Spindle power	SWA
Overnight change in cued recall				
CA	.092	.38	.47*	.69***
DY	-.18	.04	.28	-.01
Overnight change in lexical competition				
CA	.12	.44	.56**	.16
DY	.10	.01	.14	.23

* $p < .05$; ** $p < .01$; *** $p < .001$

Table 6. Hierarchical regression analyses predicting Day 2 cued recall performance (Model 1) and lexical competition effects (Competitor RT – Control RT; Model 2) from log spindle power and log SWA, when controlling for Day 1 cued recall and lexical competition effects. Results are presented for each group separately.

Typically developing group (CA controls)

Model	Step	Predictors	R^2	ΔR^2	F change	β	p
1	1	Cued recall (Day 1)	.45	.45	17.69	.74	<.001*
	2	SWA	.78	.33	15.07	.48	<.001*
		Spindle Power				.20	.092
2	1	Lexical competition (Day 1)	.01	.01	.28	.20	.287
	2	Spindle Power	.36	.34	11.22	.59	.003*

Group with dyslexia (DY)

Model	Step	Predictors	R^2	ΔR^2	F change	β	p
1	1	Cued recall (Day 1)	.59	.59	24.25**	.75**	<.001*
	2	SWA	.62	.03	.66	.01	.940
		Spindle Power				.19	.268
2	1	Lexical competition (Day 1)	.004	.004	.06	.05	.846
	2	Spindle Power	.005	.001	.02	.04	.895

Table 7. Hierarchical regression analyses predicting Day 2 cued recall performance and lexical competition effects (Competitor RT – Control RT) from log SWA and log spindle power respectively, group status and interactions between sleep features and group.

Cued Recall

Step	Predictors	R^2	ΔR^2	F change	β	p
1	Cued recall (Day 1)	.60	.60	60.32	.77	<.001*
2		.70	.11	4.46		
	SWA				.04	.743
	Group				3.00	.037*
	Group x SWA				3.09	.030*

Lexical Competition

Step	Predictors	R^2	ΔR^2	F change	β	p
1	Lexical Competition (Day 1)	.01	.01	.24	.10	.530
2		.14	.13	1.94		
	Spindle power				.03	.913
	Group				1.05	.199
	Group x Spindle power				1.23	.138

Table 8. Pearson's r correlations between the cognitive and sleep-dependent consolidation measures for the typically developing group and the group with dyslexia.

CA

	Nonverbal ability	Literacy composite	Phonological composite	Language composite	Attention composite
Spindle Density	.368	.082	.048	.279	-.173
Spindle Power	.283	.148	.164	.169	-.060
SWA	.055	.000	.032	.134	-.359
Cued recall (Day 2 – Day 1)	-.111	-.067	-.057	-.069	-.108
Lexical Comp Day 2 – Day 1)	.281	.114	.104	.125	.287

DY

	Nonverbal ability	Literacy composite	Phonological composite	Language composite	Attention composite
Spindle Density	.283	.087	.055	.018	.246
Spindle Frequency	-.091	.118	.060	-.081	.161
SWA	.019	.077	-.132	.038	-.15
Cued recall (Day 2 – Day 1)	-.24	-.07	.096	-.03	.154
Lexical Comp Day 2 – Day 1)	-.048	-.173	-.267	-.280	.183

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