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Impaired implicit sequence learning in children with developmental dyslexia

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ABSTRACT

It has been proposed that an impairment of procedural memory underlies a range of linguistic, cognitive and motor impairments observed in developmental dyslexia (DD). However, studies designed to test this hypothesis using the implicit sequence learning paradigm have yielded inconsistent results. A fundamental aspect of procedural learning is that it takes place over an extended time-period that may be divided into distinct stages based on both behavioural characteristics and neural correlates of performance. Yet, no study of implicit sequence learning in children with DD has included learning stages beyond a single practice session. The present study was designed to fill this important gap by extending the investigation to include the effects of overnight consolidation as well as those of further practice on a subsequent day. The results suggest that the most pronounced procedural learning impairment in DD may emerge only after extended practice, in learning stages beyond a single practice session.

1. Introduction

Developmental dyslexia (DD) is characterized by unexpected difficulties with reading, in the context of typical educational opportunities and intact intellectual and sensory abilities (Lyon, Shaywitz, & Shaywitz, 2003). The disorder, which has a strong genetic component (Hensler, Schatschneider, Taylor, & Wagner, 2010), has been estimated to affect about 5–12% of children (Shaywitz, Shaywitz, Fletcher, & Escobar, 1990). Children with DD have difficulties with written word recognition and phonological decoding (using letter-sound mapping knowledge to decode novel words), which is widely believed to result from underlying phonological impairments (Snowling, 2000; Stanovich, 1988; Vellutino, Fletcher, Snowling, & Scanlon, 2004).

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However, while phonological impairments are indeed found in an overwhelming majority of studies of DD (Ramus & Ahissar, 2012), other impairments, which are not easily explained by a specific phonological deficit, are also commonly reported. These include impairments of motor functions (Nicolson, Fawcett, & Dean, 2001), working memory (Smith-Spark & Fisk, 2007; Swanson, Xinhua, & Jerman, 2009), executive functions (Brosnan et al., 2002), oculomotor and visuoperceptual functions (Quercia, Feiss, & Michel, 2013), implicit sequence learning (Howard, Howard, Japikse, & Eden, 2006; Jimenez-Fernandez, Vaquero, Jimenez, & Defior, 2011; Vicari, Marotta, Menghiai, Molinari, & Petrosini, 2003), artificial grammar learning (Pavlidou, Williams, & Kelly, 2009) as well as problems with other aspects of language that appear to be primary in nature (i.e. not only a consequence of impaired reading; Lyytinen et al., 2004; Snowling, Gallagher, & Frith, 2003; Wimmer & Schurz, 2010).

This pattern of wide-ranging impairments has encouraged attempts to provide a unitary explanation for DD that may account for both the phonological and non-phonological deficits in the form of a more general underlying deficit. Such theoretical accounts include the proposals that DD is the result of impaired temporal perception (Tallal, 1980), of an abnormal development of the brain's magnocellular systems (Stein, 2001), of a deficit in attentional mechanisms (Hari & Renvall, 2001), or in general processing speed (Wolf & Bowers, 1999). One influential theoretical view, which is the focus of the present study, posits that the underlying deficit in DD is caused by a dysfunction in the procedural memory system, specifically to the cortico-cerebellar (Nicolson, Fawcett, Brookes, & Needle, 2010; Nicolson et al., 2001) and/or to the cortical–striatal (Ullman, 2004) circuits in the brain.

The procedural memory system underlies the non-declarative/implicit acquisition, consolidation and processing of skills and habits (Gabrieli, 1998; Henke, 2010; Squire & Zola, 1996; Willingham, Salidis, & Gabrieli, 2002). The system relies on a network of brain structures in which the cortico-striatal and cortical–cerebellar circuits play crucial, and largely overlapping, roles (for a review, see Doyon and colleagues, 2009).

Although previously considered to be important mainly for motor functions (such as learning how to ride a bicycle), it is becoming increasingly clear that this system also underlies a range of perceptual, cognitive, and linguistic skills. A large literature suggests that the procedural memory system plays a crucial role in the learning and computation of sequences (Aldridge & Berridge, 1998; Knowlton, Mangels, & Squire, 1996; Poldrack, Prabhakaran, Seger, & Gabrieli, 1999; Saint-Cyr, Taylor, & Lang, 1988; Willingham et al., 2002). This system also appears to be important for other functions, including statistical learning (McNealy, Mazziotta, & Dapretto, 2010; Reeder, Newport, & Aslin, 2013; Saffran, Aslin, & Newport, 1996), probabilistic classification learning (Poldrack et al., 2001; Poldrack & Rodriguez, 2004), and tasks tapping complex learned motor skills (Ullman & Pierpont, 2005). Accumulating evidence indicates that procedural memory also underlies the learning and use of rule-governed aspects of grammar, across syntax, morphology and phonology (Conway, Bauernschmidt, Huang, & Pisoni, 2010; Conway & Pisoni, 2008; Dominey, Hoen, Blanc, & Lelekov-Boissard, 2003; Karuza et al., 2013; Teichmann, Dupoux, Kouider, & Bachoud-Levi, 2006; Ullman, 2001, 2004; Ullman et al., 1997; Ullman & Pierpont, 2005).

An extensively used task for the study of non-language procedural memory, specifically implicit sequence learning, is the serial reaction time (SRT) task (Nissen & Bullemer, 1987). In this task, participants are typically shown four boxes or circles arranged horizontally across a computer screen. Whenever a stimulus appears in one of the four positions, subjects are to press one of four corresponding response keys as quickly and accurately as possible. In the implicit version of this task, participants are not told that the stimuli are presented according to a fixed sequence (as opposed to the explicit version, in which the sequential pattern is verbalized and memorized prior to practice). Sequence learning is operationalized as improvements in the accuracy and/or reaction times of responses to the sequence, as compared to randomly ordered items introduced as a control condition at the end of practice. When administered as an implicit task, learning in the SRT task appears to be largely, though not completely, incidental and non-conscious (Howard & Howard, 1992; Willingham, Nissen, & Bullemer, 1989).

A fundamental aspect of sequence learning in the SRT task, and of procedural learning more generally, is that it takes place over an extended time-period. This period may be divided into distinct stages on the basis of both behavioural characteristics and neural correlates of performance (Debas et al., 2010; Hauptmann, Reinhart, Brandt, & Karni, 2005; Korman, Raz, Flash, & Karni, 2003; Orban et al., 2010; Robertson, Pascual-Leone, & Miall, 2004).

Typically, an initial fast acquisition stage, characterized by a rapid improvement in performance (as evidenced by a decrease in both response speed and errors), is followed by a gradual decrease in the learning rate and a trend towards an asymptote (Hauptmann et al., 2005; Korman et al., 2003). This asymptotic shape of the learning curve has been suggested to reflect a saturation of learning that appears to be necessary for consolidation processes to occur normally (Hauptmann & Karni, 2002; Hauptmann et al., 2005; Karni et al., 1998). Consolidation refers to the process by which an initially labile memory trace becomes more robust and resistant to interference (Doyon et al., 2009; Robertson et al., 2004). Sometimes consolidation involves an actual increase in performance, without further practice, a phenomenon referred to as off-line learning (Hauptmann et al., 2005; Nemeth et al., 2010; Song, 2009; Song, Howard, & Howard, 2007). The end point of procedural learning is automaticity of the learned behaviour. When a skill is automatized it can be performed effortlessly even when attention is directed elsewhere (as in dual task situations; Seger & Spiering, 2011).

Brain imaging studies suggest that implicit sequence learning in the SRT task depends largely on procedural memory brain structures, in particular the striatum, cerebellum, associated motor cortical regions, as well as portions of prefrontal and parietal cortex (Grafton, Hazeltine, & Ivry, 1995; Peigneux et al., 2000; Rauch et al., 1997; for a review, see Doyon and colleagues, 2009). In addition, recent studies have highlighted a role for the medial temporal lobe in sequence learning

(Albouy et al., 2008; Gheysen, Van Opstal, Roggeman, Van Waelvelde, & Fias, 2011; Schendan, Searl, Melrose, & Stern, 2003; Simon, Vaidya, Howard, & Howard, 2012) under both explicit and implicit conditions.

Importantly, the neural correlates of sequence learning have been shown to be modulated by the amount of practice with the task; whereas early learning is characterized by a rather widespread activation pattern that includes the medial temporal lobe, prefrontal cortex, striatum and cerebellum, studies including extended practice, and later learning stages, suggest an increasingly important role for the striatum in learning stages beyond the fast acquisition stage (Doyon et al., 2009; Rieckmann & Backman, 2009; Rieckmann, Fischer, & Backman, 2010; Simon et al., 2012).

Studies investigating implicit sequence learning in DD using variants of the SRT task have not yielded consistent results. Although the majority of published studies have found a sequence learning deficit in adults (Du & Kelly, 2013; Howard et al., 2006; Menghini, Hagberg, Caltagirone, Petrosini, & Vicaria, 2006; Stoodley, Harrison, & Stein, 2006) and children (Jimenez-Fernandez et al., 2011; Vicari et al., 2003, 2005) with DD, intact performance has also been reported (Deroost et al., 2010; Kelly, Griffiths, & Frith, 2002; Russeler, Gerth, & Munte, 2006).

It has been proposed that the type of sequence used as well as the time interval examined, could account for some of this inconsistency (Du & Kelly, 2013; Orban, Lungu, & Doyon, 2008). Nevertheless, previous studies have focused almost exclusively on learning in a single practice session, and the fast acquisition stage, while neglecting overnight consolidation and later learning. This narrow experimental focus is troublesome since the effect of DD on performance in later learning stages should be of theoretical as well as clinical and pedagogical interest.

The present study extended the examination of procedural learning in children with DD to include not only an initial practice session, but also overnight consolidation and further practice on a subsequent day. To our knowledge, our study is the first to investigate these important, and potentially theoretically informative, aspects of procedural memory functions in children with DD. In addition, we used a variant of the SRT task that has not previously been used with children with DD, the alternating serial reaction time (ASRT) task (Howard et al., 2004; Howard & Howard, 1997).

The ASRT task has an important advantage over the SRT tasks used in previous studies; in the ASRT task random trials are alternating with trials following the fixed sequence throughout the task, e.g. 1-r-2-r-4-r-3 (where the numbers correspond to locations on the screen and r stands for random locations among the four positions). The alternating structure in this design allows for an assessment of sequence specific learning separate from general motor skill learning (i.e. the overall improvement in response speed that is due to practice with the task). Moreover, it is possible to examine sequence learning continuously throughout the task, rather than only at a single point at the end of the task. The present study may thus provide new information about the nature of a potential procedural learning deficit in DD.

2. Method

2.1. Ethics statement

The study was approved by the ethical review board in the city of Uppsala. All parents or guardians provided informed written consent; children provided informed written assent and received a cinema ticket for their participation.

2.2. Participants

Twelve children with developmental dyslexia (DD) and 17 typically developing (TD) control children participated in the study. The groups were matched for sex, age and handedness (Table 1).

Children with DD were recruited via speech–language pathology clinics in the cities of Stockholm, Uppsala, Gävle and Västerås, in Sweden. All children with DD had been independently tested and diagnosed with dyslexia by a certified speech–language pathologist within 1.5 years prior to participation. The TD group consisted of a subset of children who were

Table 1
Participant demographics and cognitive characteristics.

Variable	DD ($n = 12$)		TD ($n = 17$)		Comparison	
	Mean	*SD*	*Mean*	*SD*	*t*	*p*
Age in years	11.0	0.71	11.1	0.68	0.388	.701
Sex (f/m)	5/7		5/12		$\chi^2 = 0.47$	.494
Handedness	85.1	16.4	92.2	10.2	1.42	.167
PIQ	87.9	12.1	97.1	15.0	1.74	.093
Phonological decoding	1.75	0.87	5.24	1.15	8.87	<.001
Orthographic reading	1.92	1.16	5.76	1.15	8.84	<.001
NWR	106	5.2	111	5.4	2.29	.030
TROG	57.9	30.1	75.6	18.4	1.96	.060
PPVT	152	16.4	160	13.5	1.47	.154

Abbreviations: PIQ, Performance IQ (Raven, 1998); NWR, Nonword repetition (Wass et al., 2008); TROG, Test for Reception of Grammar (Bishop, 1982); PPVT, Peabody Picture Vocabulary Scale – Third Edition (Dunn & Dunn, 1997). *Note*: Handedness scores are based on the Edinburgh Handedness Inventory (Oldfield, 1971).

recruited from schools in and around the cities of Stockholm and Uppsala as part of a larger study on memory and language in typically developing children. All children in the study were reported by their parents to be monolingual Swedish-speaking, to have normal (or corrected to normal) vision and hearing, and to have no known cognitive or motor impairment, except for reading problems in the DD group.

Participants were given a set of behavioural tests in order to characterize their vocabulary (Dunn & Dunn, 1997), grammatical comprehension (Bishop, 1982; Holmberg & Lundälv, 2002), verbal working memory (nonword repetition; Wass et al., 2008), orthographic reading, phonological decoding, and performance IQ (PIQ; Raven, 1998) As expected, there were significant group differences for the two reading measures as well as for verbal working memory. By contrast, the two groups did not differ significantly in vocabulary, grammatical comprehension or PIQ, although the differences in both grammatical comprehension and PIQ approached significance (Table 1).

Performance IQ was included for descriptive rather than exclusionary purposes in this study since we wanted our DD sample to reflect the population of children who are clinically identified and diagnosed with DD in Sweden. In line with evidence suggesting that there is a weak, if any, relationship between PIQ and the reading problems characteristic of dyslexia (Ferrer, Shaywitz, Holahan, Marchione, & Shaywitz, 2010; Rispens, van Yperen, & van Duijn, 1991), PIQ is not used as an exclusionary factor for a clinical diagnosis of DD in Sweden. The PIQ range in the DD group was 70–115 and the TD range was 80–140.

The two reading tests were paper and pencil Swedish adaptations (Olofsson, 2003) of the computerized phonological decoding and orthographic reading tasks developed by Olson, Forsberg, Wise, and Rack (1994). In the phonological decoding test, the task was to decide, and underline with a pencil, which one of three or four pseudo-words was a pseudo-homophone of a real word. (i.e. "sounds" like a real word). The score corresponds to the number of correctly identified pseudohomophones within 2 min, with a maximum score of 80. In the orthographic reading test, participants were asked to underline the true word in true word-pseudohomophone pairs. Because the phonological codes for the pairs were identical, the word and its pseudo-homophone would be pronounced the same in Swedish. Thus, in order to make a correct response subjects had to use word-specific orthographic knowledge. The score was the number of correctly chosen words in 2 min, with a maximum score of 120.

All TD children had stanine scores ≥ 4 out of 9 on both reading tests (corresponding to performance at or above – 0.75 *SD*). All children in the DD group had stanine scores of ≤ 3 on both tests, except for one child who had a stanine score of 5 on the orthographic reading test. The performance of this child is consistent with previous evidence suggesting that the phonological decoding problems characteristic of DD can sometimes occur together with intact or even superior orthographic skills (Siegel, Share, & Geva, 1995).

2.3. Stimuli and procedure

Sequence learning was tested with a version of the Alternating Serial Reaction Time (ASRT) task (Howard et al., 2004; Howard & Howard, 1997). As with classic SRT tasks, a sequence of visual stimuli appeared in one of four horizontally arranged locations (indicated by open circles) on the computer screen. The stimulus was a picture of a dog. Subjects were asked to press one of four horizontally arranged buttons whenever the stimulus appeared in the corresponding location on the screen. Specifically, they were asked to "catch the dog" as quickly and accurately as possible by pressing the button corresponding to the circle in which the dog appeared (Hedenius et al., 2011). The sequence regularity was not mentioned. Subjects were instructed to use the middle and index fingers of both hands, and responses were collected with a PST Serial Response Box. Stimuli were presented (on a LCD screen), and response times were acquired, with E-Prime version 1.2.

Target locations were determined by a repeating eight-element structure in which fixed and random locations alternated. All participants received the same 8-item sequence pattern, 1r2r4r3r. That is, the dog appeared in position 1 (the left-most circle), then randomly in any of the four circles, then in position 2, and so on. On random trials, the events were sampled from a uniform distribution such that the four locations were equally likely.

Each stimulus presentation and response constituted one trial. Trials were organized into blocks of 85 trials: 5 warm-up random trials (not included in analyses) followed by 10 repetitions of the 8-item sequence (Howard et al., 2004). Short breaks were offered between blocks. Feedback was given at the end of each block, to guide subjects to an accuracy level of about 92%. All instructions and feedback were displayed visually on the screen as well as read aloud to the participants. The task was self-paced, such that the correct button had to be pressed before a new stimulus would appear on the screen. However, the experimenter controlled the beginning of each new block by clicking the mouse. Reaction time (RT) was measured from target onset to the first response. The next stimulus followed the correct response after a fixed 120 ms delay.

Testing was carried out over two separate sessions on consecutive days. On day 1, subjects completed 20 blocks (1700 trials and 200 repetitions of the 8-item sequence). Twenty-four hours later they completed 5 blocks (425 trials and 50 repetitions of the sequence).

2.4. Statistical analysis

Following previous ASRT studies, trials that constituted the final trial in "trills" (e.g. 121) and "repetitions" (e.g. 111) were excluded, as they have been shown to be associated with pre-existing response tendencies (for details, see Howard et al., 2004). For RTs, median values were calculated separately for pattern and random trials for each block and each subject. Next,

these median values were averaged across 10 consecutive blocks in session 1, and across the 5 blocks in session 2, in order to obtain mean values for pattern and random trials, again for each subject, for an early learning stage (stage 1: blocks 1–10), an intermediate learning stage (stage 2: blocks 11–20) and a late learning stage, which occurred after an overnight interval (stage 3: blocks 21–25). A similar data reduction procedure was performed on accuracy.

In the ASRT task, sequence specific learning is typically expressed as an increasing *difference* between trials that follow the sequential pattern and trials that are random. For RTs, sequence learning is seen as increasingly faster responses to pattern compared to random trials. For accuracy, sequence learning is typically expressed as a progressive decrease in the accuracy of responses to random trials whereas accuracy for pattern trials tends to remain stable throughout the task. General skill learning, on the other hand, is seen in the form of the sequence independent (across both pattern and random trials) reduction in response time as a function of practice.

Group differences in general skill learning and/or sequence specific learning were examined with 2 (group: DD vs TD) × 2 (trial type: pattern vs random) × 3 (learning stage: 1–3) mixed design ANOVAs, for RT and accuracy, with group as a between-subject variable and trial type and learning stage as within-subject (repeated measures) variables.

3. Results

3.1. Reaction time analyses

The two groups did not differ with respect to overall RT (RT in milliseconds (ms): DD *mean* = 450, *SD* = 93; TD *mean* = 433, *SD* = 59, main effect of group: $F(1, 27) < 1$). Consistent with Fig. 1, general skill learning was seen as a significant main effect of learning stage ($F(1, 54) = 84.9$, $p < .0001$, $\eta_p^2 = .759$), in which responses for both pattern and random trials became faster with practice. The lack of any group × learning stage interaction ($F(2, 54) < 1$) suggests that this effect was similar in the two groups.

Evidence for sequence specific learning was found in that RTs were significantly faster for pattern (*mean* = 438, *SD* = 73) compared to random (*mean* = 442, *SD* = 75) trials across the task as a whole (main effect of trial type: $F(1, 27) = 6.56$, $p = .016$, $\eta_p^2 = .195$). This effect was stable throughout the task and did not increase significantly with practice (non-significant trial-type × learning stage interaction: $F(2, 54) < 1$). Of particular interest here, the two groups did not differ with respect to sequence learning effects on RT (group × trial-type interaction: $F(1, 27) < 1$; group × trial-type × learning stage interaction: $F(2, 54) = 1.51$, $p = .230$, $\eta_p^2 = .053$).

In sum, the group × trial type × learning stage ANOVA with RT as the dependent variable revealed significant general skill learning that continued to develop throughout the task. Evidence for sequence specific learning was somewhat weaker as it was observed only as a main effect across the task as whole, and there was no evidence for an increase in sensitivity to the sequence regularity with practice. Notably, the RT analyses did not reveal any group differences in overall RT, general skill learning, or sequence specific learning.

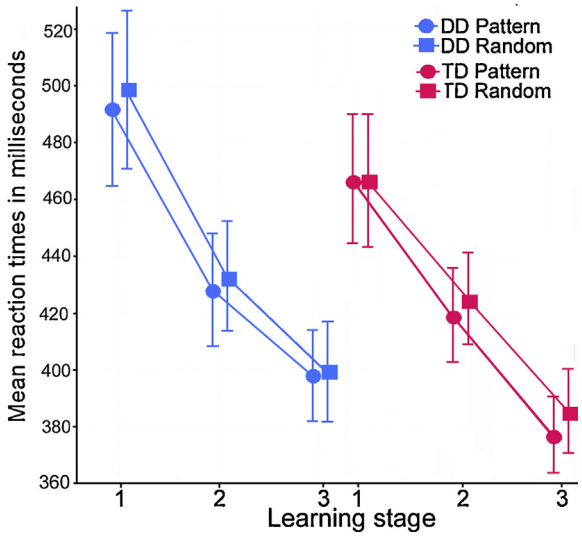

Fig. 1. Reaction times in milliseconds for pattern and random trials as a function of learning stage for the developmental dyslexia (DD) and typically developing (TD) groups. The figure displays means and standard errors for the three learning stages.

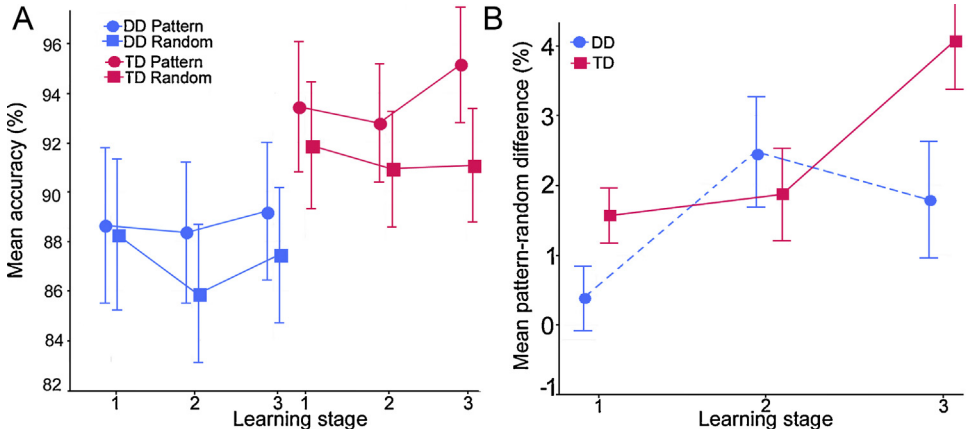

Fig. 2. (A) Accuracy in percent for pattern and random trials as a function of learning stage for the developmental dyslexia (DD) and typically developing (TD) groups. (B) Accuracy *difference* between pattern and random trials as a function of learning stage for the developmental dyslexia (DD) and typically developing (TD) groups. The figure displays means and standard errors for the three learning stages.

3.2. Accuracy analyses

The two groups performed at similar accuracy levels across the task as a whole (DD *mean* = 87.9%, *SD* = 15.2%; TD *mean* = 92.4%, *SD* = 2.73%; main effect of group: $F(1, 27) = 1.53$, $p = .227$, $\eta_p^2 = .053$; group × learning stage interaction: $F(2, 54) < 1$). Accuracy levels did not change significantly with practice (main effect of learning stage: $F(1, 27) = 2.51$, $p = .091$, $\eta_p^2 = .085$).

Fig. 2A plots the mean accuracy for pattern and random trials as a function of learning stage, and Fig. 2B plots the mean accuracy *difference* between pattern and random trials as a function of learning stage. Consistent with Fig. 2A, evidence for sequence learning, across all children, was found in that accuracy was higher for pattern (*mean* = 91.7%, *SD* = 10.1%) compared to random (*mean* = 89.6%, *SD* = 9.77%) trials (main effect of trial type: $F(1, 27) = 32.7$, $p < .0001$, $\eta_p^2 = .548$). The accuracy difference between pattern and random trials increased as a function of learning stage (trial type × learning stage interaction: $F(1, 27) = 7.04$, $p = .002$, $\eta_p^2 = .207$), suggesting that participants became increasingly sensitive to the sequence regularity with practice. Most importantly, however, there was a group difference in sequence learning (group × trial type × learning stage interaction: $F(1, 27) = 3.84$, $p = .028$, $\eta_p^2 = .124$).

The triple interaction was followed-up with between-groups one-way ANOVAs with the pattern-random accuracy difference for each learning stage as the dependent variable. These analyses confirmed the pattern suggested by Fig. 2B. There was a trend towards less sequence learning in the DD group in the first learning stage (main effect of group: $F(1, 27) = 3.82$, $p = .061$, $\eta_p^2 = .124$). At stage 2, the amount of sequence learning was highly similar in the two groups (main effect of group: $F(1, 27) < 1$). A significant group difference emerged at stage 3, with poorer learning in the DD group compared to the TD group (main effect of group: $F(1, 27) = 4.38$, $p = .046$, $\eta_p^2 = .139$).

In sum, the accuracy analyses revealed no significant group differences in the first practice session (encompassing learning stages 1 and 2), although there was a trend ($p = .061$) towards less learning in the DD group compared to the TD group in stage 1. Late in learning, by contrast, a significant group difference emerged. By learning stage 3, after extended practice and including an overnight interval, the DD group showed significantly less sequence learning compared to the TD group.

3.2.1. By-block overnight consolidation vs extended practice effects

Given that the observed accuracy-based group difference in late learning (stage 3) could be due to differences in overnight consolidation and/or in the effects of extended practice, additional analyses were performed to dissociate these effects.

A potential group difference in overnight consolidation was examined by comparing accuracy on the final block (i.e. 10 repetitions of the sequential pattern) on the first day (block 20) and the first block on the second day (block 21) with a 2 (group: DD vs TD) × 2 (trial type: pattern vs random) × 2 (block: 20 vs 21) ANOVA. Consistent with Fig. 3, which suggests similar performance in the two groups in both block 20 and 21, this analysis revealed no group differences in overnight consolidation of the learned sequence (group × trial type × block interaction: $F(1, 27) < 1$). There was evidence for sequence learning in the form of a significant main effect of trial type ($F(1, 27) = 7.40$, $p = .011$, $\eta_p^2 = .215$), reflecting overall higher accuracy for pattern compared to random trials, with a similar effect in both groups (group × trial type interaction: $F(1, 27) < 1$). There were no indications of off-line sequence learning effects, as the accuracy difference between pattern and random trials did not increase overnight (trial type × block interaction: $F(1, 27) < 1$). However, overall accuracy increased significantly overnight (main effect of block: $F(1, 27) = 20.4$, $p = .0001$, $\eta_p^2 = .430$), possibly reflecting fatigue effects in the final block of session 1. The overnight increase in overall accuracy was also very similar in the two groups (group × block interaction: $F(1, 27) < 1$).

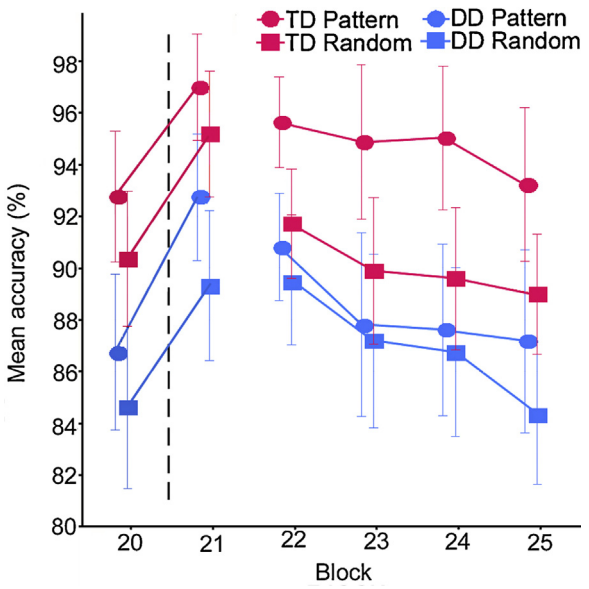

Fig. 3. Mean accuracy for pattern and random trials as a function of practice blocks for the developmental dyslexia (DD) and typically developing (TD) groups. The figure displays means and standard errors for the final block on day 1 (block 20) and the 5 blocks on day 2 (blocks 21–25). The vertical line between blocks 20 and 21 represents a 24 h interval during which no further practice was given. Group differences were examined for overnight consolidation (blocks 20–21) and for the effects of extended practice on day 2 (blocks 22–25). See Section 3.2.1, for a detailed description of the analyses and results.

The lack of any significant group differences in the overnight consolidation of the learned sequence suggests that the observed group difference in learning stage 3 may be due to a differential effect of further practice on day 2 in the two groups. This prediction was supported by a group × trial type × block ANOVA for the remaining four blocks (blocks 22–25) in learning stage 3, again with accuracy as the dependent variable (Fig. 3). In addition to a significant main effect of trial type ($F(1, 27) = 24.7$, $p < .0001$, $\eta_p^2 = .478$), this ANOVA produced a group × trial type interaction ($F(1, 27) = 7.19$, $p = .012$, $\eta_p^2 = .210$), reflecting a smaller pattern-random accuracy difference (i.e. sequence learning effect) in the DD group compared to the TD group (DD *mean* = 0.0139, *SD* = 0.0337, TD *mean* = 0.0464, *SD* = 0.0310). The lack of a group × trial type × block interaction ($F(3, 81) < 1$) suggests that the TD advantage remained stable across the final four blocks. Overall accuracy and reaction times did not differ between groups in either the consolidation analysis or the extended practice analysis (all *p*s > .208).

3.3. Correlation between implicit sequence learning and individual reading ability

Next, the relationship between implicit sequence learning and individual reading ability was examined with a correlation analysis. A composite measure of reading ability was calculated from the standardized raw scores of the phonological decoding and orthographic reading tests (Table 1). This composite reading score was plotted against the accuracy difference between pattern and random trials at the final learning stage (stage 3). As can be seen in Fig. 4, this analysis revealed a

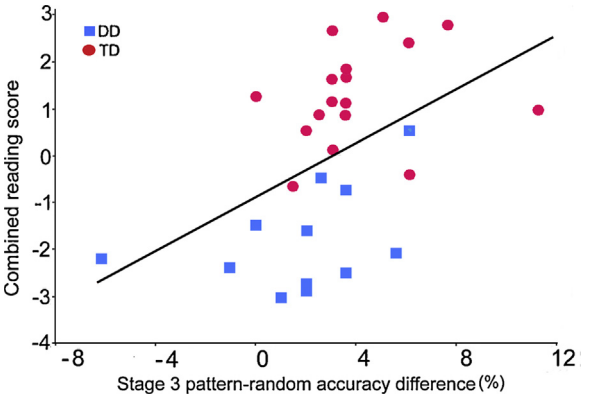

Fig. 4. Scatterplot showing the correlation between implicit sequence learning (pattern-random accuracy difference) at stage 3 and the combined word reading score.

significant positive correlation between implicit sequence learning and individual reading ability, independent of diagnostic category ($r = .470$, $p = .010$, $r^2 = .221$).

Because inspection of Fig. 4 suggests that a score in the low-left corner of the scatterplot could potentially be driving this correlation (the pattern-random accuracy difference score for this individual was −3.0 *SD* from the mean of all children, and −2.4 *SD* from the mean of the DD group), we repeated the correlation analysis with this child excluded. The correlation was still significant ($r = .426$, $p = .024$, $r^2 = .182$), thus suggesting a robust association between sequence learning and reading skill as a continuous variable.

3.4. Control analyses

In order to exclude the possibility that the group difference in sequence learning was an effect of lower general intellectual ability in the DD compared to the TD group (as indicated by the near-significant group difference in PIQ), additional analyses were performed in which the effect of PIQ was statistically controlled for. Specifically, PIQ was added as a covariate in a 2 (group) × 3 (learning stage) ANCOVA performed on the accuracy difference between pattern and random trials (i.e. as depicted in Fig. 2B). This analysis produced an identical significance pattern compared to the analyses without covariates reported above (stage 3 main effect of group: $p < .05$). Thus, the group difference in sequence learning was not explained by lower general intellectual ability in the DD group. Importantly, the results held also when excluding the child identified as an outlier in the correlation analysis above (stage 3 main effect of group: $p < .05$).

4. Discussion

The present study examined previously untested aspects of procedural memory, specifically of implicit sequence learning, in children with DD compared to typically developing (TD) control children. Our study went beyond earlier research in examining learning stages beyond a single practice session, and by allowing for a continuous assessment of sequence specific learning separate from general skill learning.

Children with DD showed a selective deficit in implicit sequence learning while general skill learning was normal. In addition, although there was a trend towards poorer learning in the DD group in the first learning stage, the sequence learning impairment became significant only after extended practice, including an overnight interval. Further analyses suggested that the impairment may not be related to overnight consolidation, but rather to the effect of further practice on day 2.

Control analyses revealed that the observed group difference in sequence learning was not explained by lower general intellectual ability in the DD group. In addition, the fact that overall reaction times and general skill learning were comparable in the two groups suggests that the results were not due to group differences in fatigue, general processing speed, or attention.

Sequence learning in the ASRT task is typically seen as increasingly faster responses to pattern compared to random trials (RT based learning measure) and/or an increasing number of errors on random trials while accuracy for pattern trials typically remains more stable (accuracy based learning measure). The pattern of an increasing frequency of errors on the unpredictable trials with practice is typical when probabilistic sequences are used. Participants often report that their fingers seem to "take over", leading them to make more "oops" errors. Unbeknownst to them, however, these errors reflect an increasing sensitivity to the sequential regularity. In the present study, significant effects of reading skill as a categorical and continuous variable were found for the accuracy based learning measure only, and not for the RT based measure. This finding is in line with two previous studies examining sequence learning in adults with DD using either the ASRT task (Howard et al., 2006) or the Triplet Learning Task, which contains a similar probabilistic sequence structure (Bennett, Romano, Howard, & Howard, 2008). In both studies, the accuracy based measure of sequence learning was shown to be more strongly related to reading ability compared to the RT based measure. Interestingly, both of these studies on adults with DD report correlations between individual reading skill and the accuracy based learning score at the end of the task, that are strikingly similar to the correlation observed in the present study performed on children.

Overall, the findings support the notion of an association between procedural memory deficits and DD. The results are in line with previous studies reporting implicit sequence learning impairments in children (Jimenez-Fernandez et al., 2011; Vicari et al., 2003, 2005) and adults (Du & Kelly, 2013; Howard et al., 2006; Menghini et al., 2006; Stoodley et al., 2006) with DD. They extend previous findings by showing, for the first time, that the sequence learning impairment in children with DD may be most pronounced in learning stages beyond a single practice session and the early, fast acquisition phase. Interestingly, a similar pattern of results has previously been reported for children with specific language impairment, a condition which often co-occurs with DD (Hedenius et al., 2011).

The majority of previous studies of implicit sequence learning in DD have found a group difference early in learning, i.e. within a single practice session. Our study did not replicate these findings in that the group difference in early learning was only marginally significant in our stage 1 ($p = .061$). However, it is possible that this reflects a lack of power due to the relatively small number of participants. Nevertheless, the lack of significant effects early in learning is interesting in light of the inconsistent results of previous studies. Indeed, all of the three studies that have reported intact implicit sequence learning in children with DD (Deroost et al., 2010; Menghini et al., 2010; Waber et al., 2003) have focused on a relatively narrow practice interval (ranging from 24 to 104 repetitions of the sequential pattern compared with the 250 repetitions in

the present study) given within a single practice session. In light of the fact that a significant group difference in the present study emerged only in day 2, after more than 200 repetitions of the sequence, it is possible that group differences may have been revealed if the studies above had included a wider practice interval.

Impairment on a non-language task, such as the one used in the present study, challenges any account that posits a deficit *specific* to phonological processing as the underlying cause of DD (Snowling, 2000; Stanovich, 1988; Vellutino et al., 2004). Per definition, any such account would predict intact non-language learning in DD, or at least, would not predict any correlation between non-language sequence learning and individual reading skill, as was found here. The present findings are thus in line with data suggesting that the neurocognitive deficits in children with DD may go beyond a selective phonological impairment (see Section 1).

Our findings are compatible with the procedural memory deficit view of DD (Nicolson et al., 2001, 2010; Ullman, 2004). The findings may also be compatible with the magnocellular theory of DD (Stein, 2001), since this theory includes the prediction that cerebellar functions, and thus aspects of procedural memory, are impaired in the disorder. Notably, the fact that a significant DD impairment emerged relatively late in learning may point to a striatal rather than, or in addition to, a cerebellar dysfunction (Doyon et al., 2009). As noted in the introduction, the relative contribution of different brain structures/circuits to sequence learning has been shown to change as learning progresses through the different stages. Brain imaging studies suggest that while the striatum, the cerebellum, and the medial temporal lobe show simultaneous learning-dependent activity in the fast acquisition stage (with the medial temporal lobe often being recruited very early in learning), activation in the cerebellum typically decreases with practice and is usually no longer detectable when the sequence is well learned (Doyon et al., 2002, 2009; Jueptner, Frith, Brooks, Frackowiak, & Passingham, 1997; Rieckmann & Backman, 2009; Rieckmann et al., 2010; Simon et al., 2012). The striatum, by contrast, remains activated through the asymptotic learning as well as the consolidation and late learning stages (Doyon, Owen, Petrides, Sziklas, & Evans, 1996; Lehericy et al., 2005). This pattern of brain activation has been taken as evidence for an increasingly critical role of the striatum in the learning stages beyond the fast acquisition stage (Bennett, Madden, Vaidya, Howard, & Howard, 2011; Doyon et al., 2009). However, any claims about the neural basis of the observed sequence learning deficit in the present study are speculative.

Even though the majority of studies to date support the notion of an association between poor reading and implicit sequence learning deficits, the nature of this association remains to be elucidated. The association revealed here, and elsewhere, may reflect one of several possible relationships. One possibility is that a general procedural memory deficit underlies the core phonological problems in DD as well as a range of previously reported cognitive and motor deficits (Nicolson et al., 2010), including that of implicit sequence learning observed in the present study. This is the proposal of the procedural memory deficit view (Nicolson et al., 2001, 2010; Ullman, 2004). On this view, a procedural memory deficit may affect reading both directly, through skill automatization problems, and indirectly, via phonological processing problems (Nicolson & Fawcett, 2011).

Another possibility is that reading and procedural memory deficits tend to co-occur in DD but are causally unrelated. That is, for reasons yet unknown, a procedural memory deficit may be more common in dyslexic children compared to their typically developing peers, but this impairment does not cause the reading problems, or affect them in any substantial way. Such a view is supported by evidence suggesting that sensori-motor impairments, such as those predicted by both the procedural deficit and magnocellular views, may neither be necessary nor sufficient to cause the reading problems observed in DD (Ramus, 2003; White et al., 2006). However, while this view is compatible with the observed group difference in sequence learning, it is somewhat difficult to reconcile with the linear relationship between reading and implicit sequence learning, independent of diagnostic category, observed here, and in other studies (Bennett et al., 2008; Howard et al., 2006).

A third possibility is that the core phonological deficit and the procedural memory deficit are causally unrelated but that the co-occurrence of these deficits may lead to reading problems that are severe enough to draw pedagogical and clinical attention. On this view, procedural learning problems may not cause the phonological impairment in DD but may contribute independent variance to individual reading ability by affecting, for example, the ability to extract orthographic sequential regularities and the automatization of reading skills. Such a "double deficit" view has previously been proposed for phonological processing problems and rapid naming ability (Wolf & Bowers, 1999). This view would be in line with studies of children at risk for dyslexia, in which it has been shown that phonological problems alone are not sufficient to cause severe reading problems (Gallagher, Frith, & Snowling, 2000; Snowling et al., 2003; Wimmer & Schurz, 2010).

The design of the present study does not allow us to specify the nature of the relationship between procedural memory and reading skill, but only to demonstrate that such a relationship exists. Future studies, with a longitudinal design starting at birth, and including a wider range of linguistic, cognitive and motor tasks, may help shed light on this issue.

The present study has various methodological limitations that may be addressed by future research. One such limitation is that sample sizes were relatively small. As noted above, this may have affected the power to detect a group difference in early learning. Thus, the present findings need to be replicated with larger samples in order to ensure their generalizability. In addition, future studies of procedural memory consolidation and extended practice effects in children with DD would benefit from including a more direct test of consolidation (such as the amount of interference by a new or random sequence; Robertson et al., 2004), as well as more learning trials for the examination of further practice on day 2. Such paradigms may help shed further light on the DD deficit in late learning demonstrated here.

Future studies would also benefit from neuroimaging data to support the assumption that children rely on procedural memory for sequence learning in the ASRT task. Although previous fMRI-studies have indeed revealed activation of procedural memory brain structures associated with ASRT learning in adults (Bo, Peltier, Noll, & Seidler, 2011), the lack of

data from studies of children performing this task precludes any strong conclusions regarding brain correlates of learning in this study.

Keeping these limitations in mind, the present findings lend support to the notion that DD is associated with a procedural memory dysfunction. Further, the results emphasize the importance of studying not only initial learning, but also memory consolidation and longer-term learning in children with DD in order to obtain a deeper understanding of learning and memory functions in affected children. Such knowledge would potentially be of great importance to the development of effective pedagogical and therapeutic remediation strategies.

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