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The nature and specificity of paired associate learning deficits in children with dyslexia

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ABSTRACT

We report three experiments investigating the specificity and nature of paired associate learning (PAL) deficits in children with dyslexia. Experiments 1 and 2 compared children with dyslexia and age-matched controls across the following stimulus–response mapping conditions, designed to dissociate crossmodal and verbal demands: visual–verbal, verbal–verbal, visual–visual, and verbal–visual. Children with dyslexia exhibited deficits in visual–verbal and verbal–verbal PAL only. Experiment 3 investigated the stage of learning in which PAL deficits arise by separating the verbal learning and associative learning components of a visual–verbal PAL task. Results revealed an item-specific relationship between phonological form learning and later associative learning success. Visual–verbal PAL deficits were fully accounted for by the preceding deficit in phonological form learning. Together, our results show that PAL deficits in dyslexia are not a consequence of difficulties with associative learning; instead, they are best characterized as deficits in phonological form learning. The implications of these findings for theories of reading development and dyslexia are discussed.

Introduction

Paired associate learning (PAL) is thought to tap basic associative learning mechanisms by requiring the pairing of a stimulus and response item in memory. Evidence suggests, however, that not all PAL tasks are created equal when it comes to the relationship with reading ability. Decades of research have documented visual–verbal PAL (i.e., pairing a visually-presented symbol with a verbal output, normally a nonword) deficits in children with dyslexia, despite age-appropriate performance in nonverbal tasks such as visual–visual PAL (i.e., pairing a visually-presented symbol with another visually presented symbol) (Liberman, Mann, Shankweiler, & Werfman, 1982; Messbauer & de Jong, 2003; Vellutino, Steger, Harding, & Phillips, 1975; Vellutino, Steger, & Pruzek, 1973). This reliable pattern of PAL deficits in dyslexia is observed across languages, despite variations in orthographic, phonological, and morphological complexity (Li, Shu, McBride-Chang, Liu, & Xue, 2009; Mayringer & Wimmer, 2000; Messbauer & de Jong, 2003). Indeed, across the range of abilities, visual–verbal PAL shares a robust and specific relationship with reading skill (Hulme, Goetz, Gooch, Adams, & Snowling, 2007; Litt, de Jong, van Bergen, & Nation, 2013).

In the dyslexia literature, visual–verbal PAL deficits have typically been interpreted within the phonological deficit hypothesis of dyslexia (Mayringer & Wimmer, 2000; Messbauer & de Jong, 2003, 2006; Vellutino, Scanlon, & Spearing, 1995; Vellutino et al., 1973, 1975). This prominent theory views difficulties in phonological processing as the primary cognitive-level cause of dyslexia (e.g., Fowler, 1991; Snowling, 1995; Snowling & Hulme, 1994). Difficulties with phonological processing can give rise to deficits on any task that places demands on the phonological system, including phonological awareness, verbal short term memory, speech perception and production, naming, and of course, reading and spelling. The finding

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of visual–verbal PAL deficits in children with dyslexia, in the absence of visual–visual PAL deficits fits comfortably within this framework (Messbauer & de Jong, 2003; Vellutino et al., 1973, 1975).

Evidence for the phonological nature of PAL deficits comes from the finding that the errors produced by children with dyslexia are more likely to be phonological, rather than associative in nature (Mayringer & Wimmer, 2000; Messbauer & de Jong, 2003). Additionally, differences between dyslexic and control groups in visual–verbal PAL can largely be accounted for by differences in phonological processing skills (Messbauer & de Jong, 2003, 2006). The importance of phonological skills in determining visual–verbal PAL performance is also observed in typically developing readers. For example, individual differences in phonological skills predict visual–verbal PAL performance (de Jong, Seveke, & van Veen, 2000; Windfuhr & Snowling, 2001). Furthermore, de Jong and colleagues (2000) showed that training in phonological awareness significantly improved visual–verbal PAL performance in kindergartners.

A phonological locus of PAL deficits can also account for the graded pattern of PAL deficits observed in dyslexia. When the verbal response to be learned is a nonword, PAL deficits are both robust and reliable, but when the response to be learned is a word, deficits are more equivocal (Elbro & Jensen, 2005; Mayringer & Wimmer, 2000; Messbauer & de Jong, 2003, 2006; Vellutino, Bentley, & Phillips, 1978; Vellutino, Scanlon, & Bentley, 1983; Vellutino et al., 1975). Whether visual–verbal PAL deficits emerge for words seems to depend upon the nature of the verbal stimuli and the degree to which they tax the phonological system (e.g., de Jong et al., 2000; Mayringer & Wimmer, 2000; Messbauer & de Jong, 2003, 2006; Vellutino et al., 1995). For example, deficits are observed when stimuli comprise abstract or low frequency words, but are typically absent for concrete words, or words that are high in frequency or imageability (Elbro & Jensen, 2005; Messbauer & de Jong, 2006; Samuels & Anderson, 1973; Vellutino et al., 1995). Such findings suggest that both the phonological demands of the stimuli (e.g., frequency, complexity) and the availability of non-phonological information (e.g., visual, semantic) influence the likelihood of observing PAL deficits in dyslexia. Viewed in this manner, the pattern of deficits observed in visual–verbal PAL seems a natural consequence of the degree to which learning hinges on phonological processes.

Clearly, the verbal component of visual–verbal PAL is crucial to explaining PAL deficits in dyslexia. However, evidence also suggests that visual–verbal PAL may index broader abilities than phonological processing alone. For example, Wimmer, Mayringer, and Landerl (1998) reported PAL deficits in children with dyslexia even when they were matched to controls for phonological awareness. Additionally, visual–verbal PAL accounts for unique variance in reading ability in typically developing readers, above and beyond two of the best known cognitive predictors of reading ability: phonological awareness and rapid automatized naming (RAN) (Hulme et al., 2007; Litt et al., 2013; Warmington & Hulme, 2012; Windfuhr & Snowling, 2001). Thus, although it is tempting to conclude that deficits in visual–verbal PAL (in the absence of deficits in visual–visual PAL) arise as a consequence of the verbal demands that are inherent in visual–verbal PAL, this conclusion is premature. Additional differences between these tasks must not be overlooked. One important difference is that visual–verbal PAL requires crossmodal (between-modality) mappings, whereas visual–visual PAL requires unimodal (within-modality) mappings. Because the contrast between these PAL mapping conditions confounds verbal and crossmodal demands, it does not allow for firm conclusions regarding the locus of PAL deficits in dyslexia.

Some researchers have argued that the crossmodal nature of visual–verbal PAL is central to its association with reading, as both require the establishment of visual-phonology mappings (Hulme et al., 2007; Warmington & Hulme, 2012; Windfuhr & Snowling, 2001). According to their view, visual–verbal PAL taps a crossmodal mapping mechanism akin to that operating in connectionist models of reading, in which learning occurs via the alteration of connection weights between orthographic and phonological units (Hulme et al., 2007; Seidenberg & McClelland, 1989; Snowling, 2000; Windfuhr & Snowling, 2001). Crucially, it is the learning of the *associations* between visual and verbal stimuli, rather than the learning of the verbal stimuli itself, that is proposed to drive the relationship between visual–verbal PAL and reading. The primary evidence for the crossmodal hypothesis comes from a study by Hulme and colleagues (2007), in which the authors examined the specificity of the relationship between visual–verbal PAL and reading in typically developing children. The design included three mapping conditions: visual–verbal, visual–visual, and verbal–verbal. The verbal–verbal PAL task allowed Hulme et al. to evaluate whether crossmodal learning (e.g., visual–verbal), rather than unimodal verbal learning (e.g., verbal–verbal) drives the relationship with reading ability. Although both visual–verbal and verbal–verbal PAL correlated significantly with reading skill, visual–verbal PAL was the only PAL task to predict unique variance in reading ability, consistent with there being a specific role for crossmodal mechanisms in reading.

In contrast, an experiment by Litt and colleagues (2013) is at odds with a crossmodal account of the PAL–reading relationship. The authors included four mapping conditions: visual–verbal, verbal–verbal, visual–visual, and verbal–visual. The addition of verbal–visual PAL to the design allowed for a strong test of the crossmodal hypothesis: if crossmodal associative learning is the crucial component of the task, both visual–verbal and verbal–visual PAL should show robust relationships with reading ability, as both require crossmodal mappings. The results were not in accordance with this hypothesis. Both visual–verbal and verbal–verbal PAL predicted unique variance in reading above and beyond phonological awareness and RAN, whereas verbal–visual and visual–visual PAL were unrelated to reading ability. The lack of a relationship between verbal–visual PAL and reading is difficult to reconcile with the crossmodal hypothesis, as this task, like visual–verbal PAL, has a crossmodal mapping demand. Instead, the results strongly suggest that verbal output demands are responsible for the PAL–reading relationship: both visual–verbal and verbal–verbal PAL explained variance in reading ability. Moreover, there was no evidence to support the view that visual–verbal PAL shares a unique relationship with reading ability. The shared variance between visual–verbal and verbal–verbal PAL fully accounted for the observed relationship with reading; visual–verbal PAL could not explain unique variance in reading ability above this shared "verbal output" component.

Litt and colleagues reported data from typically-developing children and thus it remains possible that PAL deficits in dyslexia result from deficient crossmodal associative learning. Very few studies have addressed this issue, however. Vellutino and colleagues (1975) tested children with dyslexia in both visual–verbal PAL and visual–auditory PAL, a task in which associations are made between visual symbols and oral sounds (e.g., humming, coughing, puckering lips). Although both tasks required crossmodal associations (and oral production), deficits only emerged in visual–verbal PAL. Additionally, Torgesen and Murphey (1979) showed that these results held even when the oral output required was more complex, comprising a sequence of sounds. This is perhaps not surprising given that visual–auditory PAL does not tap the phonological system. However, while these findings clearly rule out a global deficit in between-modality associative learning, they remain difficult to interpret. Although it is clear that the linguistic component of the task is imperative, it is not clear whether it is the verbal component alone, or the specific demand on visual–verbal associative learning that is responsible for the observed deficits.

Distinguishing between these two theoretical accounts is crucial to informing our understanding of the relationship between PAL deficits and the reading and spelling difficulties characteristic of dyslexia. Learning to read and spell undoubtedly requires the establishment of orthography-phonology mappings, yet it is unclear whether the mappings themselves impede learning in dyslexia. If PAL deficits reflect difficulties with crossmodal associative learning, this would provide evidence for a dyslexic deficit that is specifically implicated in learning to read (via the formation of orthography-phonology mappings). On the other hand, if PAL deficits arise from the phonological demands of the tasks, this would suggest that PAL deficits are neither a reflection of poor associative learning, nor specific to reading. In other words, the difficulties children with dyslexia experience in the acquisition of orthography-phonology mappings would be viewed as a manifestation of underlying phonological deficits, rather than a difficulty learning the mappings themselves. Moreover, these phonological deficits would be expected to compromise the development of both written and oral language skills (e.g., expressive word learning), when these skills rely on the integrity of the phonological system.

We present three experiments designed to evaluate the specificity and locus of PAL deficits in dyslexia. In Experiments 1 and 2 we sought to determine whether PAL deficits are a consequence of verbal or crossmodal task demands. To distinguish between these two theoretical accounts, we used the factorial design introduced by Litt et al. (2013), which allowed us to dissociate crossmodal and verbal demands in driving poor PAL performance in dyslexia. Experiment 3 expanded on the findings of Experiments 1 and 2 by investigating the stage of learning in which PAL deficits arise. Specifically, we were interested in whether PAL deficits stem from difficulties in phonological form learning, or whether they emerge in the process of binding verbal information to a referent in memory. To determine the stage at which deficits arise in dyslexia, we separated the verbal learning and associative learning components of a visual–verbal PAL task.

Experiment 1

The primary aim of this experiment was to dissociate between crossmodal and verbal demands in driving PAL deficits in dyslexia. To do so, we tested the following PAL mapping conditions: visual–verbal, verbal–verbal, visual–visual, and verbal–visual. We predicted that if PAL deficits reflect crossmodal associative learning, deficits should emerge in both visual–verbal and verbal–visual PAL. On the contrary, if PAL deficits are a consequence of verbal deficits, impaired performance should emerge in both visual–verbal and verbal–verbal PAL. As outlined above, underlying phonological deficits are likely to lead to poor performance on any PAL task with significant verbal demands. Thus we might expect verbal–verbal PAL to be impaired as a consequence of underlying phonological deficits. However, these demands may not fully account for PAL deficits in dyslexia; crossmodal demands may pose an additional and specific constraint on PAL performance. In this case, we would expect impairments in visual–verbal, verbal–visual, and verbal–verbal PAL. Note that although such a finding would implicate both verbal and crossmodal demands in poor PAL performance, it would still provide support for deficient crossmodal learning in dyslexia.

Method

Participants

Children with dyslexia (aged 8–12 years) were recruited from the local Dyslexia Association on the basis of attendance at a weekly tuition scheme for children with dyslexia. Enrollment in the tuition scheme is dependent upon a literacy screening battery administered by the local Dyslexia Association. Thus, all children attending the tuition scheme have been previously identified as having significant literacy difficulties that warranted specialist teaching outside of school. Typically developing readers (aged 8–12 years) were recruited from two state primary schools serving socially-mixed catchment areas in Oxfordshire, UK. All participants spoke English as a first language. Upon obtaining consent, the children were screened via a battery of five standardized assessments of reading, two literacy-related measures, and a test of nonverbal reasoning. These allowed us to select children to participate in our experiment (see below for criteria):

Nonverbal reasoning. The Wechsler Abbreviated Scale of Intelligence (WASI) Matrix Reasoning (WASI; Wechsler, 1999) subtest provided an estimate of cognitive ability.

The test consists of 35 abstract spatial reasoning problems in which an array is presented with one missing section. Participants must choose the item that belongs in the array from five options.

Passage reading accuracy. The York Assessment of Reading for Comprehension (YARC; Snowling et al., 2009) provided a measure of passage reading accuracy. In this test, children are instructed to read short passages aloud as quickly and accurately as possible.

Passage reading rate. The reading rate subscale of the YARC provided a measure of reading fluency. Standardized scores are computed based on the total time (in seconds) taken to read each passage.

Single word reading. The Single Word Reading Test (SWRT; Foster, 2007) was administered as a measure of untimed single word reading ability.

Word reading fluency. The Sight Word Efficiency subtest from the Test of Word Reading Efficiency (TOWRE; Torgesen, Wagner, & Rashotte, 1999) measured word reading fluency. The raw score is defined as the total number of words (out of 104) that can be read correctly in 45 s.

Nonword reading fluency. The Phonemic Decoding Efficiency subtest from the TOWRE (Torgesen et al., 1999) measured nonword reading fluency. The raw score is the total number of nonwords read correctly in 45 s.

Phoneme deletion. The Elision subtest of the Comprehensive Test of Phonological Processing (CTOPP; Wagner, Torgesen, & Rashotte, 1999) assessed phonological awareness (PA). In this test children are asked to delete single phonemes from words (e.g., "Say *bold* without the *b*").

Rapid automatized naming. In the rapid digit naming subtest of the CTOPP, six digits (e.g., 4, 7, 8, 5, 2, 3) are repeated six times throughout a 36-item matrix arranged in four rows of nine items. Children must name the items in the matrix sequentially, starting at the top of the page from left to right as quickly as possible until they have named all 36 items. Children complete two separate arrays that differ only in the order the digits are presented. The time (in seconds) taken to name the items in each array is summed to produce a total naming time.

Group selection

Participants were included in the dyslexia group if they (a) had a formal diagnosis of dyslexia and (b) obtained standard scores one standard deviation or below the mean on three or more of the five measures of reading. This yielded a final group of 18 children with dyslexia (9 boys, 9 girls). A control group of 18 typically developing readers (11 boys, 7 girls) was next selected and matched to the dyslexic group for chronological age and nonverbal reasoning. All children in this group scored within the average range on five or more of the seven literacy measures. The descriptive characteristics of the groups are displayed in Table 1. As expected, the children with dyslexia performed more than one standard deviation below the mean across measures of reading and reading-related skill. In contrast, the typically developing readers performed within the normal range.

Design

The design was within-participant with all children completing all four PAL mapping conditions: visual–verbal, verbal–verbal, visual–visual, and verbal–visual. Children were tested in four sessions across a four-week period. Only one condition was tested per week to minimize interference between conditions and the order in which children completed the conditions was counterbalanced.

Materials and procedure

Children learned six stimulus–response pairs per mapping condition. The stimulus–response pairs were fixed, such that all participants learned the same pairings for each condition. Within each of the four conditions, the presentation of the stimulus–response pairs was randomized in every trial. Stimulus–response pairs for all experiments reported in this paper can be found in the online supplementary materials (see Appendix A).

Visual stimuli. The abstract symbols were chosen from extinct written languages (e.g., Akkadian). Fifteen adults rated 54 potential stimulus items on a scale of 1–10 (with 10 being most difficult) for degree of verbalization (i.e., "how easy is it to assign a verbal label or description to this item?"), and ease of drawing (i.e., "how easy is it to draw this item?"). The 24 symbols rated the hardest to verbalize were assigned to four sets (i.e., two sets of stimulus items, and two sets of response items) that comprised the visual stimuli for the PAL conditions. The visual stimuli were matched for degree of verbalization across the four sets, $F(3,42) = 2.11$, $p = .114$. Additionally, we ensured that the two sets comprising visual response items were matched for ease of drawing, $F(1,14) = 1.26$, $p = .280$.

Verbal stimuli. The verbal stimuli comprised spoken phonotactically legal CVC nonwords, chosen from the ARC

Table 1
Descriptive characteristics of dyslexic and control groups.

Measures	Dyslexic		Control	
	M	*SD*	*M*	*SD*
Age (years)	10.14	1.07	10.01	0.87
WASI Matrices[a]	53.22	5.20	51.51	6.11
Selection measures				
Sight Word Efficiency[b]	82.72	11.71	105.44	7.67
Phonemic Decoding Efficiency[b]	79.33	8.33	108.61	8.90
Single Word Reading[b]	76.61	6.35	105.11	10.32
Passage Reading Accuracy[b]	77.06	4.92	103.72	7.31
Passage Reading Rate[b]	78.59	8.60	110.39	10.31
Reading-related skills				
Phoneme Deletion[c]	6.89	1.84	11.06	2.49
RAN Digits[c]	6.78	1.80	10.00	2.35

[a] *T* score, $M = 50$, $SD = 10$.
[b] Standard score, $M = 100$, $SD = 15$.
[c] Scaled score, $M = 10$, $SD = 3$.

Nonword Database (Rastle, Harrington, & Coltheart, 2002). Each of the short vowel sounds (i.e., a, e, i, o, u) and one long vowel sound (e.g., ee) were used once per stimulus set. Additionally, no two items shared consonant positioning (i.e., first or last sound) within a stimulus set, in an effort to minimize phonological similarity between items. The verbal stimuli were always presented aurally; children were not exposed to their orthographic forms.

Paired associate learning. Children were tested individually in a quiet room in their school. The experiment was run using EPrime Version 2.0 (Schneider, Eschman, & Zuccolotto, 2007) presented on a Dell laptop. In each condition, the six stimulus–response pairs were presented in two presentation blocks before the test trials began. In the first presentation block, children were instructed to repeat the response item after each presentation to ensure that the responses could be accurately reproduced. For verbal response items, children were instructed to repeat each nonword aloud; for visual response items, children were instructed to draw the abstract symbol inside a booklet of blank index cards, one item per page to prevent them from referring to previous responses throughout a block. If a nonword or symbol was reproduced incorrectly the experimenter corrected the child by saying "that's not quite right," and then proceeded to repeat the correct nonword or draw the correct response in the booklet for the child. In the second presentation block the children were asked to focus on learning the pairs of items rather than reproduce the response item. This followed from pilot testing in which instructing participants to focus on the stimulus–response pairings resulted in better learning than instructing them to reproduce the response items in the second presentation block.

Five blocks of learning trials followed the presentation blocks. Each block consisted of six trials, one for each stimulus–response pair. In each trial, the stimulus item was presented on the computer and the participant was then asked to provide the appropriate response (e.g., "What goes with hib?"). Depending on the output required, the child either drew the symbol in the booklet or verbally produced the nonword that corresponded to the stimulus item. This procedure required production for all conditions regardless of modality, thus avoiding a common confound in the literature in which recall is required for verbal stimuli and recognition for visual stimuli (Messbauer & de Jong, 2003). Regardless of the accuracy of the child's response, the correct answer was presented on the computer immediately following each trial. This procedure was repeated for each trial across all five blocks. All participants completed all trials, and accuracy per trial and total learning across trials was recorded. The final score was calculated as the total number of correct trials across all five blocks (maximum score of 30).

Results

The mean scores and standard deviations across the four PAL mapping conditions for both reader groups are shown in Table 2. Learning across blocks by group and mapping condition is displayed in Fig. 1.

Upon initial observation it is clear that overall performance varied by mapping condition, with both groups of children finding verbal output conditions (visual–verbal, verbal–verbal) more difficult than the visual output conditions (visual–visual, verbal–visual). This observation is in line with the pattern of performance reported in other studies of PAL in children of this age (Hulme et al., 2007; Litt et al., 2013). Despite this general trend in performance, however, it is apparent that the dyslexic group performed less well than the control group in visual–verbal and verbal–verbal PAL. This pattern immediately suggests that PAL deficits stem from verbal, rather than crossmodal or general associative learning difficulties, as performance is comparable to the control group in verbal–visual and visual–visual PAL. To examine the effects of PAL modality and output on group performance, the results were analyzed in a logistic linear mixed-effects model implemented in the lme4 package in R (Bates, Maechler, & Bolker, 2011). This analysis was chosen over traditional ANOVA for two reasons. First, such models allow for crossed random effects, such that variability can be accounted for across both participants and items simultaneously (Baayen, Davidson, & Bates, 2008). Second, ANOVA can lead to spurious results when the dependent variable constitutes a binary outcome, even when classic transformations have been applied (Jaeger, 2008).

Table 2
Mean (*SD*) performance on PAL tasks in Experiment 1.

	Dyslexic		Control	
	M	*SD*	*M*	*SD*
Visual–verbal PAL (max. 30)	3.00	2.97	6.72	4.50
Verbal–verbal PAL (max. 30)	2.67	2.25	5.11	3.92
Visual–visual PAL (max. 30)	10.28	5.63	10.06	5.40
Verbal–visual PAL (max. 30)	11.39	5.18	12.61	4.51

In all mixed-effects analyses reported below we utilized a maximal random effects structure (i.e., by-subject and by-item slopes for each fixed main effect and interaction). Failing to include random slopes in models with substantial by-participants or by-items differences can lead to inaccurate inferences and impose limitations on the generalization of the fixed effects (see Barr, Levy, Scheepers, & Tily, 2012; Roland, 2009). Additionally, we centered all fixed effects around their mean, thus minimizing collinearity and aiding in model interpretation. As suggested by Baayen et al. (2008), *p*-values for mixed logistic regression models were calculated based on the Wald *Z* statistic. Finally, we report odds ratios (OR) as an index of effect size (e.g., Fleiss, 1994; Haddock, Rindskopf, & Shadish, 1998).

The effect of PAL mapping modality and output demand on experimental group was tested in a model with modality (crossmodal, unimodal), output (verbal, visual), group (dyslexic, control), and block (1–5) as fixed effects, and participant and item as random effects. Fixed effects are shown in Appendix A. Results revealed main effects of output (OR = 4.91, 95% CI [3.25 7.42], $Z = 7.56$, $p < .001$), group (OR = 0.62, 95% CI [0.40 0.96], $Z = -2.14$, $p = .03$), and block (OR = 1.30, 95% CI [1.20 1.40], $Z = 6.32$, $p < .001$), but no main effect of modality (OR = 0.79, 95% CI [0.51 1.22], $Z = -1.07$, $p = .283$). The main effect of block indicates that learning occurred across tasks and groups; however, there were no significant interactions with any of the fixed effects. In contrast to the predictions of the crossmodal

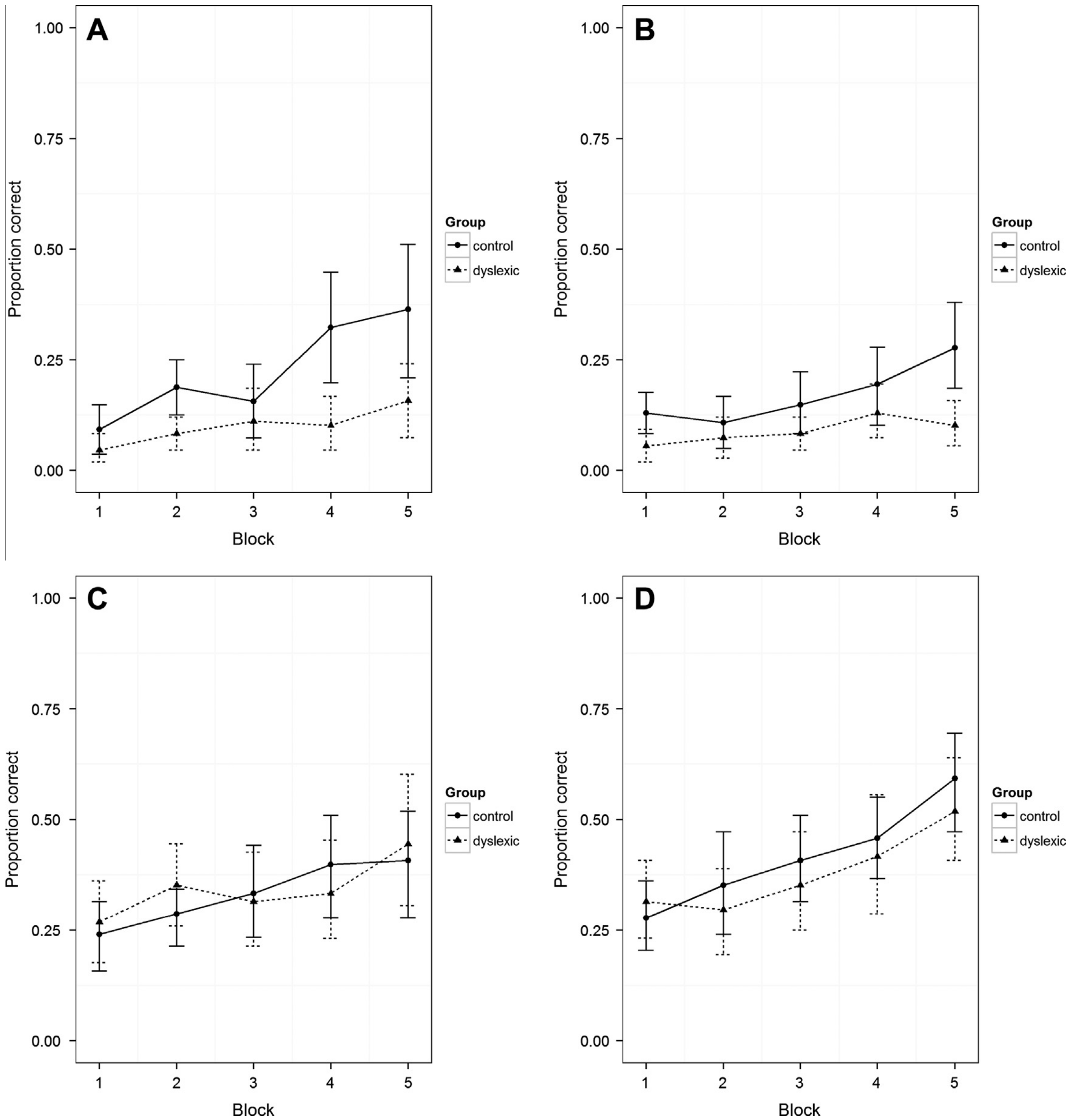

Fig. 1. Proportion correct across learning blocks for the dyslexic and control group in Experiment 1: (A) visual–verbal PAL, (B) verbal–verbal PAL, (C) visual–visual PAL, or (D) verbal–visual PAL. Error bars represent 95% confidence intervals.

hypothesis, the interaction of group and modality was not significant (OR = 1.28, 95% CI [0.67 2.44], $Z = 0.75$, $p = .453$). Instead, there was a significant interaction between output and group (OR = 2.28, 95% CI [1.29 4.04], $Z = 2.83$, $p = .005$). Tests of simple main effects revealed that children with dyslexia performed significantly poorer than chronological age controls in verbal output conditions (OR = 0.49, 95% CI [0.26 0.93], $Z = -2.20$, $p = .028$) but equally well in visual output conditions (OR = 1.00, 95% CI [0.69 1.46], $Z = -0.00$, $p = .998$).

Discussion

Experiment 1 examined whether the locus of PAL deficits in children with dyslexia resides at the level of modality or output. Consistent with the literature, children with dyslexia were impaired in visual–verbal PAL, but performed as well as age-matched controls in visual–visual PAL (Messbauer & de Jong, 2003; Steger, Vellutino, & Meshoulam, 1972; Vellutino et al., 1975). The addition of the verbal–verbal and verbal–visual conditions to our design made it possible to dissociate between a verbal and cross-modal explanation of visual–verbal PAL deficits in dyslexia. The results provided clear support for a verbal explanation of PAL deficits: children with dyslexia were selectively impaired in visual–verbal and verbal–verbal PAL, but not visual–visual or verbal–visual PAL. The lack of deficits in verbal–visual PAL in the face of robust visual–verbal PAL deficits provides direct evidence against a theory of deficient crossmodal mapping in dyslexia.

Although the data speak clearly to our experimental question, the relatively low performance levels across PAL mapping conditions indicate that children found the tasks difficult. This is especially apparent in the verbal-output tasks, which may have been limited in their sensitivity to capture the full range of abilities. Floor effects are of particular concern in the dyslexic group, in which there is little evidence of learning across trials in visual–verbal and verbal–verbal PAL. It is important to note that although floor effects may influence the accuracy of the estimate of the population mean and effect size, reliable differences between the dyslexic and control groups still emerged. Thus, despite the difficulty of the task, the control group still learned significantly more than the dyslexic group in visual–verbal and verbal–verbal PAL. Perhaps more germane to our argument is the fact that group differences did not emerge in verbal–visual PAL, a task in which performance levels signal little concern. If children with dyslexia are specifically impaired in forming crossmodal associations, we would expect performance differences in this condition. Instead, the results suggest that children with dyslexia struggle only in PAL tasks with a verbal output component. Nevertheless, low performance levels and differences in the difficulty of the PAL tasks are limitations of the current study, and these will be addressed in Experiment 2.

Experiment 2

The results of Experiment 1 favor a verbal account of PAL deficits in dyslexia. However, the extent of these deficits and their relation to learning over time is difficult to discern due to low performance levels, particularly in the verbal-output conditions. Experiment 2 was designed to capture a wider range of performance by increasing performance levels across PAL conditions. To do so, we decreased the number of items per mapping condition from six to four in visual–verbal and verbal–verbal PAL and from six to five in visual–visual and verbal–visual PAL. Given the differences in task difficulty between the PAL conditions in Experiment 1, we chose to use a different number of items in the verbal-output and visual-output tasks to better equate performance across the four conditions. An additional aim of Experiment 2 was to assess the nature of the errors made by the children, and to ask whether the errors made by children with dyslexia differ from those made by typically developing children. We hypothesized that if PAL deficits are driven by verbal demands, as Experiment 1 suggests, the errors made by children with dyslexia should be phonological in nature (i.e., phonologically distorted word, correct pairing). If however, PAL deficits are caused by a specific difficulty with associative learning, we would expect a higher proportion of associative learning errors (i.e., correct word form, incorrect pairing). To examine this question, we tracked the errors made by participants in visual–verbal and verbal–verbal PAL. Finally, given the frequent co-occurrence of dyslexia and specific language impairment (SLI), it is possible that children with dyslexia perform worse than controls due to concomitant oral language deficits. To evaluate this possibility, we measured expressive vocabulary in both groups. Additionally, we measured digit span as a proxy of verbal short term memory, as group differences in this factor could potentially limit learning of the phonological forms in visual–verbal and verbal–verbal PAL.

Method

Participants

Children with dyslexia were recruited from three private schools for children with specific learning difficulties in Boulder, Denver, and Littleton, Colorado. Informed consent forms were only sent home with children who were officially identified by the school as having a specific reading disability in the absence of diagnosed attentional or behavioral difficulties. Typically developing readers aged 8–13 were recruited from a private primary school in Boulder, Colorado and a public primary school in Centennial, CO. All participants came from a middle class or upper-middle class background and spoke English as a first language.

Informed consent was obtained for 20 children with a documented history of dyslexia, and 23 typically developing readers. Upon obtaining consent, the children were assessed with a cognitive battery consisting of the following reading and reading-related measures: Word Reading Fluency (TOWRE), Nonword Reading Fluency (TOWRE), Phoneme Deletion (CTOPP), Rapid Automatized Naming (CTOPP), and Digit Span (CTOPP). In addition to these measures, children were given assessments of nonverbal reasoning (WASI), and Expressive Vocabulary (from the Clinical Evaluation of Language Fundamentals, CELF-4). Given the extensive documented history of the children with dyslexia, the test battery was utilized as a confirmatory measure; that is, children remained in the sample unless they scored above the 25th percentile on three or more of the reading-related measures. This resulted in a final sample of 18 children with dyslexia (13 boys, 5 girls). We next selected a control group of 18 typically developing readers (12 boys, 6 girls), matched to the dyslexic group on age and nonverbal reasoning. All children in this group had no documented history of special educational needs, and scored above the 25th percentile on three or more of the literacy measures. Descriptive characteristics of the groups are shown in Table 3. The dyslexic group scored significantly below the controls on all reading and reading-related measures, except expressive vocabulary, $t(34) = 1.53$, $p = .137$. Additionally, it should be noted that although the groups differed in digit span, performance in the dyslexic group was well within the normal range.

Materials and procedure

The design was identical to that of Experiment 1, except where noted below. All participants completed the four PAL mapping conditions, with one condition tested per week. The stimuli were the same as those utilized in Experiment 1. However, the number of stimulus–response pairs in each condition was reduced. Participants learned four stimulus–response pairs across five blocks for visual–verbal and verbal–verbal PAL (maximum score of 20 per condition), and five stimulus–response pairs across five

Table 3
Descriptive characteristics of dyslexic and control groups in Experiment 2.

Measures	Dyslexic		Control	
	M	*SD*	*M*	*SD*
Age (years)	11.13	1.34	10.57	1.00
WASI Matrices[a]	51.12	7.04	52.28	8.60
Expressive Vocabulary[b]	40.39	6.52	44.67	9.96
Sight Word Efficiency[c]	81.75	8.58	110.00	13.18
Phonemic Decoding Efficiency[c]	84.19	5.79	107.44	12.43
Phoneme Deletion[d]	7.56	2.07	11.00	2.30
RAN Digits[d]	5.94	1.39	10.71	2.14
Digit Span[d]	9.28	0.66	11.78	0.76

[a] *T* score, $M = 50$, $SD = 10$.
[b] Raw score (max. 54).
[c] Standard score, $M = 100$, $SD = 15$.
[d] Scaled score, $M = 10$, $SD = 3$.

blocks for visual–visual and verbal–visual PAL (maximum score of 25 per condition). The procedure was identical to that of Experiment 1 with the addition of each verbal response being transcribed, allowing a detailed error analysis to be made.

Results

The mean scores and standard deviations across the four PAL mapping conditions for both reader groups are shown in Table 4. Learning across blocks by group and mapping condition is displayed in Fig. 2.

As in Experiment 1, the data were analyzed with logistic linear mixed-effect modeling with maximal random effects structure. The effects of mapping modality and output demand on PAL performance was tested in a model with modality (crossmodal, unimodal), output (verbal, visual), group (dyslexic, control), and block (1–5) as fixed effects, and participant and item as random effects. Fixed effects are displayed in Appendix A. Results revealed significant main effects of output (OR = 2.60, 95% CI [1.48 4.57], $Z = 3.33$, $p < .001$) and block (OR = 1.44, 95% CI [1.32 1.57], $Z = 8.30$, $p < .001$), but no main effect of group (OR = 0.68, 95% CI [0.43 1.09], $Z = -1.59$, $p = .113$) or modality (OR = 0.88; 95% CI [0.52 1.50], $Z = -0.48$, $p = .635$). A significant interaction between block and group (OR = 0.79, 95% CI [0.69 0.90], $Z = -3.59$; $p < .001$) was qualified by follow-up tests indicating the groups only differed on blocks 4 (OR = 0.57, 95% CI [0.35 0.92], $Z = 2.46$, $p = .014$) and 5 (OR = 0.36, 95% CI [0.19 0.69], $Z = 4.06$, $p < .001$). Replicating the key findings of Experiment 1, the interaction between output and group was significant (OR = 2.27, 95% CI [0.85 1.21], $Z = 2.76$, $p = .006$), whereas there was no interaction between modality and group (OR = 1.01; 95% CI [0.63 1.62]; $Z = 0.06$, $p = .954$). Tests of simple main effects revealed that the interaction between group and output was driven by a significant group difference in performance for verbal output (OR = 0.32, 95% CI [0.19 0.54], $Z = -4.20$, $p < .001$), but not visual output PAL conditions (OR = 0.96, 95% CI [0.54 1.69], $p = .877$).

Table 4
Mean (*SD*) performance across PAL tasks in Experiment 2.

	Dyslexic		Control	
	M	*SD*	*M*	*SD*
Visual–verbal PAL (max. 20)	5.83	3.87	9.39	3.88
Verbal–verbal PAL (max. 20)	5.50	3.79	9.50	4.33
Visual–visual PAL (max. 25)	13.33	4.83	13.39	4.27
Verbal–visual PAL (max. 25)	14.06	4.95	14.89	3.66

Analysis of errors

The errors made in the two verbal output conditions were scored and categorized by the type of error made. Given the lack of group differences in the two visual output conditions in the overall analyses, further investigation of those errors was unnecessary. We first removed "don't know" responses from the total errors so that all proportions reflected errors in which a child attempted a response. Errors were categorized into three main categories: association errors (i.e., correctly pronounced nonword, incorrect pairing), phonological errors (i.e., incorrectly pronounced nonword), and intrusion errors (i.e., responding with an item from a previously learned condition). Because phonological errors represented a wide range of phonological distortions, these errors were further investigated by separating errors according to the extent to which they deviated from the target. Responses were classified as single feature errors if they deviated by only one phoneme (e.g., "vep" for "vek"), and multiple feature errors if they deviated by two or more phonemes (e.g., "zep" for "vek"). The majority of errors (88%) respected syllable structure (CVC) and errors occurred at all positions within that structure. A final question of interest was whether children with dyslexia repeated errors more frequently than age-matched controls. These "perseveration errors" were calculated as the proportion of pronunciation errors that were repeated across multiple trials. The error data for each group (by condition) is presented in Table 5. For all tests reported below, differences in the patterns of proportions between groups were tested in log-linear mixed effects models in the lme4 package in R. In each model, the proportion of errors was predicted by a random effect of subject, and fixed effects for error type, group, and the interaction between the two. An observation-level random effect was also included to account for overdispersion (indicated by higher observed variance than would be expected from the theoretical binomial distribution) (e.g., Maindonald & Braun, 2010; Rigby, Stasinopoulos, & Akantziliotou, 2008).

Visual–verbal PAL

"Don't know" responses accounted for 8% of errors made by children with dyslexia, and 7% made by the controls. After removing these, it is clear that the pattern of errors is similar for both groups, with phonological errors comprising the majority of errors, followed by association errors, and finally, intrusion errors, which were relatively rare. Results of a log-linear mixed effects model revealed a main effect of error type (OR = 2.36, 95% CI [1.56 3.58], $Z = 4.05$, $p < .001$), but no main effect of group (OR = 0.91, 95% CI [0.45 1.84], $Z = -0.27$, $p = .788$), and no interaction between group and error type (OR = 1.36, 95% CI [0.59 3.13], $Z = 0.73$, $p = .466$). These results indicate that

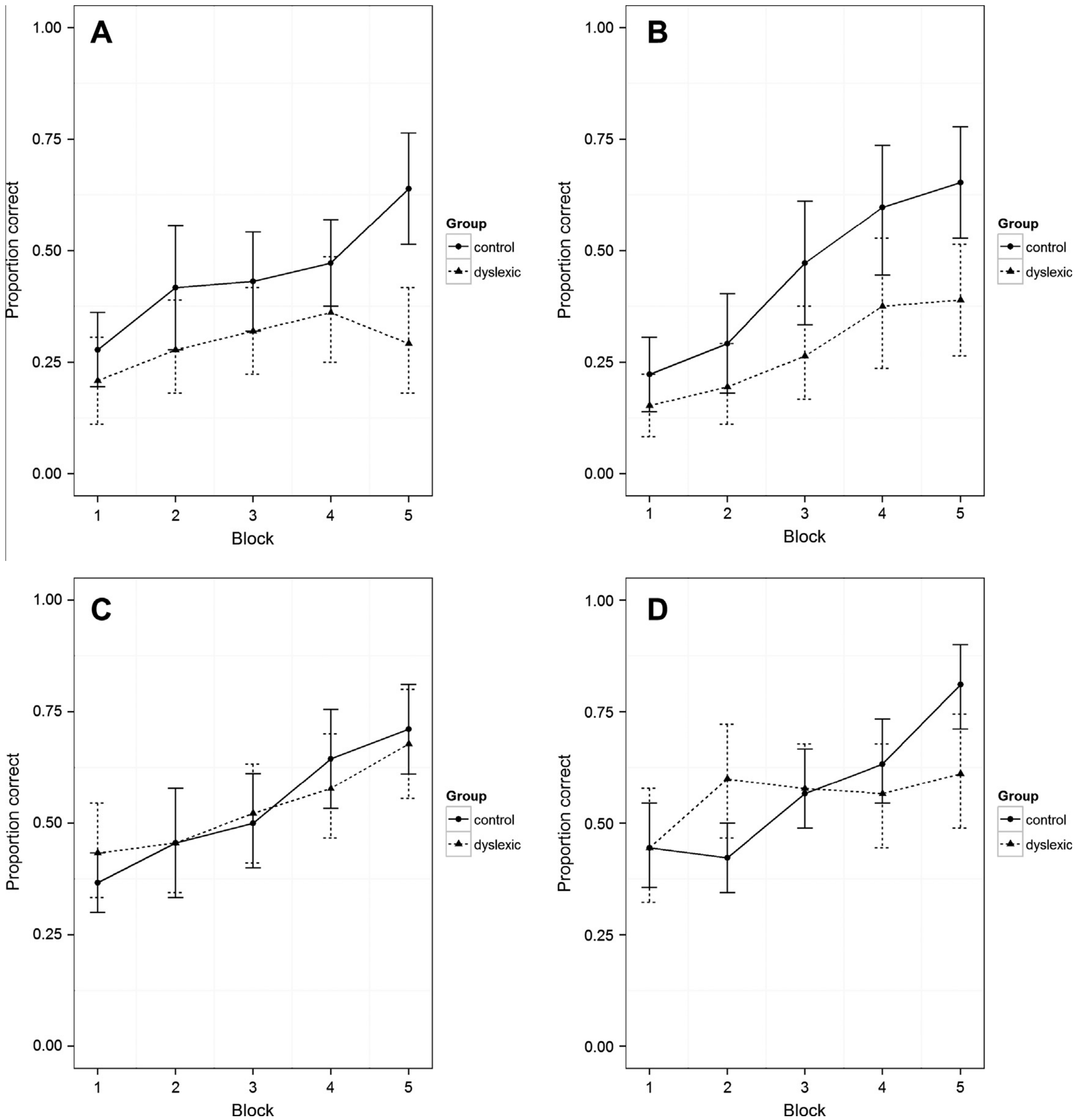

Fig. 2. Proportion correct across learning blocks for the dyslexic and control group in Experiment 2: (A) visual–verbal PAL, (B) verbal–verbal PAL, (C) visual–visual PAL, or (D) verbal–visual PAL. Error bars represent 95% confidence intervals.

although the dyslexic group made more errors than the control group overall, the pattern and relative proportions of errors were similar across groups.

Although the overall pattern of proportions did not differ, we were interested in the extent to which phonological errors deviated from the target word. Errors that share more phonemic features with the target word may be viewed as less severe phonological distortions than those sharing fewer features in common. To examine this possibility we tested for group differences in single vs. multiple feature phonological errors. Because single and multiple feature errors are mutually dependent (i.e., knowing the proportion of one category defines the proportion of the other category), group differences were only tested for multiple feature errors. Note that had we tested differences in singles feature errors the resulting models would be identical. Although the dyslexic group appeared to commit a higher proportion of multiple feature errors than controls, this difference was not statistically significant (OR = 2.00, 95% CI [0.72 5.56], $Z = 1.34$, $p = .181$). Finally, there was no indication that the groups differed in how often they repeated the same errors (i.e., perseveration errors) (OR = 0.97, 95% CI [0.36 2.61], $Z = -0.06$, $p = .953$).

Verbal–verbal PAL

"Don't know" responses constituted 9% and 12% of errors made by the dyslexic and control groups respectively. After removing these, the pattern of errors closely

Table 5
Proportion errors by type and group in visual–verbal PAL and verbal–verbal PAL in Experiment 2.

	Dyslexic		Control	
	M	*SD*	*M*	*SD*
Visual–verbal PAL				
Intrusion	0.02	0.01	0.06	0.03
Association	0.32	0.05	0.34	0.05
Phonological	0.66	0.06	0.60	0.05
Single feature	0.57	0.08	0.68	0.07
Multiple feature	0.43	0.08	0.32	0.07
Perseverations	0.48	0.08	0.45	0.08
Verbal–verbal PAL				
Intrusion	0.05	0.02	0.08	0.04
Association	0.21	0.05	0.32	0.05
Phonological	0.74	0.05	0.60	0.07
Single feature	0.33	0.06	0.54	0.07
Multiple feature	0.67	0.06	0.46	0.07
Perseverations	0.29	0.06	0.30	0.07

resembled that of visual–verbal PAL, with the majority of errors being phonological in nature. Results of a log-linear mixed effects model revealed a main effect of error type (OR = 7.05, 95% CI [4.83 10.28], $Z = 10.13$, $p < .001$), but no main effect of group (OR = 0.78, 95% CI [0.43 1.40], $Z = -0.84$, $p = .404$). A significant interaction between group and error type (OR = 2.99, 95% CI [1.40 6.37], $Z = 5.77$, $p < .001$) revealed that whereas the proportions of errors did not differ between groups for intrusion errors (OR = 0.53, 95% CI [0.16 1.78], $Z = -1.03$, $p = .303$), they differed significantly for association errors (OR = 0.33, 95% CI [0.13 0.81], $Z = -2.41$, $p = .016$), and to an even greater extent for phonological errors (OR = 3.50, 95% CI [1.43 8.53], $Z = 2.75$, $p = .006$).

These results were followed up with a closer examination of the nature of the phonological errors, as described for visual–verbal PAL. A log-linear mixed effects model revealed that the dyslexic group committed a higher proportion of multiple feature errors than the control group (OR = 2.97, 95% CI [1.39 6.32], $Z = 2.82$, $p = .005$). As in visual–verbal PAL, however, there was no difference in the proportion of perseveration errors made by each group (OR = 0.78, 95% CI [0.25 2.47], $Z = -0.42$, $p = .677$).

Discussion

The results of Experiment 2 align closely with those of Experiment 1, replicating the finding of deficits specific to verbal output conditions (i.e., visual–verbal, verbal–verbal PAL) in children with dyslexia. There was no evidence for global crossmodal associative learning deficits (i.e., both visual–verbal and verbal–visual PAL), nor were the observed PAL deficits restricted to crossmodal conditions. Importantly, these results held after adjusting task difficulty across conditions, which resulted in closely equated performance levels that were free from floor or ceiling effects. Together, the results of Experiments 1 and 2 clearly implicate verbal deficits in driving poor PAL performance in dyslexia. Further support for this conclusion comes from the error analyses. Despite making more errors in visual–verbal and verbal–verbal PAL overall, children with dyslexia did not commit a higher proportion of associative errors, suggesting that poor PAL performance does not result from difficulties with the associative demands of the task. Instead, the errors were indicative of phonological learning deficits. Children with dyslexia made a similar proportion of phonological errors as controls in visual–verbal PAL, and a greater proportion of phonological errors in verbal–verbal PAL. The higher proportion of errors in verbal–verbal PAL may have resulted from the increased phonological demands of the task (both verbal input and output demands) in comparison to visual–verbal PAL (only verbal output demands). However, in both conditions, children with dyslexia tended to produce errors that were phonologically further from the target than controls. Together, these findings clearly indicate that PAL deficits in dyslexia are specific to those conditions that require verbal output.

Experiment 3

The findings of Experiment 1 and Experiment 2 provide strong evidence that PAL deficits in dyslexia stem from the verbal demands of the task, rather than difficulties in crossmodal associative learning. However, despite demonstrating the specificity of deficits to verbal output conditions, they cannot speak to the locus of these difficulties. For example, do deficits arise during the verbal learning or associative stage of PAL? Evidence supports the notion that the phonological and associative components of PAL tasks are indeed dissociable (Duyck, Szmalec, Kemps, & Vandierendonck, 2003; Freedman & Martin, 2001). Importantly, difficulties at either (or both) stages of learning would lead to the verbal-output PAL deficits observed in Experiments 1 and 2. Traditional nonword PAL paradigms are confounded by the fact that verbal learning and associative learning occur simultaneously, making it difficult to estimate the effects of verbal learning and associative learning on performance. Yet, locating the source of failure is crucial for guiding our conceptualization of PAL deficits in dyslexia. If deficits arise solely from demands on verbal learning, PAL deficits should be viewed as a corollary of verbal deficits, rather than a defining feature of dyslexia. If, however deficits emerge in the associative learning phase, a different explanation might be necessary, giving special status to associative learning deficits in dyslexia, albeit deficits specific to associations requiring verbal output.

Experiment 3 was designed to address these confounds by separating the learning procedure into a verbal learning and associative learning phase. This was achieved by presenting the children with verbal stimuli in a pre-exposure phase, before requiring them to associate the verbal forms with input stimuli. The primary question of interest was whether verbal learning ability, as indexed by the pre-exposure phase, would fully account for any observed difference between dyslexic and control groups in a test of visual–verbal PAL. We hypothesized that differences in verbal learning in the pre-exposure phase would fully explain deficits in visual–verbal PAL in the associative learning phase. That is, we expected no difference between the dyslexic and control groups in visual–verbal PAL, *after* accounting for verbal learning ability. If, however, children with dyslexia have an additional difficulty binding verbal information to a referent in memory, we would expect PAL deficits to be additive, such that they are more severe than would be predicted solely from underlying language deficits.

Participants

Dyslexic children were selected from a specialist state school in the UK for children with specific learning difficulties. This school provides provision to children with long-standing reading difficulties that have proved resilient to intervention provided by their local school. All children undergo a comprehensive educational assessment before entry into the school. Together with teacher reports, these assessments were used to select a group of children with dyslexia, with whom informed consent forms were sent home. Control children were selected from two state schools in London, UK serving middle and upper-class catchment areas. Informed consent forms were sent home with all children in Grades 4 and 5 (ages 9–11).

Upon obtaining consent, the children were assessed with a cognitive battery consisting of the following literacy measures: Word Reading Fluency (TOWRE), Nonword Reading Fluency (TOWRE), Phoneme Deletion (CTOPP), Digit Span (CTOPP), Rapid Automatized Naming (CTOPP), and Single Word Reading from the Wechsler Objective Reading Dimensions test (WORD) (Wechsler, 1993). These measures came from a battery administered as part of a larger study. Due to the constraints of the larger study, it was unfortunately not possible to administer a test of expressive vocabulary.

Given the extensive history of the children with dyslexia, the test battery was utilized as a confirmatory measure; that is, children remained in the sample unless they scored above the 25th percentile on three or more of the reading and reading-related measures. This criterion resulted in a final sample of 14 children with dyslexia (8 boys, 6 girls). A control group of 14 typically developing readers (6 boys, 8 girls), matched to the dyslexic group on age and nonverbal reasoning was next selected. All children in this group had no documented history of special educational needs, and scored above the 25th percentile on all literacy measures. All participants spoke English as a first language.

The descriptive characteristics of the groups are displayed in Table 6. As expected, the children with dyslexia performed far below the level of controls across literacy measures, despite being matched with the controls on age and nonverbal reasoning.

Table 6
Descriptive characteristics of dyslexic and control readers in Experiment 3.

Measures	Dyslexic		Control	
	M	*SD*	*M*	*SD*
Age (years)	10.61	0.62	10.30	0.30
WASI Matrices[a]	52.50	5.08	56.21	7.61
Sight Word Efficiency[b]	86.71	7.53	115.36	8.38
Phonemic Decoding Efficiency[b]	82.64	8.13	117.93	15.01
Single Word Reading[b]	81.50	7.02	113.36	9.16
Phoneme Deletion[c]	8.00	2.60	11.64	2.90
RAN Digits[c]	8.21	2.22	11.64	3.25
Digit Span[c]	6.89	0.77	12.21	0.85

[a] *T* score, $M = 50$, $SD = 10$.
[b] Standard score, $M = 100$, $SD = 15$.
[c] Scaled score, $M = 10$, $SD = 3$.

Materials and procedure

Children were tested in two sessions on consecutive days. On day one children completed the verbal pre-exposure stage in which they were familiarized with and asked to learn the verbal response items. On day two children learned the visual–verbal paired associates. The verbal stimuli used for the paired associates were the same as those used in the verbal pre-exposure stage. The experiment was presented on a Dell laptop using EPrime Version 2.0.

Verbal pre-exposure

The verbal stimuli were five CVC nonwords chosen at random from the stimuli used in Experiment 1. The children watched a short introductory animation in which aliens crashed to earth and needed help returning to their planet. The children were told that they needed to learn some words from the "alien" language. The experiment began with a block of immediate repetition trials in which each of the five nonwords was presented in random order. Children were asked to repeat each nonword aloud immediately after hearing it. This initial block served as a check for the children's ability to hear and orally reproduce the nonwords. If a child mispronounced a word, the experimenter responded by saying, "That's not quite right. The word is 'X'". This was followed by a serial recall game with three blocks of increasing difficulty. This game served to familiarize the children with the nonwords. In the first block, two nonwords were presented sequentially, and the children asked to repeat them back in the same order. In the next block, children heard the remaining three nonwords and were asked to repeat them back in the same order in which they were presented. In the final block, all five nonwords were presented sequentially, and the children asked to repeat them in the same order. The order of the nonwords was fixed across the three serial recall blocks so that there were two exposures to each item. As the purpose of these recall games was to familiarize the children with the items, no corrective feedback was given. Following these initial familiarization trials, children completed alternating blocks of four free recall test trials and three passive presentation trials. In the first free recall block, children were asked to recall the words they had learned in the preceding presentation trials. Participants were not prompted or corrected by the experimenter and all responses were recorded in writing. Immediately following this block, children were given a passive presentation block in which they were asked to listen to the five nonwords. They were not instructed to repeat the items aloud in these blocks. Immediately following the presentation block, the children completed the next free recall block.

This procedure continued until all blocks were completed. In total the verbal pre-exposure stage consisted of 10 exposures to each of the nonwords and four blocks of free recall test trials (for a maximum free recall score of 20).

Visual–verbal PAL

The start of the second day of testing began with one free recall block in which participants were asked to recall the five nonwords from Day 1. Following this, children began the visual–verbal PAL learning stage. This commenced with two presentation blocks in which five abstract visual forms (from Experiment 1) were paired with the five nonwords. Following the presentation blocks, the children engaged in five blocks of test trials in which each of the five symbols was presented, and the child asked to produce the corresponding nonword. Regardless of the accuracy of the response, the correct answer was presented immediately following each trial. This procedure was repeated for each trial across all five blocks. All participants completed all trials, and accuracy per trial and total learning across trials was recorded. The final score was calculated as the total number of correct trials across all five blocks (maximum score of 25). The participant's response was recorded (in writing) following the presentation of each item for use in subsequent error analyses. Following the test trials, a recognition block was administered in which the participants heard each of the five nonwords and were asked to point to the correct picture on the screen. The position of the symbols on the screen was randomized for each trial.

Results

Mean performance by group and day is displayed in Fig. 3. Fixed effects for all models are shown in Appendix A.

Verbal learning

To examine group differences in verbal learning, we ran a linear mixed effects model with subject and item as random effects, and group (dyslexic, control), and block (1–5) as fixed effects. The results revealed that children with dyslexia performed significantly below the control group in verbal learning. There was a main effect of group (OR = 0.30, 95% CI [0.15 0.63], $Z = -3.21$, $p = .001$), and block (OR = 1.50, 95% CI [1.26 1.80], $Z = 4.47$, $p < .001$), but no interaction between group and block (OR = 0.98, 95% CI [0.68 1.40], $Z = -0.12$, $p = .960$). Despite equivalent exposure to the nonwords, children with dyslexia recalled significantly fewer nonwords than typically developing readers.

Visual–verbal PAL

Consistent with the results of Experiments 1 and 2, the results of a logistic linear mixed effects model with subject and item as random effects and group and block fixed effects revealed that the dyslexic group performed significantly below the level of the control group in visual–verbal PAL. There was a main effect of group (OR = 0.41, 95% CI [0.19 0.88], $Z = -2.29$, $p = .022$) and block (OR = 1.30, 95% CI [1.13 1.49], $Z = 3.74$, $p < .001$), but no interaction between group and block (OR = 1.01, 95% CI [0.77 1.31], $Z = 0.05$, $p = .960$). These results are perhaps unsurprising given the group differences that emerged in verbal learning, and the robust literature reporting visual–verbal PAL deficits in dyslexia. Our primary question of interest, however, was not whether a group difference would emerge in visual–verbal PAL, but whether this difference would be greater than expected given the difference in verbal learning. As a preliminary answer to this question, it is interesting to note that the effect of group (dyslexic, control) is similar for verbal learning ($Z = -3.21$) and visual–verbal PAL ($Z = -2.29$), suggesting that visual–verbal PAL deficits are directly proportional to verbal learning deficits.

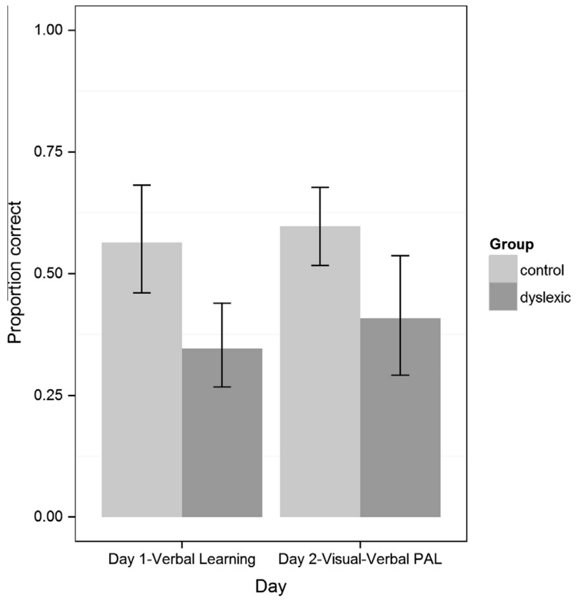

Fig. 3. Proportion correct for the dyslexic and control group in verbal learning and visual–verbal learning in Experiment 3. Error bars represent 95% confidence intervals.

To directly test whether visual–verbal PAL performance is fully accounted for by previous verbal learning performance, we ran a linear mixed effects model with a fully specified random effects structure for subjects and items, and fixed effects for group (dyslexic, control) and verbal learning. We used total verbal learning score (by-item and by-subject) to predict total visual–verbal PAL score (by-item and by-subject). Crucially, this model evaluates the item-specific relationship between verbal learning and visual–verbal PAL, asking whether the extent to which a child learns a nonword predicts the success with which that same nonword is associated with a symbol in visual–verbal PAL. Additionally, the model determines whether this relationship differs between children with dyslexia and typically developing readers. As recommended by Baayen et al. (2008), p-values are reported here based on the normal-approximation of the t-statistic because the significance of model coefficients is not available in lme4 for models with random slopes. Such an approach has been shown to be more conservative than traditional by-subjects and by-items ANOVA (Barr et al., 2012).

The results of the model supported an item specific relationship between verbal learning and visual–verbal PAL (OR = 1.53, 95% CI [1.24 1.89], $t = 3.93$, $p < .001$). The extent to which children learned the visual–verbal paired associates was predicted by their success in learning the nonwords in the pre-exposure phase on Day 1. Not only was there a strong predictive relationship between verbal learning and visual–verbal PAL, but this relationship also held regardless of group membership: group did not emerge as a significant predictor of visual–verbal PAL in the model (OR = 0.58, 95% CI [0.28 1.22], $t = -1.43$, p=.153). Model comparison showed that a model with verbal learning as the sole predictor of visual–verbal PAL performed equally as well as a model in which group (dyslexic, control) was added as a predictor ($\Delta\chi^2(1) = 1.756$, $p = .185$). In contrast, the addition of verbal learning to a model with group as the sole predictor of visual–verbal PAL significantly improved model fit ($\Delta\chi^2(1) = 10.45$, $p = .001$). Together, these results implicate the vital role of verbal learning in determining visual–verbal PAL performance.

Crucially, although the dyslexic group performed significantly below the control group in both verbal learning and visual–verbal PAL, there was no evidence to support a view of an additive PAL deficit in dyslexia. That is, if visual–verbal PAL deficits reflect specific difficulties with learning to associate two stimuli together, we would expect to find an interaction between group and verbal learning (such that children with dyslexia experience greater difficulty with visual–verbal PAL than expected from their verbal learning scores). However, the interaction between verbal learning and group (OR = 1.13, 95% CI [0.74 1.73], $t = 0.58$; $p = .562$) was not significant.

Finally, despite the significant differences in visual–verbal PAL reported above, the dyslexic and control groups demonstrated comparable levels of performance on the recognition task: dyslexic ($M = 2.79$, $SD = 0.42$), control ($M = 3.29$, $SD = 0.37$), $t(26) = 0.89$, $p = .381$.

Analysis of errors

We analyzed the errors made in each phase of the experiment, verbal leaning and PAL. Summary data are shown in Table 7.

Table 7
Proportion errors by type and group in verbal learning and visual–verbal PAL in Experiment 3.

	Dyslexic		Control	
	M	*SD*	*M*	*SD*
Verbal learning				
Refusals	0.63	0.04	0.53	0.06
Phonological errors	0.37	0.04	0.47	0.06
Single feature	0.79	0.06	0.93	0.03
Multiple feature	0.21	0.06	0.07	0.03
Perseverations	0.49	0.09	0.38	0.08
Visual–verbal PAL				
Association	0.45	0.05	0.80	0.05
Phonological errors	0.55	0.06	0.20	0.05
Single feature	0.59	0.09	0.67	0.14
Multiple feature	0.41	0.09	0.33	0.14
Perseverations	0.42	0.09	0.44	0.14

Verbal learning errors comprised 63% refusals and 37% phonological errors in the dyslexic group, and 53% refusals and 47% of errors in the control group. Because excluding refusal errors from the analysis would leave only one remaining category of errors to be analyzed (because there were only two overall error categories for verbal learning errors), only the subtypes of phonological errors were analyzed. Both groups made more single feature than multiple feature errors, with the dyslexic group committing a higher proportion of multiple feature errors than the control group. However, the results of a log-linear mixed effects model showed that the difference in proportions between groups was not significant (OR = 2.38, 95% CI [0.68 8.33], $Z = 1.36$, $p = .174$). Consistent with the error analysis in Experiment 2, there was no evidence that the dyslexic group committed a higher proportion of perseveration errors than the control group (OR = 1.17, 95% CI [0.41 3.36], $Z = .28$, $p = .777$).

Turning to the PAL phase of the experiment, refusals comprised 12% of dyslexic group errors and 8% of control group errors respectively. After removing these errors, two primary error types remained: association errors and phonological errors. An initial log-linear mixed effects model showed that the dyslexic group committed a higher proportion of phonological errors than the control group (OR = 0.21, 95% CI [0.10 0.43], $Z = -4.21$, $p < .001$). Further investigation into the nature of the phonological errors did not reveal a significant difference between the groups in the proportion of single or multiple feature errors (OR = 1.50, 95% CI [0.21 10.57], $Z = 0.40$, $p = .686$). Thus it seems that although children with dyslexia made a higher proportion of phonological errors than the control group, when these errors did occur, the likelihood of these errors being near or far from the target word did not differ between groups. Consistent with previous results, there were no differences between groups in the proportion of perseverations made (OR = 0.80, 95% CI [0.19 3.35], $Z = -0.30$, $p = .763$). Finally, there was no evidence for group differences in the proportion of errors continued from the verbal pre-exposure stage (OR = 0.41, 95% CI [0.12 1.40], $Z = -1.42$, $p = .154$).

Discussion

The results of the Experiment 3 support the view that deficits in visual–verbal PAL are a consequence of underlying verbal deficits. When assessed in separate phases, children with dyslexia demonstrated impairments in both verbal learning and visual–verbal paired associate learning. Crucially, however, children with dyslexia did not demonstrate impairments over and above those expected from their verbal learning scores. That is, performance differences in visual–verbal PAL were fully explained by initial rates of verbal learning. This strongly suggests that visual–verbal PAL deficits are a consequence of verbal learning difficulties, and are not specific to the associative demands of the task. Such a finding is in accordance with the results of Experiments 1 and 2, in which children with dyslexia demonstrated impairments only in those PAL tasks that had a verbal output component.

The error analyses further support these conclusions. On Day 1, both groups showed a similar pattern and extent of phonological errors. This indicates that although children with dyslexia were less efficient than controls (i.e., learned fewer nonwords in the same number of trials), both groups were engaging in the same general process of verbal learning. However, the error patterns diverged quite strikingly on Day 2, with phonological errors comprising the majority of errors in the dyslexic group, and associative errors comprising the majority of errors in the control group. This likely reflects a continued difficulty in phonological form learning that extended into visual–verbal PAL for the dyslexic group, but not the control group. That the control group made errors that were almost entirely associative in nature (.80) on Day 2 suggests that they were utilizing accurate phonological representations of the novel forms. In contrast, the decidedly greater proportion of phonological errors in the dyslexic group on Day 2 is indicative of a continued difficulty with the acquisition of the novel phonological forms. Note, however, that the more balanced proportion of phonological (.55) to associative (.45) errors in the dyslexic group suggests that both phonological and associative learning occurred in the visual–verbal PAL task. Indeed, despite difficulty acquiring the phonological forms, children with dyslexia demonstrated successful learning of the associations on a recognition task. Together, these results provide further support for a view of PAL deficits as specific to the learning of phonological forms.

General discussion

Three experiments assessed the nature and specificity of PAL deficits in dyslexia. Our goals were twofold: first, to determine whether deficits are specific to verbal or crossmodal mapping demands, and second, to investigate the stage of learning in which deficits emerge. Experiments 1 and 2 evaluated performance across four conditions, each uniquely characterized by mapping modality (crossmodal, unimodal) and output demand (visual, verbal). Consistent with the literature, we observed robust visual–verbal PAL deficits in the absence of visual–visual PAL deficits, confirming the lack of general associative learning deficits in dyslexia (Messbauer & de Jong, 2003; Vellutino et al., 1975). Crucially, children with dyslexia exhibited deficits only in visual–verbal and verbal–verbal PAL, with no evidence for impairments in visual–visual or verbal–visual PAL. This finding is at odds with a strong theory of impaired crossmodal associative learning, in which deficits would be expected in both visual–verbal and verbal–visual PAL. Although a direction-dependent theory might provide an explanation for the lack of verbal–visual PAL deficits (i.e., impairments are specific only to visual–verbal PAL because of its shared mapping direction with reading) this account is simply not in accordance with our data. The PAL deficits reported in Experiments 1 and 2 were not unique to visual–verbal PAL. Instead, deficits emerged in both visual–verbal and verbal–verbal PAL, tasks with a verbal output demand. This pattern of findings provides unequivocal support for a verbal account of PAL deficits in dyslexia, and mirrors the PAL–reading relationship seen in typical development (Litt et al., 2013).

Further support for this conclusion is provided by the analysis of errors in Experiment 2, and indeed, in Experiment 3. Although children with dyslexia made more errors overall in both visual–verbal and verbal–verbal PAL than control children, there was no evidence that they committed a higher proportion of associative errors, as would be expected if poor PAL performance results from difficulties with the associative demands of the task. In contrast, children with dyslexia tended to make a higher proportion of phonological errors than controls, a finding in line with Mayringer and Wimmer (2000). There was also some indication that the phonological errors were more severe in the dyslexic group, as indexed by a higher proportion of multiple feature errors. These differences were especially marked in verbal–verbal PAL, arguably because the twin demands of verbal input and verbal output place a greater demand on the phonological system than verbal output alone, as in the visual–verbal condition.

Together, the results of Experiments 1 and 2 provide support for the phonological nature of PAL deficits; however, they cannot speak directly to the stage of learning at which deficits arise. PAL is a complex task and potentially, the verbal deficits observed in Experiments 1 and 2 might arise at one of two phases, learning the phonological form (verbal learning), or associating a visual symbol with the phonological form (associative learning). Additionally, a difficulty at both phases of learning is possible. By separating these two phases within a visual–verbal paired associate learning paradigm, Experiment 3 investigated the contribution of phonological form learning and associative learning to PAL performance in children with dyslexia.

Once again, our results were clear. Children with dyslexia learned fewer phonological forms than control children in the verbal learning phase; they were also poorer at mapping visual symbols to these phonological forms, performing less well than controls in the subsequent visual–verbal PAL task. Of course, poor visual–verbal PAL performance may be an expected consequence of verbal learning deficits. Success on any given visual–verbal PAL trial requires children to output the correct phonological form; thus performance hinges critically on the integrity of the new phonological representation. Evidence for a specific difficulty with the associative component of the task would therefore be demonstrated by a finding of visual–verbal PAL deficits that are greater than expected given the initial verbal learning deficits. Against this prediction, visual–verbal PAL deficits were directly proportional to the extent to which children with dyslexia learned the nonwords in the verbal learning phase; there was no evidence that associative learning placed an additional constraint on PAL performance. In fact, the dyslexic and control groups were equally able to recognize the paired associates, indicating that successful associative learning occurred.

Taken together, the results of our experiments demonstrate that PAL deficits in dyslexia reflect difficulties with the phonological component of the task. However, two important questions remain: what is indexed by this phonological component, and how does it relate to reading ability? In addressing the first question, it is perhaps useful to distinguish between the initial encoding of a new phonological form vs. its subsequent maintenance. Numerous researchers have proposed that limitations in the phonological loop (the component of verbal working memory responsible for immediate and short term storage of phonological information) impose constraints on children's ability to learn new words in PAL tasks (e.g., Gathercole & Baddeley, 1990; Gathercole, Hitch, Service, & Martin, 1997). This explanation, however, does not sit comfortably with our data as the dyslexic children were as accurate as controls when asked to immediately repeat the new phonological forms (80% vs. 84% for the dyslexic and control groups, respectively[1]). This suggests that group differences in PAL are unlikely to be caused by failure at encoding (for similar findings, see Mayringer & Wimmer, 2000).

In contrast, our data sit more comfortably with the idea that children with dyslexia struggle to maintain the integrity of novel phonological representations over a short period of time, and that this might underpin their subsequent impairments in paired associate learning. It is quite striking that although children with dyslexia showed accurate immediate repetition, the integrity of their representations were not maintained over a relatively short period of time, as evidenced by the phonological distortions produced on test trials. This degradation occurred over the course of only four or five interceding trials despite the presentation of the correct nonword following each test trial, which presumably allowed for updating and adjustment of the phonological representation throughout the task. This is consistent with the idea that children with dyslexia have difficulty establishing stable phonological representations. The error analyses in Experiments 2 and 3 also lend support to this conclusion: although children with dyslexia made more phonological errors than controls, they did not make a higher proportion of perseverative errors (although see Mayringer & Wimmer, 2000). That the majority of the phonological errors were new, rather than repeated, strongly suggests that poor performance was not simply a case of having learned incorrect phonological forms. Instead, our results indicate that children with dyslexia experience difficulty with phonological form learning, a conclusion that echoes previous work on PAL (Litt et al., 2013; Mayringer & Wimmer, 2000; Wimmer et al., 1998).

Before continuing, it is important to note that although our results clearly implicate "verbal-output" in driving PAL deficits in dyslexia, our data do not allow us to determine if the difficulties experienced by children with dyslexia are specific to learning phonological representations for output, or the acquisition of novel phonological representations more generally (regardless of input or output). The consistently unimpaired performance of children with dyslexia in verbal–visual PAL, in concert with the finding of preserved performance in a receptive, but not expressive version of visual–verbal PAL in Experiment 3, certainly suggest that phonological-output processes are crucial to explaining PAL deficits. This is a tempting conclusion, especially given the implication of phonological-output deficits playing a causal role in reading disability (e.g., Hulme & Snowling, 1992; Nation, Marshall, & Snowling, 2001; Ramus & Szenkovits, 2008; Snowling & Hulme, 1994; Swan & Goswami, 1997; Truman & Hennessey, 2006). However, it is also possible that verbal–visual PAL lacked the sensitivity to detect individual differences in the establishment of a novel phonological representation because verbal-input PAL tasks only require the recognition of phonological forms. Whereas children could presumably perform well on a verbal–visual PAL task without having formed precise phonological representations of the novel words (e.g., by relying on partial cues), this is not the case on a verbal-output PAL task, as successful performance depends on explicit recall and output of a fully-specified phonological representation. An experiment in which the demands of a verbal–visual PAL task were increased to require fully specified phonological representations (e.g., using nonwords differing by only one phoneme) would allow for a more direct comparison of the importance of input and output phonology in explaining the PAL–reading relationship. Regardless of whether the verbal learning processes are specific to output representations, our data are clear in suggesting that the ability to acquire novel phonological representations represents the crucial source of difficulty in explaining PAL deficits in dyslexia.

We now come to the question of how to orient PAL deficits within the context of reading development and dyslexia. Although our data do not allow us to speak directly to this issue, we shall consider two potential explanations of the PAL–reading relationship. The first account views PAL as a correlate of reading and reading-related skills, while the second grounds PAL in a theory in which phonological form learning plays a direct role in the reading process and its development.

On the first view, PAL and reading may share a correlational relationship due to the influence of a third underlying factor such as phonological processing. For example, the phonemic sensitivity that is crucial to reading development is likely also crucial to phonological form learning in PAL. In support of this view, concurrent studies have indicated that individual differences in visual–verbal PAL performance can be largely accounted for by phonological processing skill, a finding that holds for both dyslexic and typically developing samples (de Jong et al., 2000; Messbauer & de Jong, 2003, 2006). Additionally, although longitudinal and training studies with PAL are sparse, the few studies that have been reported provide support for this conclusion. For example, Lervåg, Bråten, and Hulme (2009) found that visual–verbal PAL was not a longitudinal predictor of reading ability when measured in pre-readers, suggesting that the PAL–reading relationship emerges only once children are literate. Additionally, the findings by de Jong et al. (2000) implicate a direct role for phonological skills in the development of visual–verbal PAL ability: training in phoneme awareness improved visual–verbal PAL performance in kindergartners. If PAL performance relies heavily on the underlying integrity of the phonological system, PAL deficits in dyslexia may be best viewed as a consequence, rather than a cause of the disorder.

On this view, the failure to construct or maintain reliable phonological representations of the nonwords in our PAL tasks may have resulted from a lack of linguistic

[1] Due to computer coding errors on some of the presentation trials, these figures reflect data only from trials in which both computer data and hardcopy written records were available to ensure validity. However, the general pattern of findings held when examined in the full data set.

support from long-term memory, without which, novel representations become highly susceptible to the effects of interference or decay (Hulme, Maughan, & Brown, 1991; Hulme, Roodenrys, Brown, & Mercer, 1995). For example, evidence suggests that as short term memory traces decay, recall success is significantly determined by the availability of representations in long-term memory to aid in the process of redintegration, or reconstruction of these traces (Gupta & MacWhinney, 1997; Hulme et al., 1991; Thomson, Richardson, & Goswami, 2005). This support is not limited to memory for words; factors such as phonotactic probability, word-likeness, and lexical neighborhood size have all been shown to affect nonword recall (Storkel, Armbruster, & Hogan, 2006; Thorn & Frankish, 2005). Children with dyslexia are generally thought to have phonological representations that are underspecified or inaccurate (e.g., Elbro, 1998; Elbro & Jensen, 2005; Fowler, 1991; Swan & Goswami, 1997; cf. Ramus & Szenkovits, 2008), which may limit the quality and availability of support from long-term memory (Snowling & Hulme, 1994). Although the current experiment utilized nonword stimuli exclusively, this view is also compatible with the findings in the literature of PAL deficits for words. Arguably, words held in short term memory would be subject to a similar process of decay as outlined above, thus making it difficult for children with dyslexia to hold and maintain the words in memory for use in a PAL task. Of course, we would expect words that are high in frequency or semantic content to be less susceptible to decay than words that are low in frequency, abstract, or phonologically complex, because they enjoy greater support from long-term memory (Hulme et al., 1997). Indeed, this prediction matches the pattern of findings reported for PAL tasks with words (Elbro & Jensen, 2005; Messbauer & de Jong, 2006; Vellutino et al., 1995).

Although this conceptualization of PAL deficits sits comfortably with the phonological deficit theory of dyslexia, we caution against a view of PAL deficits as equivalent to phonological awareness deficits in dyslexia. Numerous researchers have found that visual–verbal PAL explains unique variance in reading ability, beyond phonological awareness (Hulme et al., 2007; Litt et al., 2013; Warmington & Hulme, 2012; Windfuhr & Snowling, 2001). Additionally, Wimmer et al. (1998) reported visual–verbal PAL deficits in children with dyslexia even though they were matched with controls for phonological awareness. It is, however, possible to reconcile these findings by considering that phonological processing is not a unitary construct; phonological awareness comprises only one component of the phonological system (Wagner & Torgesen, 1987; Wagner, Torgesen, & Rashotte, 1994). Thus the phonological component of verbal-output PAL may index different abilities than those typically tapped by tests of phonological awareness. For example, the learning and establishment of phonological representations in memory likely relies, at least in part, on processes that differ from those involved in the explicit access and manipulation of established phonological representations.

Having outlined the possibility that the PAL–reading relationship emerges from a shared dependence of both skills on phonological processes, we will now consider a second, more proximal relationship between PAL and reading development. Recent theories suggest that phonological form learning plays a specific role in reading acquisition via the formation of novel spelling-linked phonological representations (Elbro, de Jong, Houter, & Nielsen, 2012; Ranbom & Connine, 2011; Tunmer & Chapman, 2012). For example, when decoding an irregular word such as *island* for the first time, children inevitably produce the phonological form "izland." Evidence suggests that in skilled readers, this "spelling pronunciation" facilitates activation of the correct spoken representation in the lexicon, and is subsequently stored and activated alongside the spoken representation in long-term memory (Elbro et al., 2012; Ranbom & Connine, 2011; Venezky, 1999). The ability to utilize variable phonological representations to arrive at the correct spoken pronunciation has been shown to explain unique variance in word recognition skills (Elbro et al., 2012; Tunmer & Chapman, 2012; Vousden & Ellefson, 2012). Thus learning to read may depend on the establishment of novel, orthographically-derived phonological representations, as well as the binding of these spelling pronunciations to spoken pronunciations in memory. Consequently, a deficit in phonological form learning would certainly impede reading acquisition in children with dyslexia. Future research of a longitudinal nature is required to distinguish between this view and the correlational account of the PAL–reading relationship outlined above.

To summarize, the current paper evaluated the contribution of verbal and associative demands in giving rise to PAL deficits in dyslexia. Contrary to the predictions of the crossmodal hypothesis, children with dyslexia exhibited deficits specific to PAL tasks with a verbal output demand, regardless of whether the mappings were crossmodal or unimodal in nature. Additionally, PAL deficits were shown to be a consequence of difficulties in phonological form learning, rather than difficulties with associative learning. Taken together, these results caution against interpreting the reading and spelling difficulties of children with dyslexia as consequences of poor or inefficient orthography-phonology mappings. Although there is no doubt that children with dyslexia demonstrate poor learning in tasks that require such mappings (e.g., letter learning, reading), our data suggest that these deficits are neither specific to crossmodal mappings, nor do they arise at the level of the association.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jml.2013.10.005.

References

Baayen, R. H., Davidson, D. J., & Bates, D. M. (2008). Mixed-effects modelling with crossed random effects for subjects and items. *Journal of Memory and Language, 59*, 390–412.

Barr, D. J., Levy, R., Scheepers, C., & Tily, H. J. (2012). Random effects structure for confirmatory hypothesis testing: Keep it maximal. Ms. University of Glasgow.

Bates, D., Maechler, M., & Bolker, B. (2011). Package lme4. R. <http://www.cran.r-project.org>.

de Jong, P. F., Seveke, M. J., & van Veen, M. (2000). Phonological sensitivity and the acquisition of new words in children. *Journal of Experimental Child Psychology, 76*, 275–301.

Duyck, W., Szmalec, A., Kemps, E., & Vandierendonck, A. (2003). Verbal working memory is involved in associative word learning unless visual codes are available. *Journal of Memory and Language, 48*, 527–541.

Elbro, C. (1998). When reading is "readn" or somthn. Distinctness of phonological representations of lexical items in normal and disabled readers. *Scandinavian Journal of Psychology, 39*, 149–153.

Elbro, C., de Jong, P. F., Houter, D., & Nielsen, A. M. (2012). From spelling pronunciation to lexical access: A second step in word decoding? *Scientific Studies of Reading, 16*, 341–359.

Elbro, C., & Jensen, M. N. (2005). Quality of phonological representations, verbal learning, and phoneme awareness in dyslexic and normal readers. *Scandinavian Journal of Psychology, 46*, 375–384.

Fleiss, J. L. (1994). Measures of effect size for categorical data. *The Handbook of Research Synthesis*, 245–260.

Foster, H. (2007). *Single word reading test 6-16*. GL Assessment Limited.

Fowler, A. (1991). How early phonological development might set the stage for phoneme awareness. In S. Brady & D. Shankweiler (Eds.), *Phonological processes in literacy* (pp. 97–117). Hillsdale, NJ: Erlbaum.

Freedman, M. L., & Martin, R. C. (2001). Dissociable components of short-term memory and their relation to long-term learning. *Cognitive Neuropsychology, 18*, 193–226.

Gathercole, S. E., & Baddeley, A. D. (1990). The role of phonological memory in vocabulary acquisition: A study of young children learning new names. *British Journal of Psychology, 81*, 439–454.

Gathercole, S. E., Hitch, G. J., Service, E., & Martin, A. J. (1997). Phonological short-term memory and new word learning in children. *Developmental Psychology, 33*, 966.

Gupta, P., & MacWhinney, B. (1997). Vocabulary acquisition and verbal short-term memory: Computational and neural bases. *Brain and Language, 59*, 267–333.

Haddock, C. K., Rindskopf, D., & Shadish, W. R. (1998). Using odds ratios as effect sizes for meta-analysis of dichotomous data: A primer on methods and issues. *Psychological Methods, 3*, 339.

Hulme, C., Goetz, K., Gooch, D., Adams, J., & Snowling, M. J. (2007). Paired-associate learning, phoneme awareness, and learning to read. *Journal of Experimental Child Psychology, 96*, 150–166.

Hulme, C., & Snowling, M. (1992). Deficits in output phonology: An explanation of reading failure? *Cognitive Neuropsychology, 9*, 47–72.

Hulme, C., Maughan, S., & Brown, G. D. (1991). Memory for familiar and unfamiliar words: Evidence for a long-term memory contribution to short-term memory span. *Journal of Memory and Language, 30*, 685–701.

Hulme, C., Roodenrys, S., Brown, G., & Mercer, R. (1995). The role of long-term memory mechanisms in memory span. *British Journal of Psychology, 86*, 527–536.

Hulme, C., Roodenrys, S., Schweickert, R., Brown, G. D., Martin, M., & Stuart, G. (1997). Word-frequency effects on short-term memory tasks: Evidence for a redintegration process in immediate serial recall. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 23*, 1217.

Jaeger, T. F. (2008). Categorical data analysis: Away from ANOVAs (transformation or not) and towards logit mixed models. *Journal of Memory and Language, 59*, 434–446.

Lervåg, A., Bråten, I., & Hulme, C. (2009). The cognitive and linguistic foundations of early reading development: A Norwegian latent variable longitudinal study. *Developmental Psychology, 45*, 764.

Li, H., Shu, H., McBride-Chang, C., Liu, H. Y., & Xue, J. (2009). Paired associate learning in Chinese children with dyslexia. *Journal of Experimental Child Psychology, 103*, 135–151.

Liberman, I. Y., Mann, V. A., Shankweiler, D., & Werfman, M. (1982). Children's memory for recurring linguistic and non-linguistic material in relation to reading ability. *Cortex, 18*, 367–375.

Litt, R. A., de Jong, P. F., van Bergen, E., & Nation, K. (2013). Dissociating crossmodal and verbal demands in paired associate learning (PAL): What drives the PAL–reading relationship? *Journal of Experimental Child Psychology, 115*, 137–149.

Maindonald, J., & Braun, W. J. (2010). *Data analysis and graphics using R an example-based approach*. Cambridge: Cambridge University Press.

Mayringer, H., & Wimmer, H. (2000). Pseudoname learning by German-speaking children with dyslexia: Evidence for a phonological learning deficit. *Journal of Experimental Child Psychology, 75*, 116–133.

Messbauer, V. C., & de Jong, P. F. (2003). Word, nonword, and visual paired associate learning in Dutch dyslexic children. *Journal of Experimental Child Psychology, 84*, 77–96.

Messbauer, V. C., & de Jong, P. F. (2006). Effects of visual and phonological distinctness on visual–verbal paired associate learning in Dutch dyslexic and normal readers. *Reading and Writing, 19*, 393–426.

Nation, K., Marshall, C. M., & Snowling, M. J. (2001). Phonological and semantic contributions to children's picture naming skill: Evidence from children with developmental reading disorders. *Language and Cognitive Processes, 16*, 241–259.

Ramus, F., & Szenkovits, G. (2008). What phonological deficit? *The Quarterly Journal of Experimental Psychology, 61*, 129–141.

Ranbom, L. J., & Connine, C. M. (2011). Silent letters are activated in spoken word recognition. *Language and Cognitive Processes, 26*, 236–261.

Rastle, K., Harrington, J., & Coltheart, M. (2002). 358,534 nonwords: The ARC nonword database. *Quarterly Journal of Experimental Psychology A, 55*, 1339–1362.

Rigby, R. A., Stasinopoulos, D. M., & Akantziliotou, C. (2008). A framework for modelling overdispersed count data, including the Poisson-shifted generalized inverse Gaussian distribution. *Computational Statistics & Data Analysis, 53*, 381–393.

Roland, D. (2009). Relative clauses remodeled: The problem with mixed-effect models. *Poster presented at the CUNY 2009 conference on human sentence processing*.

Samuels, S. J., & Anderson, R. H. (1973). Visual recognition memory, paired-associate learning, and reading achievement. *Journal of Educational Psychology, 65*, 160–167.

Schneider, W., Eschman, A., & Zuccolotto, A. (2007). *E-prime: User's guide, Version 2.0*. Psychology Software Tools.

Seidenberg, M. S., & McClelland, J. L. (1989). A distributed, developmental model of word recognition and naming. *Psychological Review, 96*, 523–568.

Snowling, M. J. (1995). Phonological processing and developmental dyslexia. *Journal of Research in Reading, 18*, 132–138.

Snowling, M. J. (2000). *Dyslexia*. Oxford: Blackwell.

Snowling, M. J., & Hulme, C. (1994). The development of phonological skills. *Philosophical Transactions of the Royal Society of London. Series B: Biological sciences, 346*, 21–27.

Snowling, M. J., Stothard, S. E., Clarke, P., Bowyer-Crane, C., Harrington, A., Truelove, E., et al. (2009). YARC York Assessment of Reading for Comprehension Passage Reading.

Steger, J. A., Vellutino, F. R., & Meshoulam, U. (1972). Visual–tactile and tactile–tactile paired-associate learning by normal and poor readers. *Perceptual and Motor Skills, 35*, 263–266.

Storkel, H. L., Armbruster, J., & Hogan, T. P. (2006). Differentiating phonotactic probability and neighborhood density in adult word learning. *Journal of Speech, Language and Hearing Research, 49*, 1175.

Swan, D., & Goswami, U. (1997). Phonological awareness deficits in developmental dyslexia and the phonological representations hypothesis. *Journal of Experimental Child Psychology, 66*, 18–41.

Thomson, J. M., Richardson, U., & Goswami, U. (2005). Phonological similarity neighborhoods and children's short-term memory: Typical development and dyslexia. *Memory & Cognition, 33*, 1210–1219.

Thorn, A. S., & Frankish, C. R. (2005). Long-term knowledge effects on serial recall of nonwords are not exclusively lexical. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 31*, 729.

Torgesen, J. K., & Murphey, H. A. (1979). Verbal vs. nonverbal and complex vs. simple responses in the paired-associate learning of poor readers. *The Journal of General Psychology, 101*, 219–226.

Torgesen, J. K., Wagner, R. K., & Rashotte, C. A. (1999). *Test of word reading efficiency*. Austin, TX: Pro-Ed.

Truman, A., & Hennessey, N. W. (2006). The locus of naming difficulties in children with dyslexia: Evidence of inefficient phonological encoding. *Language and Cognitive Processes, 21*, 361–393.

Tunmer, W. E., & Chapman, J. W. (2012). Does set for variability mediate the influence of vocabulary knowledge on the development of word recognition skills? *Scientific Studies of Reading, 16*, 122–140.

Vellutino, F. R., Bentley, W. L., & Phillips, F. (1978). Inter-versus intra-hemispheric learning in dyslexic and normal readers. *Developmental Medicine & Child Neurology, 20*, 71–80.

Vellutino, F. R., Scanlon, D. M., & Bentley, W. L. (1983). Interhemispheric learning and speed of hemispheric transmission in dyslexic and normal readers: A replication of previous results and additional findings. *Applied Psycholinguistics, 4*, 209–228.

Vellutino, F. R., Scanlon, D. M., & Spearing, D. (1995). Semantic and phonological coding in poor and normal readers. *Journal of Experimental Child Psychology, 59*, 76–123.

Vellutino, F. R., Steger, J. A., Harding, C. J., & Phillips, F. (1975). Verbal vs non-verbal paired-associates learning in poor and normal readers. *Neuropsychologia, 13*, 75–82.

Vellutino, F. R., Steger, J. A., & Pruzek, R. M. (1973). Inter- vs. intrasensory deficit in paired associate learning in poor and normal readers. *Canadian Journal of Behavioural Science, 5*, 111.

Venezky, R. L. (1999). *The American way of spelling: The structure and origins of American English orthography*. New York, NY: Guilford.

Vousden & Ellefson (2012, July). The development of irregular word reading: The role of word knowledge. *Poster presented at the annual meeting of the Society for the Scientific Study of Reading, Montreal, CA.*

Wagner, R. K., & Torgesen, J. K. (1987). The nature of phonological processing and its causal role in the acquisition of reading skills. *Psychological Bulletin, 101*, 192–212.

Wagner, R. K., Torgesen, J. K., & Rashotte, C. A. (1994). The development of reading-related phonological processing abilities: New evidence of bi-directional causality from a latent variable longitudinal study. *Developmental Psychology, 30*, 73–87.

Wagner, R. K., Torgesen, J. K., & Rashotte, C. A. (1999). *Comprehensive test of phonological processing*. Austin, TX: Pro-Ed.

Warmington, M., & Hulme, C. (2012). Phoneme awareness, visual–verbal paired-associate learning, and rapid automatized naming as predictors of individual differences in reading ability. *Scientific Studies of Reading, 16*, 45–62.

Wechsler, D. (1993). *Wechsler objective reading dimensions*. London: The Psychological Corporation.

Wechsler, D. (1999). *Wechsler abbreviated scale of intelligence*. San Antonio, TX: Psychological Corporation.

Wimmer, H., Mayringer, H., & Landerl, K. (1998). Poor reading: A deficit in skill-automatization or a phonological deficit? *Scientific Studies of Reading, 2*, 321–340.

Windfuhr, K. L., & Snowling, M. J. (2001). The relationship between paired associate learning and phonological skills in normally developing readers. *Journal of Experimental Child Psychology, 80*, 160–173.