

■ Cognitive Profiles of Adult Developmental Dyslexics: Theoretical Implications

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The aim of this study was to establish cognitive profiles of dyslexic adults on tests developed within the three main theories of developmental dyslexia: phonological, visual magnocellular and cerebellar and to investigate which theory can account for these profiles. The sample consisted of 15 Polish university students or alumni with a formal diagnosis of dyslexia, without ADHD and 15 controls matched on education, age, gender, IQ and handedness. The results revealed a striking heterogeneity of profiles. Nine dyslexics exhibited only a phonological deficit; one a phonological and a visual magnocellular deficit; a further three a phonological and a cerebellar deficit; two either a cerebellar or a visual magnocellular deficit. None of the three main theories of dyslexia can account for all the cases studied here. It is suggested that the best account of these data is in terms of different sub-types of dyslexia with different underlying causes, such as phonological, visual magnocellular and cerebellar, or a combination of these. However, an account in terms of Ramus' (Trends, Neurosci. 2004; 27(12): 720–726) model, according to which the phonological deficit is a core deficit in dyslexia and other deficits (magnocellular and cerebellar), are just co-morbid markers without a causal relationship to dyslexics' literacy difficulties, cannot currently be ruled out. Copyright © 2006 John Wiley & Sons, Ltd.

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INTRODUCTION

Despite decades of research on dyslexia (the term 'dyslexia' is used throughout this article to denote developmental dyslexia), there is no wide agreement about the underlying cause or causes of this developmental disorder. A detailed account of all current theories of dyslexia is beyond the scope of this paper (for reviews e.g. see Miles & Miles, 1999; Ramus *et al.*, 2003; Rice & Brooks, 2004). Below we briefly describe three main current theories of dyslexia: the phonological deficit theory (PDT), the visual magnocellular deficit theory (MDT) and the cerebellar deficit theory (CDT). Note that over the years different versions of these theories have been developed. We therefore focus here on the well-established and most prominent versions of these theories. One exception here is the rapid auditory processing theory (e.g. Kujala *et al.*, 2000; Tallal, 1980; see also Farmer & Klein, 1995 for a review), more recently formulated as the auditory MDT (e.g. Stein, 2001). Evaluation of this theory has not been undertaken here, but has been included in our subsequent research.

The central claim of the PDT (e.g. Lundberg & Høien, 2001; Rack, Snowling, & Olson, 1992; Snowling, 2000) is that dyslexics have a specific impairment in representation and processing of speech sounds (phonemes). It is claimed that the phonological deficit is manifested by problems in several closely related sub-domains: phonological awareness (difficulties with analysing, blending and manipulating the sound structure of words), verbal short-term memory (difficulties with serial recall of digits, words and nameable visual stimuli, and repetition of pseudowords and sentences), word retrieval (reduced verbal fluency and naming speed) and recoding in reading and spelling (evident especially when processing unfamiliar words or pseudowords). It has been established that phonological impairment persists into adulthood (e.g. Paulesu *et al.*, 1996; Ramus *et al.*, 2003; Snowling, Nation, Moxham, Gallagher, & Frith, 1997). Furthermore, phonological impairment also remains in compensated dyslexics—individuals who despite their difficulties achieve an almost normal ability to read and write (e.g. Paulesu *et al.*, 1996). Biological support for the PDT comes from post-mortem anatomical studies (e.g., Galaburda, Sherman, Rosen, Aboitiz & Geschwind, 1985) and recent neuroimaging studies (e.g., Paulesu *et al.*, 1996; Shaywitz *et al.*, 1998). Both types of study have revealed, among other findings, significant differences between dyslexics and controls in the left perisylvian area, which is crucially involved in the auditory perception of words.

The MDT (Hansen, Stein, Orde, Winter, & Talcott, 2001; Stein, 2001; Stein & Talcott, 1999; Stein, Talcott, & Witton, 2001; Stein & Walsh, 1997) claims that the underlying cause of literacy problems in dyslexia is not language specific, but a more general impairment of the visual and/or auditory magnocellular system (with spared parvocellular system). The visual magnocellular system specializes in processing fast visual temporal information, whereas the auditory magnocellular system specializes in processing fast auditory temporal information. Stein and Walsh (1997) also suggested that the magnocellular temporal processing deficit extends to other systems, such as the vestibular and motor. In this paper we focused on the visual MDT. Three main types of evidence are cited in support of an impaired visual magnocellular system: unsteady binocular fixation (e.g. Stein, 2001), reduced motion sensitivity (e.g. Hansen *et al.*, 2001) and reduced contrast sensitivity (e.g. Lovegrove, Bowling, Badcock, & Blackwood, 1980; Lovegrove *et al.*, 1982).

According to Stein (2001) the magnocellular system helps to keep the two eyes fixated to converge on each word during reading. Many dyslexics have unsteady binocular fixation, and hence experience unstable perceptions of print (Stein *et al.*, 2001). They complain that letters seem to move around on the page, so that it is difficult to work out their order (Stein *et al.*, 2001). The binocular instability also manifests itself in significantly more visual errors made by dyslexics than by controls when reading words written in reduced letter size (Cornelissen, Bradley, Fowler, & Stein, 1991) and text where letters are relatively close to each other (Atkinson, 1991).

Studies on motion sensitivity assess the magnocellular function by determining what proportion of randomly moving dots have to move coherently for a participant to perceive the dots as moving together rather than randomly. A number of studies have reported that dyslexics have an elevated threshold for motion sensitivity as compared to controls (e.g. Hansen *et al.*, 2001).

As the magnocellular system is most sensitive to low contrasts, the MDT predicts that dyslexics will have problems with detecting low contrasts. Contrary to this, a literature review by Skottun (2000) suggests that although the results from some studies support the predictions of the MDT, the results from the majority of contrast sensitivity studies do not. A recent meta-analysis of contrast sensitivity and coherent motion detection studies has revealed, however, that magnocellular processing is reduced overall in groups of dyslexic readers in comparison with controls (Talcott, 2004).

Support for impairment in the visual magnocellular system on the biological level comes from post-mortem examinations of the brains of known dyslexics by Galaburda and colleagues. Selective disruption of magnocellular neurones was reported in the visual nuclei in the thalamus of dyslexics, with the dyslexics' magnocells being significantly smaller and more distorted than the cells of controls (e.g. Galaburda, Menard & Rosen, 1994).

Finally, we focused on the CDT (e.g. Fawcett & Nicolson, 1999; Nicolson & Fawcett, 1990; Nicolson, Fawcett, & Dean, 1995; 2001; Nicolson *et al.*, 1999). Originally this theory was formulated as an automatization deficit theory. It was supported by the findings from a dual task paradigm, which involved balancing, defined as maintaining one's body in a state of equilibrium, while performing a secondary task. The results revealed that although under optimal conditions dyslexics could balance as well as controls, in a dual task dyslexics balanced significantly worse than the controls (Nicolson & Fawcett, 1990). More recently, the automatised deficit has been linked to impaired function of the cerebellum. It has been reported that dyslexics have deficits in a range of functions, which rely on cerebellar processing, such as motor skills (Fawcett & Nicolson, 1999), eye-blink conditioning (Nicolson, Daum, Schugens, Fawcett, & Schulz, 2002) and time estimation (Nicolson *et al.*, 1995). Converging biological evidence of cerebellar impairment has come from a histological study (Finch, Nicolson, & Fawcett, 2002) that re-analysed the brain specimens of people with dyslexia originally investigated by Galaburda and colleagues (e.g. Galaburda *et al.*, 1994). The results showed significant differences in the numbers of large and small cerebellar neurones in dyslexics and controls (see also Rae *et al.*, 1998). On the basis of findings implicating cerebellar impairment in dyslexia, Nicolson *et al.* (2001) proposed a hypothetical causal chain linking cerebellar impairment with phonological processing deficits, and problems with reading and spelling.

A critical evaluation of the evidence gathered in support of the above theories is beyond the scope of this paper (see Ramus *et al.*, 2003, for critical comments). Here we would only like to emphasize two main shortcomings of the existing studies. First, the proponents of the main current theories of dyslexia, with relatively few exceptions (e.g. Kronbichler, Hutzler, & Wimmer, 2002; Ramus *et al.*, 2003), have usually restricted their investigations to a single cognitive domain that they consider crucial. However, it is becoming clear that dyslexics may show difficulties across a whole range of skills and a selective focus on a single domain can lead to limited conclusions (Reid, 2001; Reid & Szczerbinski, 2002). Second, dyslexics are a heterogeneous population, but the vast majority of studies with few exceptions (e.g. Fawcett & Nicolson, 1999; Fawcett, Nicolson, & Dean, 1996; Ramus *et al.*, 2003; van Ingelghem *et al.* 2001) have reported only group differences, so it is not possible to determine what proportion of dyslexics in a given study actually had a given deficit. In the light of growing evidence that dyslexics may be affected by a whole range of deficits, the first aim of our study was to establish what profiles are exhibited by individual dyslexic adults across the domains that are the focus of the main current theories of dyslexia outlined above. Once these profiles were identified we asked whether any of the main current theories of dyslexia could account for them.

It was hypothesized that if dyslexia is due to a phonological deficit (e.g. Snowling, 2000) dyslexics in our study would be impaired on most or all of the tests involving phonological processing (phonological awareness, phonological fluency, and rapid naming), but none of the others. If this developmental disorder is due to a visual magnocellular deficit (e.g. Stein, 2001), a raised detection threshold for motion coherence for dyslexics was predicted on the visual magnocellular test. If dyslexia is due to a cerebellar deficit (e.g. Nicolson *et al.*, 2001), it was hypothesized that dyslexics would be impaired on time estimation tests and/or the postural stability tests. The MDT and CDT would additionally predict difficulties on phonological tests, due to magnocellular or cerebellar impairment causing phonological deficit. Furthermore, the MDT and CDT would also allow for other routes to impair literacy skills. For instance, a deficit in vergence control (a result of a magnocellular deficit) could directly impair reading, whereas cerebellar impairment could directly impair acquisition and performance of reading and spelling as they require precise timing and automatization.

For clarity, it must be noted that each theory of interest can be described in terms of three levels of explanation (Frith, 1999): the biological level, the cognitive level and the behavioural level, with environmental influence on each level (for a detailed description see Frith, 1999; Ramus, 2004). The cognitive level abilities and deficits cannot be directly measured, but can be inferred from the behavioural measures (Frederickson, Frith & Reason, 1997). All the measurements across different theoretical frameworks reported here are on the behavioural level and are therefore directly comparable.

METHOD

Participants

Fifteen Polish university students or alumni with dyslexia and without any other known neurological or psychiatric disorder and 15 controls participated in the

study. Dyslexic and control groups were matched for handedness (all participants were right-handed), education (the same or related university degree), age (age range between 19 and 31 years, [$t(28) = 0.529, p = 0.601$]) and IQ (seven dyslexics and seven controls within the high average range and eight dyslexics and eight controls within the average range). There were no significant differences between the groups on verbal, performance and full scale IQ [$t(28) = 0.319, p = 0.752$; $t(28) = 1.164, p = 0.254$, $t(28) = 0.808, p = 0.426$, respectively]. The participants reported very few characteristics that could be indicative of ADHD [$t(28) = 0.634, p = 0.532$] (see Table 1).

Since no standardized literacy tests appropriate for our target population exist in Polish, we adopted double criteria. First, all individuals included in the dyslexic group had to have a statement of special educational needs indicating dyslexia. All except two dyslexics were diagnosed with dyslexia in their teenage years (12–17 years of age). DM and PS were diagnosed in their twenties. All were diagnosed in psychological clinics by a formal psychological assessment, which normally involved: WAIS-R (PL), tests tapping into literacy skills (reading aloud, spelling and writing to dictation), phonological processing, semantic knowledge and memory. Second, our own assessment had to indicate a full-scale IQ within the average or above average range (>90) (WAIS-R (PL)) because we wanted to exclude the possibility that dyslexics' literacy difficulties are due to low intellectual abilities. We have adopted the full-scale IQ score > 90 as a cut-off point following other researchers (e.g. Nicolson *et al.*, 2002). In addition, the dyslexics' performance had to be worse than 2 S.D. below the mean of the control sample on at least one of the following literacy tests: word recognition, decoding, reading text aloud and spelling. It should be noted that almost all dyslexics in our sample were impaired on the pseudoword reading rate test ($n = 12$; all except: EW, SER & TC), the text reading aloud accuracy ($n = 14$; all except SR), the text reading aloud time ($n = 11$; all except: EW, DM, SER & EP) and the spelling test ($n = 13$; all except: EW & DM) on a deviance criterion of 1.65 S.D. below the mean of the control group, which we adopted here following Ramus *et al.* (2003). In contrast, only four dyslexics (UO, KJ, KB & LS) were impaired on the word reading rate test. A plausible explanation for these results is that the pseudoword reading rate test and the spelling test are some of the most sensitive

Table 1. Characteristics of the sample

	Dyslexics (6 females, 9 males)		Controls (6 females, 9 males)	
	Mean (S.D.)	Min–max	Mean (S.D.)	Min–max
Age (in years)	23.8 (2.8)	20–31	23.3 (2.7)	19–30
Handedness ^a	85.5 (14.4)	60–100	82.7 (18.7)	33.3–100
ADHD ^b	1.1 (1.1)	0–3	0.8 (1.7)	0–5
FSIQ ^c	110.0 (9.9)	94–134	112.8 (9.1)	103–133
VIQ ^c	112.0 (8.9)	100–139	113.0 (8.3)	102–128
PIQ ^c	105.8 (11.5)	82–130	110.3 (9.7)	90–128

Note: ^aLaterality quotients (Oldfield, 1971). Range from –100 (left-handed) to 100 (right-handed).

^bADHD questionnaire (18 items).

^cfull-scale, verbal and performance IQ measured with WAIS-R (PL).

tests for identification of dyslexia in adulthood (Miles, 1993; Snowling, 2000). The text reading aloud test consisted of high frequency words and low-frequency words including technical terms, the majority of which had not been encountered by our sample before, and therefore had some properties of the pseudoword reading test. The word reading rate test, on the other hand, is very susceptible to compensation effects due to variables such as print exposure, reading strategies and others. Therefore, as far as the most sensitive tests for literacy deficits in adulthood are concerned, our dyslexic sample was relatively homogenous. To avoid a potential bias in our sample, no other selection criteria were used. One dyslexic had to be excluded as his literacy difficulties were not severe enough to meet our criteria. His matched control was also removed. Two other control participants were also excluded from the study—one male because of low scores on literacy tests and one female because of a full-scale IQ score of 86, which did not meet our criteria. They were replaced with two new controls.

Materials

Structured interview

The interview consisted of questions on the pattern of difficulties experienced by participants, health issues and the pattern of literacy and learning difficulties in the participants' family. Only the dyslexic group was interviewed.

The Edinburgh handedness inventory

This instrument (Oldfield, 1971) consists of a list of 10 questions probing direction and strength of hand preference in 10 everyday activities.

ADHD measure

We devised a structured interview probing participants' *inattention* (nine questions), *hyperactivity* (six questions) and *impulsivity* (three questions). It was based on the DSM-IV criteria for diagnosing ADHD.

WAIS-R (PL)

A full Polish version of WAIS-R was administered.

Literacy tests

Word reading rate test: The test consisted of two lists of single Polish words (75 words per list arranged in three columns). The dependent variable was the number of words read aloud correctly from each list in 30 s; the two scores were added giving the reading rate per minute.

Pseudoword reading rate test: The test consisted of 69 pseudowords which were in line with the phonotactic and graphotactic rules of Polish. The layout and the outcome measure were the same as in the word reading rate test.

Text reading aloud test: The test consisted of 19 unconnected sentences, which contained a total of 398 high- and low-frequency words. Each sentence described either medical, linguistic, statistical or genetic phenomena using technical terminology. The sentences were pseudorandomly intermixed to minimize context facilitation, and presented as text. The dependent variables were the number of reading errors and the time of reading the whole text.

Reading comprehension test: Two passages from the 2001 entry examination for the psychology undergraduate course at the Jagiellonian University (Cracow) were used: 'differences in linguistic systems in various languages' (386 words) and 'unconscious mechanisms of modelling culturally based behaviour' (530 words). Comprehension was tested with five and four multiple-choice questions, respectively. Participants read the passages silently. The dependent variables were the percentage of correct answers and the time taken to complete the whole task.

Spelling test: A modified version of the unpublished orthography test for Polish secondary school pupils was used. It consisted of 23 two-word phrases and 3 three-word phrases, plus 4 two-word orthographically easy filler items. The majority of words were orthographic exceptions (i.e. their spelling was unpredictable given the orthographic rules of Polish). The experimenter dictated the phrases. It was possible to make more than one error in a given word. The dependent variable was the number of spelling errors.

Naming and alliteration fluency tests

Rapid Automatized Naming (RAN) tests: Six tests were devised, four of them tested the ability to name items belonging to a single category (pictures, colours, digits, letters) and two tested mixed category naming (pictures and colours, digits and letters). Single category tests included five items and each item was repeated 10 times in a pseudorandom order, giving a total of 50 stimuli printed on a single page in a five-row array. The mixed category tests used the same items but combined the two types (five pictures and five colours; five digits and five letters), each item was repeated five times in a pseudorandom order on a page. Single category tests always preceded mixed category ones. Target words were 1–2 syllables long except for colour names which were 3–4 syllables long. The dependent variable for each test was the number of items named correctly in 15 s.

Alliteration fluency test: The test required participants to produce as many words starting with /t/ (first trial) and with /m/ (second trial) as possible within 30 seconds (on each trial). The dependent variable was the average number of items produced on two trials.

Semantic fluency test: The participants were requested to produce as many names of animals which have fur (first trial) and names of vegetables (second trial) as possible within 30 s (on each trial). Other details were as for the alliteration fluency test.

Phonological awareness tests

Spoonerisms test: The test consisted of 16 adjective–noun word pairs of increasing length (from 2-2 to 5-3 syllables long). The task was to swap the initial phonemes of the words in a pair. Correct responses were phonologically legal pseudowords. The whole session (including the experimenter's presentation of the stimuli and the participant's responses) was timed. The dependent variables were accuracy and time.

Sound unit deletion test: The test consisted of 16 3–5 syllable words. The participants were asked to delete phonological units of various size (single consonants, syllables, intrasyllabic units, and units larger than one syllable) at the

onset, offset and middle of words. Correct responses were phonologically legal pseudowords. The other details were the same as for the Spoonerisms test.

Magnocellular processing test

The coherent visual motion test (motion coherence) (MOTDX version 1.96; Hansen, 1995) was used to tap into the magnocellular system. The test allows one to determine what percentage of moving dots has to move in synchrony to enable a participant to detect a coherent motion.

The motion coherence stimuli were random dot kinematograms consisting of two patches of 300 high-luminance (80.6 cd/m^2) white dots (1 pixel) presented side-by-side on the black background (0.98 cd/m^2) of a laptop computer screen. Michelson contrast $[(L_{\max} - L_{\min}) / (L_{\max} + L_{\min})]$ between the black background of the laptop screen and the luminance (L) of the dots was 97.6%. The panel sizes subtended approximately $10 \times 14^\circ$ of visual angle, with a gap of 5° in between. One patch had a varying percentage of coherently moving dots, alternating to left and right and the dots reversed direction every 572 ms. The other dots presented on this patch moved randomly in a Brownian manner. The second patch consisted only of dots with Brownian motion. Each trial involved indicating (by pressing an appropriate key) the panel where dots were moving coherently. Each pair of panels was presented for 2.3 s and participants had unlimited time to make their judgement for each trial, after its presentation. Auditory feedback was provided on individual trials and after each block of trials the participant saw their score for a given block on the screen. The coherence of the dot movements was adjusted by the MOTDX software using a Kaernbach (1991) adaptive staircase technique. For each correct response this procedure reduces the coherence of the target stimulus by 1 dB (a factor of 1.122), whereas for each incorrect response the proportion of signal elements was increased by 3 dB (a factor of 1.412). The test consisted of four blocks of trials. The detection threshold for motion coherence for each block was defined as a geometric mean (robust for outliers) calculated using the scores for the final eight reversals on an adaptive Kaernbach (1991) staircase. The first two reversals were excluded from the calculation to minimize the biasing effect of early participant errors. The final calculated threshold coherence was corrected for the finite dot lifetime. The participants were tested individually in a quiet darkened room under mesopic (intermediate levels of illumination) viewing conditions and it was ensured that there was no glare on the laptop screen. The viewing distance was approximately 44 cm. The participants were not dark adapted. After each block there was a short break. The performance in the first block was treated as a practice and not included in the analysis. The dependent variable was the overall detection threshold for motion coherence—an average across the last three blocks of trials.

The static coherent visual form test (form coherence) (FORM version 1.16; Hansen, 1999) was used as a control test for the coherent visual motion task. This task was designed to bias towards the functioning of the parvocellular system. The stimuli consisted of two patches of 900 high-luminance white line segments, each 0.4° in length, presented side-by-side on the black background of a laptop screen. The values of the size of the patches and luminance of the line segments were matched to the values used in the coherent visual motion test. One patch had a varying percentage of coherent targets; these were defined as line segments

that were oriented in tangent to imaginary concentric circles within an area of 8° in diameter. The noise stimuli had random orientation. The second patch consisted only of line segments with random orientation. Each trial involved indicating, by pressing an appropriate key, the panel where the line segments formed a circle in the centre of the patch. The test consisted of four blocks of trials. The threshold for detection of the circle pattern per block was defined as the geometric mean of the last 8 of 10 reversals on an adaptive Kaernbach (1991) staircase. The dependent variable was the overall detection threshold for form coherence—averaged across the last three blocks of trials.

Cerebellar tests

The postural stability test: The postural stability test (Fawcett & Nicolson, 1998) involved testing participants' balance using a balance tester (the plastic device included in the testing kit in the Dyslexia Adult Screening Test—DAST, Fawcett & Nicolson, 1998), which was calibrated to provide a 4 kg force. The participants were instructed that the experimenter would push them gently in the back and their task was to stay as still as they could. The testing was done under two conditions: blindfolded, as specified in DAST and counting backwards, as reported in Nicolson and Fawcett (1990). In the former condition, participants were blindfolded and stood with their feet together. They had their hands by their side on two trials and their arms stretched out in front of them on another two trials. In the counting backwards condition the participants had to count backwards, instead of being blindfolded. The purpose of the blindfolding and counting backwards was to minimize the possibility of conscious compensation for any difficulties with balancing.

Both conditions were scored according to the DAST manual (Fawcett & Nicolson, 1998). The dependent variables were the sums of points over the four trials (for each condition) denoting the quality of balancing (with higher scores indicating greater imbalance).

Time and loudness estimation tests: The time estimation test (Ramus *et al.*, 2003) was identical to the one used by Nicolson *et al.* (1995). It was designed to tap into cerebellar timing functions. Each trial consisted of two tones of different duration, which were separated by a 1 s interval. The participants had to indicate (by pressing the appropriate key) whether the second tone was shorter or longer than the first one. There were 66 randomly presented experimental trials, which involved 22 comparisons (each comparison was repeated three times). The reference stimulus (a pure tone of frequency 392 Hz and length of 1200 ms) was always presented first, followed by a comparison tone which had the following durations: 400, 700, 800, 900, 950, 1000, 1050, 1100, 1140, 1160, 1180, 1220, 1240, 1260, 1300, 1350, 1400, 1450, 1500, 1600, 1700 and 2000 ms. Visual feedback was provided on eight practice trials, but not on the experimental trials. The dependent variable was the just-noticeable difference, i.e. a duration difference at which a participant was 75% correct at responding that a comparison (second) tone was shorter than the reference (first) tone.

Following Nicolson *et al.* (1995) a loudness estimation test (Ramus *et al.*, 2003) was used as the control task. The design of this test closely matched the design of the time estimation test. All tones were 1000 Hz and 1 s long and differed only in loudness. The comparison tones had amplitudes greater or smaller than the

reference stimulus by the following values: 4, 8, 12, 16, 20, 26, 32, 38, 46, 56 and 70%. The participants' task was to indicate by pressing the appropriate key whether the comparison (second) tone was louder or quieter than the reference (first) one. The dependent variable was the just-noticeable difference at which each participant was 75% correct on responding that a comparison tone was 'softer' (i.e. quieter) than the reference tone. Both tests were presented on a laptop computer through Panasonic (RP-HT237) digital stereo headphones at approximately 75 dB SPL.

Procedure

Every participant was tested individually in a quiet room. The testing time was divided into two sessions. Each session lasted approximately 2–2.5 h with at least one break in the middle. Each session took place on two different days separated by from 1 to 26 days, dependent on participants' schedules. The first testing session started with an ADHD assessment for all the participants. The order of tests was fixed, interchanging tests of different levels of difficulty where possible. This order was reversed for half of each group of participants.

RESULTS

Group comparisons

Literacy tests

Dyslexics were significantly worse than controls on five out of the seven literacy indices: word reading rate [$t(28) = 2.990, p = 0.006$], pseudoword reading rate [$t(28) = 6.785, p < 0.001$], text reading aloud errors [$t(28) = 7.021, p < 0.001$], text reading aloud time [$t(28) = 6.116, p < 0.001$], and spelling accuracy [$t(28) = 7.308, p < 0.001$]. However, the groups did not differ on reading comprehension—either on accuracy [$t(28) = 0.018, p = 0.986$], or on time [$t(28) = 1.230, p = 0.229$] (see Table 2). The literacy profile of our dyslexic sample is thus consistent with the conceptualization of dyslexia as a specific bottom-up difficulty in processing written language whose core symptoms lie at the level of individual words (Lyon, Shaywitz, & Shaywitz, 2003; Reason, Frederickson, Heffernan, Martin, & Woods, 1999) and which can be compensated for by relying on top-down contextual information.

Naming and phonological fluency tests

Dyslexics' performance differed significantly from the performance of controls on all RAN tasks: pictures [$t(27) = 2.155, p = 0.040$], colours [$t(28) = 2.519, p = 0.018$], pictures and colours [$t(28) = 2.277, p = 0.031$], digits [$t(28) = 3.272, p = 0.003$], letters [$t(28) = 2.899, p = 0.007$], and digits and letters [$t(28) = 3.431, p = 0.002$] (see Table 3). There were no significant differences between the performance of dyslexics and controls on the alliteration and semantic fluency tests [$t(28) = 0.579, p = 0.568$; $t(28) = 0.484, p = 0.632$, respectively]. From our experience gained during the testing sessions, it seems that these tasks can be significantly affected by strategic effects used by participants.

Table 2. Performance on literacy tests

Literacy tests	Dyslexics		Controls		Effect size (<i>d</i>)
	Mean (S.D.)	Min-max	Mean (S.D.)	Min-max	
Word reading rate (per min)	113.7 (14.5)	87-139	129.3 (14.1)	102-152	-1.1*
Pseudoword reading rate (per min)	60.1 (10.3)	47-87	86.1 (10.7)	65-101	-2.5**
Text reading aloud (number of errors)	20.3 (7.0)	9-35	6.5 (3.0)	2-12	-2.6**
Text reading aloud (time in s)	346.7 (46.6)	277-426	259.7 (29.4)	215-338	-2.2**
Reading comprehension ^a	67.8 (20.2)	33-100	67.7 (20.8)	30-100	0.0
Reading comprehension (time in s)	466.0 (106.9)	293-626	413.2 (127.3)	226-700.5	-0.4
Spelling (number of errors)	12.9 (5.0)	3-22	2.7 (2.1)	0-6	-2.7**

Note: Effect size = Cohen's *d*.

* $p < 0.01$; ** $p = 0.001$ (*t*-test).

^a(% correct; 9=100%).

Table 3. Performance on naming, alliteration fluency and phonological awareness tests

Tests	Dyslexics			Controls			Effect size (<i>d</i>)
	Mean (S.D.)	Min-max	Mean (S.D.)	Min-max	Mean (S.D.)	Min-max	
RAN pictures (items named in 15 s)	25.0 (5.2)	14-34	28.7 (4.1)	22-36			-0.8*
RAN colours (items named in 15 s)	25.4 (5.1)	15-32	29.2 (2.8)	24-34			-0.9*
RAN pictures and colours (items named in 15 s)	25.5 (5.2)	13-35	29.4 (4.0)	23-37			-0.8*
RAN digits (items named in 15 s)	39.9 (7.6)	25-50	47.9 (5.6)	40-63			-1.2**
RAN letters (items named in 15 s)	41.6 (7.1)	30-50	48.4 (5.7)	37-58			-1.1**
RAN digits and letters (items named in 15 s)	37.5 (5.7)	28-50	45.3 (6.6)	30-54			-1.3**
Alliteration fluency (items produced in 15 s)	10.6 (2.2)	7-14	11.1 (2.5)	6-15			-0.2
Semantic fluency (items produced in 30 s)	10.9 (2.8)	7.5-17	11.4 (2.5)	5.5-15.5			-0.2
Spoonerisms—accuracy (max=16)	9.1 (3.5)	2-14	11.8 (4.7)	2-16			-0.7^
Spoonerisms—time (s)	245.6 (97.6)	125-435	184.2 (73.4)	94-351			-0.7^
Sound unit deletion—accuracy (max=16)	11.7 (2.3)	8-15	13.3 (2.0)	9-16			-0.7^
Sound unit deletion—time (s)	110.5 (18.3)	86-142	95.5 (17.4)	62-141			-0.8*

Note: ** $p < 0.01$; * $p < 0.05$; ^ $p < 0.10$ (*t*-test).

For instance, some dyslexics used semantic categories (e.g. food or clothes) to retrieve words starting with a given sound in the alliteration task.

Phonological awareness tests

Dyslexics were significantly worse than the control group on sound unit deletion time [$t(28) = 2.302, p = 0.029$]. The between group differences on the remaining tests: Spoonerisms accuracy, Spoonerism time and phoneme deletion accuracy approached significance [$t(28) = 1.749, p = 0.091$; $t(28) = 1.947, p = 0.062$; $t(28) = 1.935, p = 0.063$], respectively.

Coherent visual motion test

There was no significant difference between the two groups on either the coherent visual motion test [$t(28) = 1.232, p = 0.228$] or the control test—the static coherent visual form test [$t(28) = 0.092, p = 0.927$] (see Table 4) although a trend in the direction predicted by the MDT (dyslexics having higher than the controls' threshold for the coherent visual motion test, but not for the static coherent visual form test) could be seen.

Cerebellar tests

The time estimation data were converted into the classification function, fitted separately for each participant using logistic regression analysis. The function described the relationship between the percentage of 'shorter' responses and the duration of the comparison tone. The procedure for the loudness estimation test was the same, but the classification function described the relationship between the percentage of 'softer' responses and the amplitude of the comparison tone. The parameters (slopes and intercepts) of these logistic functions were then used to estimate the just-noticeable difference (jnd) at which a given participant was 75% correct. All individual classification functions had slopes significantly greater than zero, indicating performance above chance level.

No significant differences were found between the dyslexics and controls on the time estimation test [$t(28) = 1.382, p = 0.178$] or the control test or the

Table 4. Performance on magnocellular and cerebellar tests and the control tests

Tests	Dyslexics		Controls		Effect size (<i>d</i>)
	Mean (S.D.)	Min-max	Mean (S.D.)	Min-max	
Coherent visual motion test ^a	9.3 (5.5)	2.8–23.3	7.3 (3.4)	3.2–14.0	–0.4
Static coherent visual form test ^a	16.6 (6.5)	10.6–35.5	16.4 (3.9)	11.8–24.6	0.04
Postural stability test (blindfolded) ^b	3.1 (4.6)	0–18	2.3 (3.8)	0–14	–0.2
Postural stability test (counting backwards) ^b	3.1 (4.7)	0–19	1.9 (3.3)	0–12	–0.3
Time estimation test – jnd (ms)	90.5 (26.3)	74.7–179.6	80.7 (6.9)	75.2–101.2	–0.5
Loudness estimation test – jnd (% amplitude)	3.7 (0.2)	3.5–4.2	3.6 (0.1)	3.5–3.9	–0.6

Note: ^a coherence at threshold (%).

^b Max=24. Higher scores indicate greater imbalance.

loudness estimation test [$t(28) = 1.160, p = 0.256$] (see Table 4). The groups also did not differ significantly on the postural stability tests [$t(28) = 0.565, p = 0.577$ for the blindfolded condition; $t(28) = 0.814, p = 0.423$ for the counting backwards condition]. It should be noted, however, that the postural stability test was designed as a screening test and as such might not be sensitive enough to detect subtle differences. We are currently running a research project where differences in postural stability in dyslexic and control groups are assessed using the Polhemus motion tracking system which allows for recording three-dimensional movement at a sampling rate of 12 points per millisecond.

Deviance Analysis

Two issues were addressed here. First, as the group analyses cannot usually reveal what proportion of dyslexics actually have a given deficit and most of the published studies did not investigate this issue, we wanted to address it in our study. Second, as there is growing evidence that dyslexics have heterogeneous profiles, we wanted to obtain detailed individual profiles and test whether they could be accounted for by the main theories of dyslexia.

To address these issues we followed the approach of Ramus *et al.* (2003) and used a deviance analysis. First, the raw scores for each measure (e.g. picture naming) for each participant were converted into standard (z) scores, relative to the mean and S.D. of the control group. Second, individual z scores were averaged across relevant measures to give five composite variables: LITERACY, PHONOLOGICAL FLUENCY, PHONOLOGICAL AWARENESS, CEREBELLAR and VISUAL MAGNOCELLULAR. The latter one was represented by just one variable—motion coherence. Averaging several imperfectly correlated standard score variables resulted in composites which, for the control group, had a mean of zero, but reduced standard deviations (less than one)—a consequence of the regression to the mean. We then restandardized those composites by dividing them by the S.D. of the control group. This way, we received standard composite scores for the control group (Mean=0, S.D.=1), with dyslexics' z scores being relative to the controls' z scores. As control participants can sometimes show abnormality on a given composite variable, we identified those controls whose composite z scores were 1.65 S.D. below the mean of the control group and excluded them (see below) from that composite. We then used those trimmed data to recalculate the mean and S.D. of the control group and used them to calculate the individual composite z scores for the dyslexic group. Dyslexics with abnormal performance (defined as $z < -1.65$, i.e. worse than 1.65 S.D. below the mean of the control group) were identified. All deficits reported below are at 1.65 S.D., or lower, below the trimmed control mean.

The performance on the literacy tests was summarized in LITERACY—a composite variable (see Table 5), which was computed as the average of z scores for all seven literacy measures. The two groups significantly differed on this variable [$t(28) = 8.775, p < 0.001$]. The deviance analysis on LITERACY (based on trimmed control data with one abnormal control score removed) revealed that all dyslexics had abnormal performance.

The outcome from the six RAN tasks and the phonological fluency test were summarized in PHONOLOGICAL FLUENCY—a composite variable (see Table 5 and Figure 1). There was a significant difference between dyslexics and controls

Table 5. Performance on the composite variables

Composite variables	Dyslexics Mean (S.D.)	Controls Mean (S.D.)
Literacy**	-3.6 (1.3)	0.0 (1.0)
Naming and phonological fluency*	-1.6 (1.4)	0.0 (1.0)
Phonological awareness*	-1.0 (1.0)	0.0 (1.0)
Visual magnocellular	-0.6 (1.6)	0.0 (1.0)
Cerebellar	-0.8 (1.8)	0.0 (1.0)

Note: z scores are relative to the Mean and S.D. of the untrimmed control sample.

** $p < 0.001$; * $p < 0.01$ (independent samples t -test).

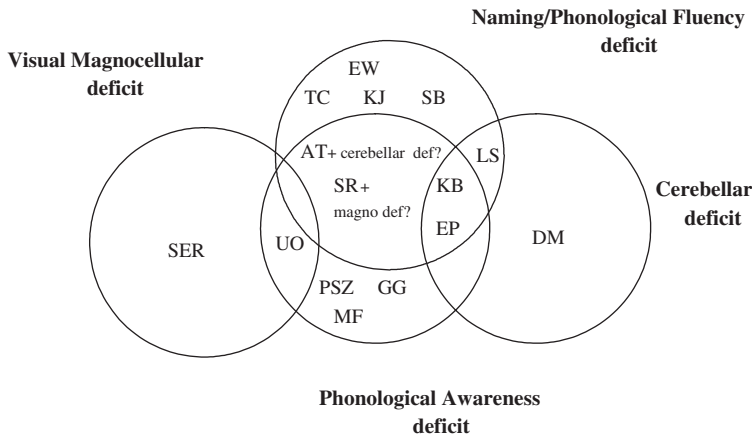


Figure 1. The distribution of cognitive and psychophysical deficits (on the 1.65 S.D. below the trimmed control mean criterion) among individuals with dyslexia. Initials denote names of individual dyslexics. The question mark denotes that it is not clear whether a person with dyslexia has a specific deficit because they also exhibited a deficit on the control task.

on this variable [$t(28) = 3.633, p = 0.001$]. The deviance analysis on the PHONOLOGICAL FLUENCY (based on trimmed control data with one abnormal control score removed) showed that nine dyslexics (EW, SR, EP, KJ, AT, KB, LS, SB & TC) and one control participant (WM) were impaired on this variable.

The performance on accuracy and speed in the Spoonerism test as well as accuracy and speed in the sound unit deletion test was summarized in the PHONOLOGICAL AWARENESS composite variable (see Table 5 and Figure 1). The two groups significantly differed on this variable [$t(28) = 2.806, p = 0.009$]. The deviance analysis on the PHONOLOGICAL AWARENESS (based on trimmed control data with two abnormal control scores removed) revealed that eight dyslexics (SR, UO, PSZ, MF, GG, EP, AT & KB) and one control participant (DP) were impaired.

As the earlier analysis indicated, there was no significant difference between dyslexics and controls on the VISUAL MAGNOCELLULAR variable [$t(28) = 1.243, p = 0.224$]. The deviance analysis, based on trimmed control data

with one abnormal control score removed, revealed that three dyslexics (SER, UO & SR) were impaired on the VISUAL MAGNOCELLULAR variable. As one of them (SR) also showed a deficit on the control test (the static coherent visual form) it is not clear whether her deviant performance on the experimental test was due to a visual magnocellular deficit or to another deficit. One control participant (PS) also showed deviance on both the experimental and control tasks. Another control participant (GB) was only impaired on the control task.

Finally, the performance on the two balance tests and the time estimation test were summarized in the CEREBELLAR variable. There was no significant difference between the groups on this variable [$t(28) = 1.651, p = 0.110$]. The deviance analysis (based on trimmed control data with one abnormal control score removed) showed that five dyslexics (DM, EP, AT, KB & LS) and two control participants (KM & DAM) were impaired. One dyslexic (AT) showed impairment on both time and loudness estimation and therefore it is not clear whether his deficit was due to a cerebellar or other deficit. Three dyslexics (DM, AT & SB) were impaired on the control test—the loudness estimation.

Further Analyses

Regression analysis of composite variables on Literacy

To investigate whether one can predict literacy performance from the composite variables, correlation and regression analyses were carried out. Only two out of four composite variables showed a significant correlation with LITERACY (see Table 6). These two variables (PHONOLOGICAL FLUENCY and PHONOLOGICAL AWARENESS) were then entered into a simultaneous multiple regression as predictors of LITERACY. Both the PHONOLOGICAL FLUENCY and PHONOLOGICAL AWARENESS composite variables accounted for unique variance in LITERACY (23% and 6%, respectively). However, note that the contribution of PHONOLOGICAL AWARENESS only approached significance ($p = 0.09$).

Table 6. Correlation and regression analyses for the untrimmed composite variables

	Zero-order Pearson correlations				Multiple regression		
	Lit	PhFlu	PhAw	Magno	β	ΔR^2	df
PhFlu	0.67**				0.565	0.27**	
PhAw	0.48**	0.38*			0.259	0.06 [^]	
Magno	0.17	-0.20	0.33 [^]				
Cereb	0.27	0.34 [^]	0.15	0.01			
					Total $R^2 = 0.50$ **		2, 29
					Shared $R^2 = 0.17$		
					Unique $R^2 = 0.33$		
					Adjusted $R^2 = 0.46$		

Note: PhFlu=PHONOLOGICAL FLUENCY; PhAw=PHONOLOGICAL AWARENESS; Magno=VISUAL MAGNOCELLULAR; Cereb=CEREBELLAR.

** $p < 0.01$; * $p < 0.05$; [^] $p < 0.10$.

Interview

Regarding literacy problems, all but three dyslexics (EW, AT & SB) reported that they had problems with learning to read. However, all of them reported having problems with reading in adulthood. All dyslexics had problems with learning to spell, which persisted into their adult life. Six dyslexics (UO, PSZ, MF, GG, EP & LS) reported literacy difficulties either in one of their parents and/or siblings.

Focusing on the visual problems possibly linked to the magnocellular deficit, all dyslexics except SB said that they often swapped the order of letters in words when reading. Nine (UO, SER, MF, GG, EP, KJ, AT, KB & SB) said that their eyes got tired quickly when they read. Two (MF & GG) reported unconscious skipping of lines when reading.

Regarding problems possibly linked to cerebellar function, all dyslexics except four (EW, SR, DM & UO) reported that they had problems with neat handwriting. Four (EW, OU, EP & TC) said that they would describe themselves as people with low manual dexterity. Five reported that they had problems with learning to ride a bike (UO, MF, EP, SB & TC). Four (EP, KB, LS & KJ) had problems with keeping to the rhythm of music. Interestingly, EP, KB and LS had a cerebellar deficit.

Other problems were also reported. For instance, all dyslexics, except four (SER, MF, SB & TC) reported problems with determining left and right side. Seven reported difficulties with structuring their essays logically (UO, PSZ, EP, KJ, AT, SB & TC) and nine (PSZ, SER, MF, GG, EP, KJ, AT, LS & SB) problems with the grammatical structure of sentences. Ten (SR, DM, UO, PSZ, SER, EP, KJ, KB, LS & SB) had problems with learning new terminology for their studies. Nine (EW, SR, DM, UO, PSZ, GG, EP, LS & TC) reported difficulties in getting all the sounds in the right order when pronouncing some low-frequency long words.

DISCUSSION

Two issues have been addressed in this report. First, we have established individual profiles of adult dyslexics across the domains corresponding to the three main theories of dyslexia: phonological, visual magnocellular and cerebellar. Second, we have asked whether any of the main current theories of dyslexia can account for these profiles.

Group comparisons revealed that the dyslexics were impaired on all the literacy tests, except the reading comprehension test. This suggests that these highly performing dyslexics could compensate for their reading deficit by relying on context. This is consistent with the conceptualization of dyslexic literacy difficulties as a bottom-up difficulty in processing written language, which can be compensated for by top-down processing (Lyon *et al.*, 2003; Reason *et al.*, 1999). As a group, the dyslexics were also impaired on most of the phonological tests, but not on the visual magnocellular and cerebellar tests.

Despite the straightforward group analysis results, the deviance analysis and the data collected in the interview showed that the individual dyslexics revealed complex and heterogeneous cognitive and psychophysical profiles (see Figure 1). These results are consistent with some earlier reports of heterogeneous difficulties in dyslexia (e.g. Miles, 1993; Riddick, Farmer, & Sterling, 1997).

We focus on the phonological deficit first. If we assume, as do the majority of the protagonists of the PDT, that phonological awareness, phonological fluency

and naming tasks are all indices of phonological processing, then 13 dyslexics showed a phonological deficit. The frequency of occurrence of this deficit in our sample was somewhat smaller (86.7%) than in Ramus *et al.*'s (2003) study (where the frequency of occurrence was 100%). Two of our dyslexics (SER & DM) did not exhibit a phonological deficit, despite having literacy difficulties.

Moving on to the visual magnocellular deficit, only two dyslexics showed a clear visual magnocellular deficit. The incidence of the visual deficit in dyslexics in our study was smaller (13.3%) than predicted by the estimates that two-thirds (66.7%) of the dyslexic population suffer from a visual magnocellular deficit (Stein, 2001). However, our result is consistent with studies, which have also reported a relatively low incidence of the visual magnocellular deficit (e.g. Ramus *et al.*, 2003—12.5%).

Finally four dyslexics suffered from a cerebellar deficit. The frequency of occurrence of this disorder in our study was lower (26.7%) than in studies reported by Nicolson *et al.* (2001), where 80% of dyslexics had a cerebellar deficit. Our results on the frequency of occurrence of a cerebellar deficit are congruent with the 25% estimate obtained by Ramus *et al.* (2003). However, the profiles of cerebellar deficits in our sample differed from those reported in Ramus *et al.* (2003). Three dyslexics in our sample (LS, EP & KB) had a deficit in time estimation without an automatic balance deficit and one (DM) had an automatic balance deficit without a time estimation deficit. In contrast, in Ramus *et al.*'s (2003) study, one dyslexic had a time estimation deficit (together with a loudness estimation deficit) and two dyslexics had a balance deficit (one with an automatic balance deficit).

It seems that none of the main current theories of dyslexia can account for all the cases of dyslexia we studied. The PDT can account for 13, the MDT for two (possibly three) and the CDT for four (possibly five) cases. Note however, that the more recent version of the MDT (Stein, 2001) states that the cerebellum can be considered as the most important part of the magnocellular timing system. If this is the case, then the MDT may perhaps predict that dyslexics will not only be deficient on the motion coherence task, but also on the cerebellar tests, in which case the MDT may perhaps account for 6 out of 15 cases (40%). Also, if one interprets dyslexics' impairments on RAN and alliteration tasks as due to a deficit in a timing mechanism (e.g. Wolf, Bowers, & Biddle, 2000) the cause of which lies in the impaired cerebellum, then the CDT would possibly account for the additional six cases (EW, TC, KJ, SB, AT & SR). However, it is not clear why these dyslexics, with the possible exception of AT, did not show a deficit on the time estimation test. It might be the case that the timing mechanism involved in RAN tasks and the timing mechanism needed to successfully complete the time estimation task involve different or partially different mechanisms.

Lack of a given deficit in adulthood does not necessarily imply that it was not present in the ontogenetic development of a given person. It could be that a deficit, or any combination of deficits were present at birth for the studied dyslexics, and although the developing system was compromised, so as to make literacy acquisition difficult, it compensated for the deficits in later development so they are not detectable in adulthood. Therefore to fully establish a causal relationship between dyslexia and the phonological, magnocellular and cerebellar deficits longitudinal data across these domains from birth to adulthood are necessary from individuals with familial risk of dyslexia.

In line with the deviance analysis, the interview data suggest heterogeneous and complex profiles of dyslexic difficulties. Interestingly, they also reveal that most of the dyslexics reported other difficulties in addition to the problems identified in the deviance analysis. For instance, 14 dyslexics (including four dyslexics who only exhibited a phonological deficit), reported visual problems which are possibly linked to magnocellular processing, such as a tendency to swap the order of letters in words when reading and/or to unconsciously skip lines when reading. Furthermore, all dyslexics, except SR, reported at least one problem possibly linked to a cerebellar deficit. Although the interview data need to be treated with caution because of potential confounding variables, they certainly raise the issues of whether the widely used cognitive and psychophysical tests are sensitive enough and whether they tapped into all crucial factors which contribute to literacy deficits. Unfortunately, we do not have interview data for the control group in this study. However, a comparable study (Reid & Hansen, 2006) revealed that English dyslexic university students indeed reported significantly more problems than controls, including difficulties, which may be related to visual magnocellular and cerebellar processing. These issues warrant further research.

Given this heterogeneity of dyslexics' psychophysical and cognitive profiles we put forward an explanation in terms of different sub-types of dyslexia with different underlying causes, such as phonological, visual magnocellular and cerebellar deficits or a combination of these. This is in line with an earlier hypothesis (e.g. Doehring, Trites, Patel, & Fiedorowicz, 1981) according to which a variety of factors may contribute to reading impairment, such as linguistic, visual and other deficits. Support for this earlier hypothesis came mainly from experimental subtyping studies (e.g. Doehring *et al.*, 1981; Watson, 1990). The difference between our hypothesis and the earlier hypothesis, however, is that we postulate a range of deficits, which have become much more precisely defined through work on the single underlying causes of dyslexia within the framework of the most up-to-date theories. In contrast to many subtyping studies, our hypothesis is based on data for adult dyslexics and therefore the deficits cannot be interpreted as due to developmental lag. Furthermore, contrary to many subtyping studies, every effort was made to exclude from our study dyslexics with other impairments, such as ADHD and low IQ, which may have confounded the work of the subtyping studies. It should also be noted that the approach taken in our research differs from the current approach to dyslexia taken by a significant number of researchers who have focused on a single underlying cause of this disorder (e.g. Nicolson *et al.*, 2001; Shaywitz, 1996; Snowling, 2000; Stein, 2001).

One problem with the account presented here is that while the PHONOLOGICAL AWARENESS composite approached significance in predicting literacy and the PHONOLOGICAL FLUENCY composite was a statistically significant predictor of literacy, the VISUAL MAGNOCELLULAR and CEREBELLAR composites were not, suggesting that the latter two deficits have no bearing on the literacy problems exhibited by dyslexics. However, these negative results are likely to reflect the low statistical power of our small sample analyses. Also, failing to detect a deficit in a behavioural or psychophysical task does not necessarily imply that there is no deficit on the neurophysiological level (e.g. see Paulesu *et al.*, 1996), and that it could not be detected on the behavioural and

psychophysiological levels with more sensitive tests. Furthermore, there are data which suggest that visual magnocellular and cerebellar processing may have an impact on literacy skills. Focusing first on visual magnocellular processing, Talcott *et al.*'s (2002) study, with a large sample ($N = 350$) of randomly selected primary school children, established that visual magnocellular processing, as measured with the coherent visual motion test, was a small, but significant predictor of literacy. It accounted for 7% of the variance in literacy and 4% of this variance overlapped with non-verbal intelligence. The evidence that cerebellar impairment is a significant predictor of literacy skills is limited. Ramus and colleagues (Ramus, Pidgeon, & Frith, 2003; Ramus *et al.*, 2004) addressed this issue and concluded that performance on cerebellar/motor tests does not predict literacy skills. Despite the difficulties in establishing that cerebellar skills are a significant predictor of literacy skills, there is growing evidence from patient and neuroimaging studies, which suggests that the cerebellum is involved in literacy skills. For instance, studies by Moretti, Bava, Antonello, & Torre (2002) have reported reading deficits in patients with cerebellar vermis lesions. Also, Turkeltaub, Eden, Jones and Zeffiro (2002) reported a meta-analysis of 11 PET studies and concluded that the cerebellum is reliably activated together with other areas during single word reading. Furthermore, Fulbright *et al.*'s (1999) study showed differential activation of cerebellar structures for phonological and semantic processing during reading.

Another potential criticism of this account is that magnocellular and cerebellar deficits also occur in other populations, such as in autistic children without reading problems (Ramus *et al.*, 2004) and the normal population. Therefore, it could be argued that visual magnocellular and cerebellar deficits are neither necessary, nor sufficient to cause literacy difficulties (Ramus *et al.*, 2004). Additional analysis of our data (not reported here) showed that if the untrimmed composite z scores of dyslexics' and controls' are compared, (a comparison which makes it harder to detect deficits in the dyslexic sample), then similar (low) numbers of dyslexics and controls exhibited the magnocellular and cerebellar deficits. This certainly poses the question of whether these deficits could determine dyslexia, because they are also exhibited by participants without literacy problems. Interestingly, the same analysis showed that although many more dyslexics than controls exhibited phonological awareness and phonological fluency deficits, one control participant exhibited a phonological deficit and another control participant exhibited a phonological fluency deficit. Therefore, how can one have a phonological deficit and do not exhibit literacy problems? One explanation might be that if a person has a phonological and/or magnocellular, and/or cerebellar deficit, but some protective factors operate in their ontogenetic development, such as good language skills (e.g. Snowling, 2001), strengths in other relevant areas of cognitive and psychophysical processing, successful reading and spelling instruction, etc., literacy deficits can be prevented.

It is possible that a causal model of dyslexia recently put forward by Ramus (2004) could also account for our data. According to this model the phonological deficit is a core deficit in dyslexia and other deficits, such as magnocellular and cerebellar, are just simple co-morbid markers without a causal relationship to dyslexics' literacy difficulties. However, an explanation along the lines of Ramus (2004) has three shortcomings. First, it is not clear how it could account for the cases such as SER and DM, who despite literacy difficulties did not exhibit a

phonological deficit (see e.g. Valdois, Bosse, & Tainturier, 2004, for a review of case studies of developmental dyslexics without a phonological deficit). Second, although most advocates of the PDT interpret a deficit in naming (such as found in EW, TC, KJ & SB) as a deficit in phonological retrieval, it could also be due to other—non-phonological factors, such as a deficit in the precise timing mechanism (e.g. Wolf, Bowers, & Biddle, 2000). If this is the case, then it is hard to see how such cases can be accounted for by Ramus' (2004) model.

Third, in cases where a given dyslexic has two deficits, such as phonological and magnocellular (as in the case of UO in our study), he/she may (as UO did) report difficulties such as swapping the order of letters in words—which would influence the reading process over and above the phonological problems. In such cases, therefore it does not seem reasonable to claim that the phonological deficit is a core one, and the other deficit just co-occurs and does not influence the literacy skills. According to the PDT and MDT these deficits are both inborn and would operate independently in ontogenetic development, at least to a certain degree (see the hypotheses in the introduction). One may argue, nevertheless, that if a magnocellular deficit co-occurs with a phonological deficit, as in case of UO, it may exacerbate dyslexic difficulties, but on its own it cannot cause dyslexia. However, the case of SER in our study, who has literacy difficulties, exhibited only the magnocellular deficit and reported swapping the letters in words when reading, seems to provide evidence against this argument.

One shortcoming of this study is that the number of measures obtained for each theoretical domain was not equal, with the largest number of phonological measures, then cerebellar measures and with only one visual magnocellular measure. It should be noted, however, that the frequency of occurrence of the deficits in our dyslexic group is very similar to that obtained in Ramus *et al.*'s (2003) study, which used a more balanced number of tasks for each domain. The other shortcoming of our study, similarly to the majority of studies reported on dyslexia, is that it is based on a relatively small sample. This has two consequences. First, the results cannot be generalized onto the population of dyslexics. Second, a wide heterogeneity of dyslexic profiles and relatively low frequency of some deficits makes it difficult to establish the causal link between the less frequently occurring deficits and literacy skills. However, the lack of evidence for such a causal link in a small sample cannot be interpreted as evidence of no link. Longitudinal studies of individuals with a familial risk of dyslexia, from birth to adulthood, based on large and representative samples, tapping into all key factors for literacy skills, are necessary to provide data which could decide between our account in terms of different sub-types of dyslexia with different underlying causes and Ramus' (2004) account.

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