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TARGET ARTICLE WITH COMMENTARIES AND RESPONSE

The role of sensorimotor impairments in dyslexia: a multiple case study of dyslexic children

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For commentaries on this article see Bishop (2006), Goswami (2006), Nicolson and Fawcett (2006) and Tallal (2006).

Abstract

This study attempts to investigate the role of sensorimotor impairments in the reading disability that characterizes dyslexia. Twenty-three children with dyslexia were compared to 22 control children, matched for age and non-verbal intelligence, on tasks assessing literacy as well as phonological, visual, auditory and motor abilities. The dyslexic group as a whole were significantly impaired on phonological, but not sensorimotor, tasks. Analysis of individual data suggests that the most common impairments were on phonological and visual stress tasks and the vast majority of dyslexics had one of these two impairments. Furthermore, phonological skill was able to account for variation in literacy skill, to the exclusion of all sensorimotor factors, while neither auditory nor motor skill predicted any variance in phonological skill. Visual stress seems to account for a small proportion of dyslexics, independently of the commonly reported phonological deficit. However, there is little evidence for a causal role of auditory, motor or other visual impairments.

Introduction

A classical account of the phonological theory of dyslexia assumes that an impairment in the cognitive representation of speech sounds results in dyslexia, as defined by the characteristic discrepancy between reading skills and general cognitive ability (Stanovich, 1988; see Snowling, 2000, for a recent review). Indeed, learning to read involves acquiring a mapping between phonology and orthography, between speech sounds and letter symbols. A phonological deficit would affect the learning of such mapping and hence hinder reading acquisition. The role of phonology in literacy attainment has been well supported in the research literature since Bradley and Bryant's study (1983), in which phonological awareness in preschoolers was found to predict later reading ability, irrespective of IQ. Support for the presence of a phonological deficit in dyslexia comes from numerous studies of dyslexics' poor performance on tasks involving phonological awareness (e.g. Bradley & Bryant, 1978).

However, alternative theories of the cause of dyslexia exist, in particular those that advance lower-level sensorimotor impairments. These alternative theories look to more basic and less specific causes of the reading disability, in the visual system, auditory system and the cerebellum. Each of these theories accepts that a phonological deficit may be present and both the auditory and cerebellar theories attempt to account for it as a secondary impairment. However, they also implicate a range of other impairments.

The magnocellular theory of dyslexia proposes that dyslexia is caused by an impairment in the visual system, stemming specifically from the dysfunction of magnocells in the lateral geniculate nucleus (LGN) (Livingstone, Rosen,

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Drislane & Galaburda, 1991). The visual system is thought to exist in two main divisions at this level, magnocellular and parvocellular, each processing different aspects of incoming visual information. Magnocellular dysfunction produces a deficit in the processing of visual information at low luminance, low spatial frequency and high temporal frequency (Lovegrove, Bowling, Badcock & Blackwood, 1980), while parvocellular dysfunction produces a deficit at high luminance, high spatial frequency and low temporal frequency. A similar impairment has also been proposed in the auditory system; the temporal processing theory of dyslexia (Tallal, 1980) proposes that the reading and spelling difficulties characterizing dyslexia are the result of an auditory impairment, producing a deficit in the processing of rapidly changing auditory stimuli. Lastly, the cerebellum has been put forward as a further site of dysfunction (Nicolson & Fawcett, 1990) producing deficits in motor and timing skills as well as automaticity and balance.

The magnocellular theory has now been extended to account for auditory and motor, as well as visual, impairments (Stein, 2001; Stein & Walsh, 1997). The magnocells of the medial geniculate nucleus (MGN) may also be dysfunctional, producing the auditory impairments, as these cells are thought to process rapidly changing auditory inputs. Both visual and auditory impairments can therefore be seen as dynamic processing deficits. Cerebellar dysfunction would result indirectly from deficient input from the magnocells, as the cerebellum receives strong projections from the magnocellular pathway.

Outstanding questions and the present study

The goal of the present study is to investigate the role of sensorimotor impairments in the causality of dyslexia by addressing each of the current theories of dyslexia discussed above. In order to address this issue of causality, the question 'Do sensorimotor impairments play a causal role in dyslexia?' can be broken down into a number of more simple questions.

Can visual impairments explain literacy impairments?

One possible mechanism by which visual impairments have been suggested to act on literacy skill is through the role of the magnocellular system in controlling eye movements, mediated by its input to the posterior parietal cortex (Stein & Talcott, 1999). An impaired magnocellular system may not correctly control eye movements, leading to binocular dysfunction and visual instability, making it hard to read. However, magnocellular visual dysfunction seems to be often associated with the phonological deficit (Cestnick & Coltheart, 1999; Ramus, Rosen, Dakin, Day, Castellote, White & Frith, 2003) so it is unclear whether it makes an independent contribution to reading difficulties.

Visual stress is another condition with symptoms similar to those reported by Stein and Walsh (1997): perceptual distortions including the movement of letters, blurring, coloured halos and pattern glare (Irlen, 1991; Wilkins, 1995). However, visual stress is not related to magnocellular function (Simmers, Bex, Smith & Wilkins, 2001) and can occur both with dyslexia and independently of it. The role of visual factors in reading disability therefore needs clarifying.

Can auditory impairments explain literacy impairments?

The auditory impairments that have been proposed involve a deficit in the processing of rapidly changing stimuli, also referred to as a temporal processing deficit (Tallal, 1980). Certain phonemic contrasts, such as /ba/ and /da/, differ in formant transitions only in the first 40 milliseconds, so a temporal processing deficit would impair the ability to discriminate between such stimuli. In this way, an auditory impairment has been suggested to cause the phonological deficit and therefore the literacy impairment seen in dyslexia. Although initially attributed only to those dyslexics with oral language problems, this theory has frequently been extended to dyslexics without oral language problems (Farmer & Klein, 1995; Temple, 2002), possibly indicating more wide-ranging language problems (Tallal, 2004). Other auditory theories of dyslexia have also been suggested, for example, an impairment in the perception of speech rhythm (Goswami, Thomson, Richardson, Stainthorp, Hughes, Rosen & Scott, 2002; Muneaux, Ziegler, Truc, Thomson & Goswami, 2004).

A number of studies have failed to replicate findings of an auditory deficit, or have found one only in a subset of dyslexic children (Heath, Hogben & Clark, 1999; Hill, Bailey, Griffiths & Snowling, 1999; Marshall, Snowling & Bailey, 2001; McArthur & Hogben, 2001; for a review, see Ramus, 2003) and others that do find a deficit show it to be unrelated to reading skill once IQ is controlled (Hulslander, Talcott, Witton, DeFries, Pennington, Wadsworth, Willcutt & Olson, 2004). Also, the deficit appears to be more prominent for speech than for non-speech sounds distinguished by the same rapid acoustic feature (Mody, Studdert-Kennedy & Brady, 1997; Rosen & Manganari, 2001). It is therefore unclear whether auditory dysfunction can explain the phonological deficit of children with dyslexia.

Can cerebellar impairments explain literacy impairments?

Cerebellar dysfunction was suggested as a cause of dyslexia from the observation that many dyslexic children were clumsy and had poor motor control (Nicolson & Fawcett, 1990). The proponents of this theory predict that all aspects of cerebellar function would be affected, including motor and timing skills, automaticity and balance. Reading ability would be affected in terms of automaticity while a phonological deficit is thought to emerge through poor articulatory skills (Nicolson, Fawcett & Dean, 2001; Fawcett & Nicolson, 2002). However, empirical evidence suggests that there is no link between articulatory ability and phonological ability or literacy skills (see Ramus, Pidgeon & Frith, 2003, for a discussion of this issue). It should also be noted that the majority of experiments reported in the literature involve only motor tasks and therefore poor performance may not necessarily be attributable to a cerebellar impairment. The extent of these motor difficulties and their putative role in the dyslexic population are still debated (Ramus, Pidgeon & Frith, 2003; Wimmer, Mayringer & Landerl, 1998; Yap & van der Leij, 1994), especially whether cerebellar dysfunction can independently cause reading disability, or whether it co-occurs, with a different cause of reading impairment.

Are sensorimotor impairments found in all dyslexics?

Few studies evaluate individual performance on these tasks. Cornelissen, Richardson, Mason and Fowler (1995) found, at most, eight out of 29 dyslexics who performed outside the control range on a visual magnocellular task. Similarly, Witton, Talcott, Hansen, Richardson, Griffiths, Rees, Stein and Green (1998) detected performance out of the range of controls in approximately 25% of dyslexics on a visual task and 50% on an auditory task while Talcott, Gram, Van Ingelghem, Witton, Stein and Toennessen (2003) found such performance in approximately 25% of dyslexics on an auditory task but none on a visual task. In a recent meta-analysis of studies investigating auditory, visual or motor deficits, Ramus (2003) estimated the prevalence of auditory and visual deficits at 39% and 29% respectively, while motor deficits would seem to affect between 30 and 50% of dyslexic individuals. Furthermore, Ramus, Rosen *et al.* (2003), testing auditory, visual and motor deficits, found a subgroup of dyslexics (with a clear phonological deficit) performing perfectly normally on all the sensorimotor tasks. Any theory that postulates sensorimotor deficits as the cause of the reading difficulties characterizing dyslexia must therefore be questioned.

In summary, this study attempts to elucidate whether a sensorimotor deficit plays a causal role in the aetiology of the reading impairment in dyslexia. The shortfalls of other studies will be addressed by using a wide range of tasks and focusing on individual as well as group performance through a multiple case study design. Performance on sensory and motor tasks is therefore studied within-subject and compared to reading and phonological abilities.

Such an approach has already been followed in our previous study (Ramus, Rosen *et al.*, 2003) conducted with university students with dyslexia. However, these subjects may have been an unrepresentative, high functioning, compensated sample and the study may therefore have underestimated sensorimotor problems. Similarly, it is possible that sensorimotor impairments play a role in development but are undetectable later on in adulthood. The relevance of that study to the whole of the dyslexia research field therefore has to be addressed. The present study is a more stringent test of the theory as it looks at the occurrence of sensorimotor impairments in a more representative and heterogeneous sample of dyslexic children, encompassing a wide range of abilities and typical of a large dyslexia clinic.

Method

Participants

In total, 23 dyslexic and 22 control children took part, aged from 8 to 12 years. All children had a non-verbal IQ of at least 85, as measured by the Raven's Standard Progressive Matrices (Raven, Court & Raven, 1988; raw scores converted to standardized scores by interpolation and extrapolation from percentile scores given) and all control children had a standard reading score of at least 90. The controls were selected from a larger sample to match the dyslexic group on gender ($\chi^2 = 1.793$) and on their range of ages and non-verbal IQs (age $t(43) < 1$; non-verbal IQ $t(43) < 1$) (see Table 1); this sample was not self-selected and no knowledge of literacy levels was available at the time of selection. The dyslexic children had all previously received a diagnosis of dyslexia from a chartered educational psychologist and were mainly recruited through the Dyslexia Institute (DI); all those who fulfilled the above age and ability criteria and whose parents gave permission were included. The remaining dyslexics and the control children were recruited from schools located in the area where the children from the DI lived. The control children whose parents gave permission for participation were screened, and a sample then selected to match the dyslexic group in terms of gender, age range and non-verbal IQ.

The majority of the dyslexic children were referred from the DI and had therefore received the same neuropsychological assessment from the same highly experienced educational psychologist. A classification system (Turner, 1997) had been used to specify the severity of their dyslexic symptoms, based on performance in IQ, reading, spelling and other diagnostic tests, such as digit span and speed of information processing. This system employs a six-point scale, ranging from 'not dyslexic' to 'very severe dyslexia' and all children taking part in this study had been classified on the highest three points of the scale (moderate, severe or very severe). Any children with a suspicion of broader language impairment would have been screened out at diagnosis. Furthermore, sensorimotor measures or direct measures of phonological awareness were not used in the diagnostic process and therefore the children were thought to comprise a representative sample of dyslexics with reading and spelling disability. In the dyslexic group, four children also had diagnoses of dyspraxia, one of Attention Deficit Hyperactivity Disorder (ADHD) and one of both. Such diagnoses are relatively common alongside dyslexia and so, in order to maintain a representative sample, these children were not excluded.[1]

Procedure

Ethical approval for the study was obtained from the Joint UCL/UCLH Committees on the Ethics of Human Research and informed consent to participate was given by both parent and child. Children were tested individually in a quiet room either at their home, at their school or at the Institute of Cognitive Neuroscience, University College London (UK). Testing was divided into three sessions of approximately an hour and every child completed a battery of tasks assessing psychometric, phonological, auditory, visual and motor abilities. For the majority of children, the first session consisted of tests of non-verbal IQ and phonology, the second session of auditory and visual tasks and the last of literacy, motor and visual stress tasks in the order stated. The sensorimotor tests were chosen to reflect those currently in use by the proponents of each theory and on which they have found significant group differences. This allowed direct comparison to be made with previous studies and, therefore, any differences between this and previous results could not be attributed to the use of different experimental measures.

[1] Although one child with ADHD was on medication, his results showed that he was not an outlier on any sensorimotor measure. Poor sensorimotor performance could therefore not be accounted for by the effect of his medication and so he was not excluded.

Literacy tests

Literacy tests included standardized assessments of each child's reading and spelling abilities. The children were tested on the Wide Range Achievement Test (WRAT3; Wilkinson, 1993) to provide a measure of their reading and spelling skills.

Phonology tasks

From here on we use the term 'phonology' or 'phonological' to refer to tasks assessing phonological awareness, short-term memory and rapid automatic naming. The Phonological Assessment Battery (PhAB; Frederickson, Frith & Reason, 1997) was used to assess these skills and was administered according to the test manual, although the alliteration test was excluded due to ceiling effects. The following subtests were therefore administered (trial accuracy was the measure recorded unless otherwise stated).

Rhyme

The child identified which two words out of three ended with the same sound (21 items).

Spoonerisms

The child replaced the first sound of a word with a new sound (10 items) or exchanged the initial sounds of two words (10 items).

Non-word reading

The child read one or two syllable nonsense words aloud (20 items).

Naming speed

The child named each item in a randomized series of 50 pictures of five common objects, or of the digits 1 to 9, and the time taken was recorded (two trials per stimulus type).

Fluency

The child said as many words as possible in a given category: by alliteration, rhyme or semantic (non-phonological) category (two trials per category).

Visual tasks

Following the main proponents of the magnocellular theory (Cornelissen *et al.*, 1995; Hansen, Stein, Orde, Winter & Talcott, 2001; Talcott *et al.*, 2003) we adopted coherent motion detection as our main measure of magnocellular/dorsal visual stream function because, from previous studies, it seems to be more sensitive than the more specific contrast sensitivity tasks. Coherent form detection was the control, static counterpart.

In both visual tasks, which have been used in previous studies of dyslexia, an identical two-alternative forced-choice (2AFC) psychophysical procedure was adopted (for further details see Hansen *et al.*, 2001). Stimuli were viewed binocularly from a distance of 40 cm under mesopic lighting conditions, with participants seated. Participants were instructed to examine the two panels and to make a judgement as to which one contained the coherent signal. The initial value of the coherent signal was set to 75%. The two panels were presented on the screen for 2300 ms, following which time the screen was blanked and the participant had to make a response using the computer keyboard. The coherence level was then adjusted using a weighted (1.5:0.5 dB ratio) 1-up, 1-down adaptive staircase (Kaernbach, 1991) and a new stimulus was presented. On each trial, the panel containing the coherent signal was randomized. In addition, approximately 10% (randomly) of the trials were catch trials, used to exclude participants who could not undertake the task, and were set at a high coherence level (75%). The detection threshold was defined to be the geometric mean of the last 8 of 10 reversal points, and thus low thresholds indicate good performance. Two staircase measurements were conducted for each task, the mean of which was defined to be the threshold estimate, and the order of the tasks was counterbalanced across the children.

Motion coherence

A standard random dot kinematogram (RDK) stimulus was used to determine psychophysical thresholds for coherent motion detection for each participant, as a measure of magnocellular/dorsal stream function. The stimuli consisted of two horizontally adjacent panels of moving dots, each containing 300 white dots (each 1 pixel) of high Michelson contrast (~90%) superimposed on the black background of the computer screen. One panel contained a variable proportion of target dots that moved coherently to either left or right over successive screen refreshes, while the remaining noise dots in the panel moved with the same speed but in a direction that randomly changed between refreshes (Brownian motion). The coherent motion also reversed in direction every 570 ms. The other panel contained only noise dots moving in a Brownian fashion. To prevent tracking of individual dots, the lifetime for each dot was fixed at 85 ms, after which time the dot was regenerated at a random position inside the same panel. The consequence of this finite dot lifetime is to decrease the number of dots moving coherently at any particular point in time, as those dots that expire effectively add to the noise background. Motion coherence values were therefore corrected for this factor.

Form coherence

The form coherence task was a control task tapping parvocellular visual function, designed to be as similar as possible in application to the motion coherence task. As before, two rectangular panels were presented side by side, matched in size and overall luminance to the motion task. Each panel consisted of 600 short, high contrast line elements. In one panel there was a coherent form signal, defined by line elements that were oriented tangentially to imaginary concentric circles within a fixed diameter. The circle itself was always centred in the middle of the panel. Signal coherence was varied by modifying the percentage of aligned elements. At 100% coherence therefore, all line elements within the fixed diameter would be perfectly aligned and the circle would be easy to perceive. As the coherence value was lowered, the proportion of elements that were aligned was correspondingly reduced and the circular form was harder to detect. Elements outside the fixed diameter were orientated randomly, as were those in the other panel.

Visual stress

For purely exploratory purposes, we added another measure to our visual battery: sensitivity to visual stress. Although visual stress has never been advocated as a cause of dyslexia, some of its symptoms (blurring, letter superposition, apparent movement) are similar to those reported for magnocellular dysfunction by its proponents (Stein & Walsh, 1997) and visual stress is thought to impact on reading fluency. It is therefore possible that some children diagnosed as dyslexic might have visual stress. By including a measure of visual stress, we were able to address this hypothesis.

A test involving reading words through different coloured overlays was used to look for the presence of visual stress (Wilkins, 1994; Wilkins, Jeanes, Pumfrey & Laskier, 1996). Children were first familiarized with a page of text, consisting of 15 simple words in a randomized order per line. They then chose the overlay that was clearest and most comfortable to see with from a range of 10 colours, by sequential comparisons of two different colours, each time choosing the best colour. They were also given the option of choosing a double overlay, consisting of their chosen overlay and another of the same or adjacent colour, again by comparison of two different colour combinations. Each child then read aloud for one minute from the page of text they had been familiarized with, first with their chosen overlay, twice without it and then once with it again, and the number of words read correctly was recorded. The percentage increase in reading speed with the overlay over these four trials was calculated, with high scores indicating the presence of visual stress.

Auditory tasks

A large variety of auditory tasks were chosen in order to cover as much theoretical and empirical ground as possible. As a basic auditory task, we chose 2-Hz frequency modulation detection, as it has been found to be highly sensitive in recent dyslexia research. Given the slow modulations, this task does not strictly speaking address Tallal's theory of dyslexia. For this purpose, we included a formant discrimination task, which precisely taps the ability to process this 40-msec-long spectral transition that differs between [b] and [d], that requires rapid temporal processing and that is supposed to be deficient in dyslexics (e.g. Tallal, 2004). Furthermore, we embedded this formant transition in both speech and non-speech sounds, in order to take into account the debate on the possible specificity of the deficit to speech (Mody *et al.*, 1997). Finally, we included additional speech discrimination and categorization tasks, since any auditory theory of dyslexia (and specifically Tallal's, 2004) must assume that an auditory deficit produces an effect on reading acquisition through its disruption of speech perception.

Audiological screening

All participants were required to pass a pure tone screen using a standard clinical audiometer at or better than 25 dB HL at frequencies of 0.5, 1, 2, 4 and 8 kHz, in both ears.

All auditory tasks were run on a laptop computer using special purpose software known as SPA (Speech Pattern Audiometry). For single interval identification tasks, two independent randomly interleaved tracks were run, estimating the stimulus leading to 29% and 71% responses of one of the two possible responses (e.g. 'coat' vs. 'goat'). In the multiple-interval discrimination tasks (e.g. detection of frequency modulation), the tracks were linked in order to emulate standard adaptive procedures. Cumulative Gaussian distributions were fitted to all trials in a particular test (probit analysis), in order to estimate the category boundary (the point on the continuum which results in 50% of each of the two responses) and a measure of function slope. Slopes were converted to units of 'just-noticeable difference' (jnd), the stimulus difference necessary to change from 50% to 75% of a particular label. Thus smaller jnds indicate better performance. Two consecutive tests were run for each task, with the best result recorded, and the order of tasks counterbalanced between subjects. If the function obtained for a test result was not significantly different from chance performance ($p < .1$), it was replaced with the worst result above chance taken from all the children, on the assumption that the threshold was meaningless.

Phonemic categorization

Categorization functions were obtained for two synthetic speech sound continua created with a formant synthesizer. Continua encompassing a contrast in two of the main phonetic features were used: voicing and place. Place contrasts have often been used in previous studies of auditory processing deficits, because they can be cued by highly dynamic spectral transitions, which are meant to be particularly vulnerable to the kinds of deficits in perception proposed by Tallal, among others. The voicing continuum (*coat-goat*) was modified slightly from the 'combined-cue' continuum developed by Hazan and Barrett (2000); the cues present were voice onset time (VOT), and the concomitant changes in the onset frequency and extent of transition in the first formant at vowel onset. VOT varied in 1-ms steps across the 51 stimuli. These stimuli were modelled closely on a particular speaker's tokens, and so sounded quite natural. In contrast, the /ba/-/da/ continuum, varying place of articulation, was highly schematic. The stimuli were based on those specified by Mody *et al.* (1997) but with only the lower two formants and with a monotone fundamental frequency. Only the onset frequency of the second formant (F2), and hence the direction and extent of the formant transition, varied across the 41 stimuli in the continuum. On each trial of the test, participants heard a single stimulus and indicated which they had heard by clicking with a mouse on one of two relevant buttons on the computer screen. The buttons were labelled either with pictures or with 'BA' and 'DA' spelled out in upper case letters. In order to assist in the stability of the phoneme categories, continuum endpoints were randomly interspersed throughout the test on 20% of the trials.

Formant discrimination

The ability of subjects to discriminate second-formant transitions in speech and non-speech sounds was assessed. The /ba/-/da/ stimuli described above served as the speech sounds. In this particular place contrast, it is the rapidly varying second formant transitions that are the primary cue to the distinction. Thus we also tested perception of them as a non-speech analogue. Non-speech isolated-F2 stimuli were obtained simply by outputting from the synthesizer the waveforms from the F2 resonator on their own. A four-interval, two-alternative forced-choice task (4IAX) was used. On each trial, two pairs of stimuli were heard, one pair being identical (/ba/ or its non-speech analogue), the other being different (/ba/ paired with another stimulus on the continuum). The participants were required to indicate which pair of stimuli was different by clicking with a mouse on one of two relevant buttons on the computer screen. The buttons were labelled with two pairs of shapes arranged from left to right as follows: two red circles followed by a red circle and a yellow triangle, and a red circle and a yellow triangle followed by two red circles. Feedback was provided in the form of appropriate pictures (a happy face for correct responses and a sad face for incorrect ones).

Detection of frequency modulation (FM)

Although this task involves much slower auditory variations than would be expected to be impaired from Tallal's theory (Tallal, 1980), it has been assimilated into the multi-modal magnocellular theory (Stein, 2001; Stein & Walsh, 1997) as a dynamic auditory processing task and provides one of the more consistent findings of auditory impairment in the literature. This task was therefore included and stimuli were modelled closely on those used by Talcott, Witton, McLean, Hansen, Rees, Green and Stein (2000). Each trial consisted of a pair of 1-s tones, one of which was a sinusoid of 1 kHz, while the other was frequency modulated. Participants indicated which tone was modulated by clicking on an appropriate graphic, either of a straight line followed by a wavy line or vice-versa. Two modulation frequencies were used, 2 Hz as an experimental and 240 Hz as a control task, with the depth of frequency modulation adaptively varied and with graphical corrective feedback after every trial (as before).

Motor tasks

Previous research on dyslexia led us to consider two broad categories of motor tasks: those involving balance and those involving fine manual skills. None of these tasks are pure tests of cerebellar function, but cerebellar dysfunction would be expected to affect performance on these tasks.

Bead threading

The child was required to thread 15 large beads onto a string as quickly as possible, holding the string in their dominant hand (Fawcett & Nicolson, 1996). This task was performed twice and the completion time was measured, with the best time recorded.

Finger and thumb

The thumbs and index fingers of opposite hands are joined, the lower thumb-finger pair is released and the hands are rotated in opposite directions in order to join them again at the top. The child practised this sequence of movements until they could perform it fluently five times and then repeated it ten times as quickly as possible (Dow & Moruzzi, 1958; Fawcett, Nicolson & Dean, 1996). This task was performed twice and the completion time was measured, with the best time recorded.

Stork balance

The child was required to stand on one foot and place the other foot on the supporting knee, with their hands on their hips (Henderson & Sugden, 1992). The time spent standing on one foot, without moving the other foot from the supporting knee or the hands from the hips, was recorded for up to 20 seconds. This task was conducted twice on each leg, the best performance on each leg was recorded and these times were averaged.

Heel-to-toe

The child walked along a line, placing the heel of one foot against the toe of the other for up to 15 steps (Henderson & Sugden, 1992). The number of steps achieved before placing a foot off the line or away from the toe of the other foot was recorded. Each child performed this task twice and their best performance was recorded.

Results

Independent samples *t*-tests (two-tailed) were used to assess the differences between the groups, unless otherwise stated. As well as group differences, individual differences in performance were studied and outliers with abnormally low performance were identified. To detect the outliers on each task, any control outliers more than 1.65 standard deviations (SDs) below the control mean[2] were removed in order to obtain a better estimate of normal performance, regardless of controls who might have performed abnormally on any one task. The control mean and SD were then recalculated and outliers were defined as those

[2] 1.65 SDs below the mean corresponds to the bottom 5% of a normal distribution.

lying more than 1.65 new SDs below this new control mean (procedure as in Ramus, Rosen *et al.*, 2003). Also, in order to look at performance by modality rather than by task, summary factors accounting for all tasks in a given modality were calculated by averaging *z*-scores (calculated in relation to control performance) for each participant on each group of tasks, giving equal weighting to each task. Positive scores indicate good performance and negative scores indicate poor performance.

Figure 1 shows individual performances for both the control and dyslexic groups for each summary factor. The cut-off (of 1.65 SDs from the control mean) is shown by a broken line and children below this line are outliers and are labelled (although dyslexic non-outliers are labelled on literacy and phonology measures).

Literacy tests

As expected, there were significant differences between the groups for reading ($t(43) = 8.004$, $p < .001$) and spelling ($t(43) = 8.494$, $p < .001$) (see Table 1). Although the controls, as a group, were performing above average on these tasks, it should be noted that their non-verbal IQ was approximately average. Their higher reading ability is possibly due to the influence that the literacy hour[3] has had in recent years that has not been accounted for in the test standardization. As the dyslexics are likely to have received just as much, if not more, instruction and were matched to the controls for age and non-verbal IQ, the groups were compared on all measures rather than comparing the dyslexics to the population norms.

For reading, 22 of the 23 dyslexics were outliers (three control outliers), and for spelling, 17 out of 23 dyslexics were outliers (no control outliers). The non-word reading test from the PhAB also revealed significant group differences ($t(31.7) = 7.075$, $p < .001$) with 18 out of 23 dyslexic outliers (two control outliers). A literacy factor was calculated by combining reading, spelling and non-word reading scores (group difference $t(43) = 10.814$, $p < .001$). Twenty-two of the 23 dyslexics showed deviant performance (four control outliers). Participant 2 was the only dyslexic to lie within the cut-off and was still more than 1 SD below the corrected control mean. He was also noted to have a particularly high IQ score of 119, more than 1 SD above the corrected control mean, indicating that a larger difference existed between literacy and intelligence than this literacy measure revealed. Similarly, the four controls who were literacy outliers all had nvIQs well below 100 and reading scores greater than their nvIQ.

[3] The literacy hour is an educational strategy introduced into schools in the UK in September 1998 and is heavily based on phonics training.

Table 1 *Background and literacy test means (and standard deviations)*

	Control	Dyslexic
Number (M:F)	22 (9:13)	23 (14:9)
Age (months)	123.82 (13.73)	126.04 (15.01)
Non-verbal IQ (Raven's)	102.95 (13.88)	102.13 (13.21)
Reading (WRAT3)***	112.64 (10.57)	85.78 (11.86)
Spelling (WRAT3)***	113.23 (12.94)	84.83 (9.27)
Non-word reading (PhAB)***	114.95 (12.68)	93.39 (6.74)
Literacy factor***	0.00 (1.00)	–3.19 (0.85)

*** $p < .001$.
Test scores are standardized scores; literacy factor scores are averaged *z*-scores.

The correlation between literacy ability and non-verbal IQ was significant for the whole sample ($r = 0.352$, $p = .018$) and within each group (control $r = 0.569$, $p = .006$; dyslexic $r = 0.554$, $p = .006$). All the dyslexic children lay outside the 90% confidence interval for this regression line in the control population (see Figure 2), which provides a post-hoc confirmation that they fulfilled a discrepancy definition of dyslexia. Given this correlation, it was decided that individual variations in non-verbal IQ should be accounted for; each summary factor was subsequently entered as the dependent variable into a regression analysis with non-verbal IQ as the independent variable. Unstandardized residuals for each participant were recorded from this analysis as the corrected summary factor. The literacy factor was therefore recalculated (group difference $t(43) = 10.814$, $p < .001$) and all 23 dyslexics were found to be outliers (see Figure 1a), while only one control was. This control was removed from all further analysis as it could not be assumed that his literacy development was normal.

Phonology

Group differences were found on all but one of the phonological tasks of the PhAB (excluding non-word reading), but not on the non-phonological semantic fluency task, indicating that the dyslexics had significant phonological problems (see Table 2). Group differences were highly significant for the rhyme, spoonerisms, picture naming, digit naming and rhyme fluency tasks ($t(42) > 3.5$, $p \leq .001$). The alliteration fluency task did not produce group differences ($t(42) < 1$), which was unexpected, and is due both to the dyslexic group performing better and the control group performing worse than expected from their general performance level. After combining the scores from these six phonological tasks, and accounting for non-verbal IQ, to make a phonology factor (group difference $t(42) = 5.648$, $p < .001$), 12 of the 23 dyslexic children were found to be outliers (no control outliers) (see Figure 1b).

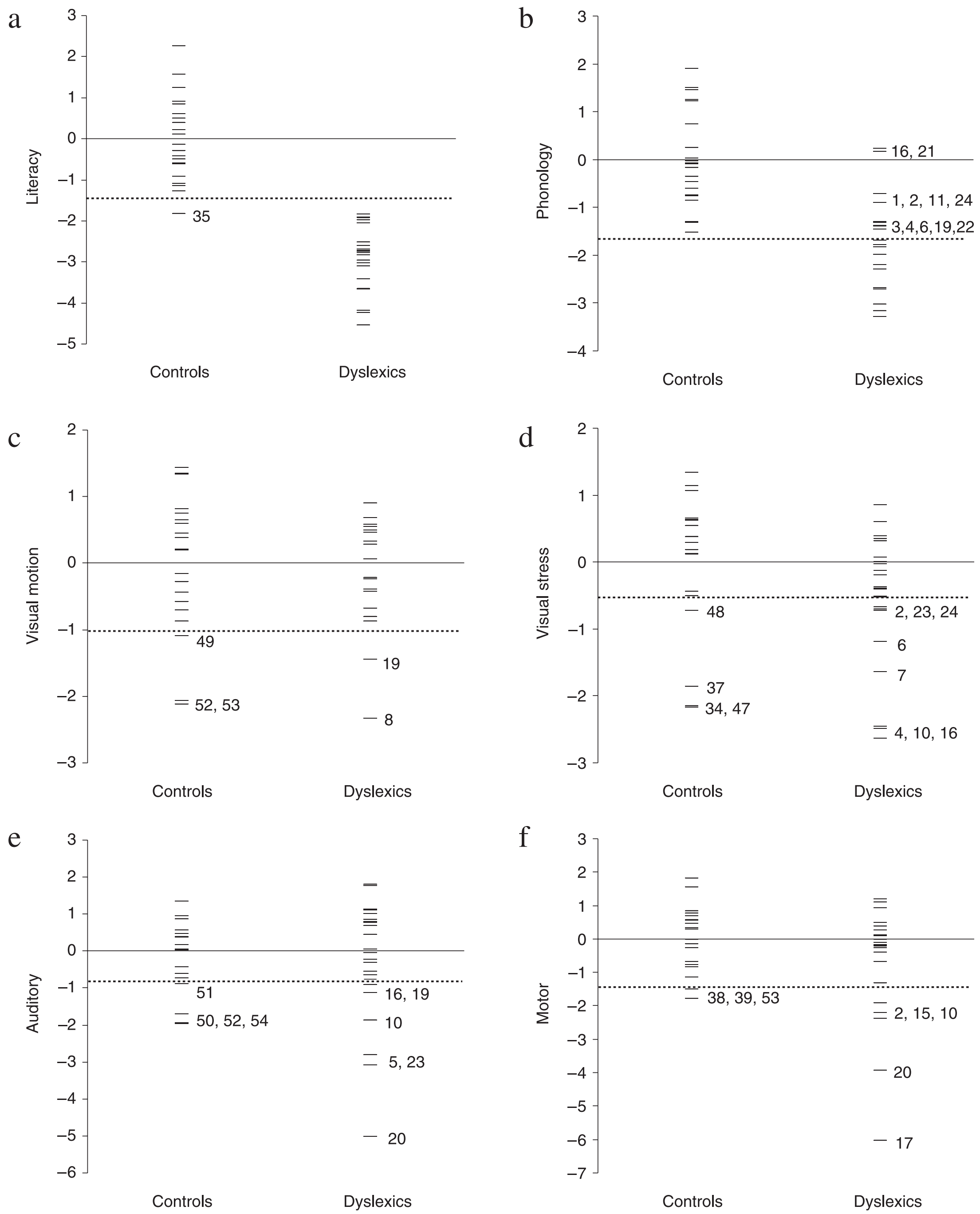

Figure 1 *Graphs of individual performance for each summary factor. All summary factors have nvIQ partialled out, while the sensorimotor factors also have age partialled out. The y-axis values are z-scores, the position of the x-axis indicates the control mean, and the cut-off (of 1.65 SDs below the control mean) is shown by a broken line. Children below this line are outliers and are labelled (although for literacy and phonology factors, dyslexic non-outliers are labelled instead). a = literacy, b = phonology, c = visual motion, d = visual stress, e = auditory, f = motor.*

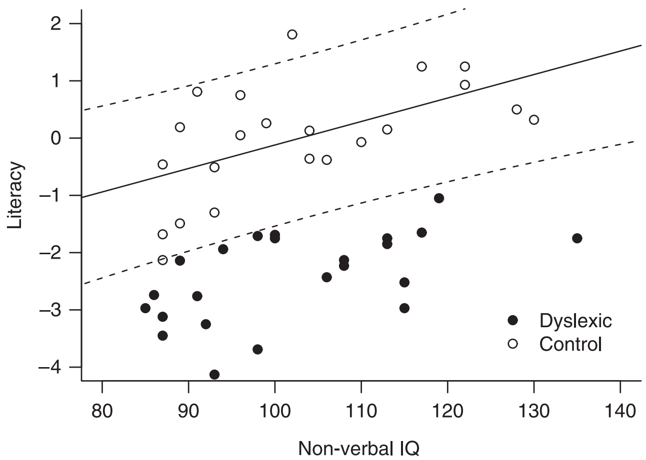

Figure 2 *The relationship between literacy performance and non-verbal IQ. The regression line and 90% confidence limits are based on control performance.*

Table 2 *Phonology test means (and standard deviations)*

	Control	Dyslexic
Rhyme***	112.00 (11.67)	96.26 (14.09)
Spoonerisms***	112.48 (10.90)	98.00 (9.19)
Picture naming***	109.24 (12.80)	92.22 (12.76)
Digit naming***	108.62 (12.54)	88.83 (9.67)
Alliteration fluency	100.10 (7.78)	99.61 (10.66)
Rhyme fluency**	114.38 (11.21)	101.70 (11.93)
Semantic fluency	107.48 (10.92)	100.65 (15.29)
Phonology factor***	0.00 (1.00)	−1.69 (0.99)

*** $p < .001$; ** $p < .01$.
Test scores are standardized scores; phonology factor scores are averaged *z*-scores.

Table 3 *Visual test means (and standard deviations)*

	Control	Dyslexic
Motion	10.18 (4.15)	10.69 (3.40)
Form	26.67 (4.58)	27.92 (7.07)
Visual stress	−1.30 (11.39)	5.19 (11.93)

All group differences are non-significant.
Motion and form scores are coherence thresholds (low scores indicate good performance); visual stress scores are the percentage increase in reading speed with a coloured overlay (positive scores indicate visual stress).

Table 4 *Auditory test means (and standard deviations)*

	Control	Dyslexic
/ba/-/da/	3.98 (4.70)	3.99 (4.73)
coat-goat	3.43 (2.12)	5.05 (5.31)
FM 2 Hz	2.04 (2.17)	2.64 (2.44)
Formant (speech) discrimination	6.36 (3.46)	6.27 (4.88)
Formant (non-speech) discrimination	6.73 (4.14)	6.05 (4.03)
Auditory factor	0.00 (1.00)	−0.30 (1.65)

All group differences are non-significant.
Test scores are quoted as jnds / modulation index for FM tasks, with low scores indicating good performance; auditory factor scores are averaged *z*-scores.

Visual tasks

The majority of children performed all catch trials correctly, while a minority failed only one catch trial. This meant that no results were removed from the analysis, although data for the motion coherence task were lost for one child with dyslexia. The visual tasks were not combined to give a single visual factor as they were believed to probe different visual functions and so individual tasks were considered as factors. After accounting for age as well as non-verbal IQ (as the sensorimotor tasks were not standardized for age, unlike the literacy and phonological tasks), neither the motion nor form coherence tasks produced significant differences between the groups (motion $t(41) < 1$; form $t(42) < 1$) (see Table 3). Two dyslexics were outliers on motion coherence (participants 8, 19) with three control outliers (see Figure 1c), and five on form coherence (participants 6, 7, 16, 17, 20) with two control outliers. The visual stress measure again did not produce a significant group difference, although there was a trend towards the dyslexics increasing their reading speed more with an overlay ($t(43) = 1.843$, $p = .072$). Eight dyslexic outliers were found (subjects 2, 4, 6, 7, 10, 16, 23, 24) with four control outliers (see Figure 1d) and none of these were outliers on the motion coherence task, confirming that visual stress is not accounted for by magnocellular dysfunction (Simmers *et al.*, 2001). Three of the dyslexic visual stress outliers were also outliers on the form coherence task, although there was no group correlation between performance on these two tasks ($r = 0.053$, $p = .733$).

Auditory tasks

The majority of children obtained good Gaussian fits for all tests, while a minority performed at chance (one child on three tasks, two children on two tasks and 11 children on a single task; in total, seven controls and seven dyslexics; of these children, nine were at chance on /ba/-/da/ categorization, two on FM at 2 Hz, four on speech formant discrimination and three on non-speech formant discrimination). In these cases, it was impossible to know whether this was due to poor auditory skill or other factors, although the fact that no child was at chance on all five tasks indicates that this may not be due to non-sensory factors. Again, no significant group differences were found on any task ($t < 1.3$) (see Table 4) but between five and seven outliers were found on each task, with between one and six control outliers. A large number of children reported being unable to hear any difference between the stimuli in the FM at 240 Hz control task due to low intensity, which was reflected in their extremely poor results, and so this task was not included in the analysis. Unexpectedly, the dyslexic group performed slightly better than the control group on the formant discrimination tasks, although not significantly so, and this seems to be due to a subgroup of dyslexics performing out of the range of the controls. If this is not mere noise, then this is reminiscent of Serniclaes, Sprenger-Charolles, Carre and Demonet's (2001) hypothesis that dyslexics have enhanced within-category discrimination.

Performance across the tasks was inconsistent, with different children as outliers on different tasks. In order to further investigate the relationship between performance in the different auditory tasks, we computed all cross-correlations (partialling out age and non-verbal IQ). Tallal's rapid temporal processing theory would predict correlations between all tasks involving rapid transitions (i.e. all but FM at 2 Hz); Talcott *et al.*'s (2000) dynamic processing theory would predict correlations between all tasks; and Mody *et al.*'s (1997) speech-specific theory would predict correlations only between tasks involving speech stimuli (all but FM at 2 Hz and non-speech formant discrimination). We found that only coat/goat categorization and frequency modulation detection at 2 Hz were significantly correlated ($r = 0.57$, $p < .001$) due to three dyslexic outliers on both measures. Marginally significant correlations were found between ba/da and coat/goat categorization ($r = 0.33$, $p = .053$) and between speech and non-speech formant discrimination ($r = 0.30$, $p = .076$). Overall this pattern of correlations does not support any existing theory and does not obviously suggest any other one. This is consistent with previous studies showing that there is no general pattern to dyslexics' auditory deficit, and that the nature of the deficit cannot be accounted for by a rapid temporal processing deficit, nor by a dynamic processing deficit, nor by a speech-specific deficit (Amitay, Ahissar & Nelken, 2002; Ramus, Rosen *et al.*, 2003).

Given that the present data set does not favour any particular theory or method to group the different tasks, we combined all five tasks together to form a general (a-theoretical) auditory factor, again factoring out non-verbal IQ and age (no group difference $t(43) < 1$). Six out of 23 children with dyslexia were found to be outliers (participants 5, 10, 16, 19, 20, 23) with four control outliers (see Figure 1e).

Motor tasks

The data from the bead threading task and both balance tasks were found to have distributions significantly different

Table 5 *Motor test means (and standard deviations)*

	Control	Dyslexic
Bead threading (secs)	56.22 (7.37)	54.50 (13.14)
Finger & thumb (secs)	8.86 (2.97)	8.33 (1.57)
Heel-to-toe* (no. of steps)	15.00 (0.00)	13.57 (3.60)
Stork balance** (secs)	19.36 (2.23)	16.88 (5.20)
Motor factor	0.00 (1.00)	−0.68 (1.68)

** $p < .01$; * $p < .05$.
Low scores in bead threading and finger and thumb indicate good performance; high scores in heel-to-toe and stork balance indicate good performance. Motor factor scores are averaged *z*-scores.

from a normal distribution (Shapiro-Wilk's test) so non-parametric analysis was required (Mann-Whitney U-test). On tests of manual dexterity there were no significant differences between the groups (bead threading $U = 189.5$, $p = .291$; finger and thumb $t(29.7) < 1$) (see Table 5). Three dyslexic children were outliers on the bead threading task (two control outliers), but no outliers were found on finger and thumb (three control outliers). However, differences between groups were significant for the balance tasks (heel-to-toe $U = 199.5$, $p = .048$; stork balance $U = 139.5$, $p = .004$), with four dyslexic outliers on heel-to-toe (no control outliers) and eight on stork balance (two control outliers). A motor factor was produced by combining scores over all these tasks and factoring out age and non-verbal IQ ($t(42) = 1.604$, $p = .116$), and five dyslexic outliers were found (participants 2, 10, 15, 17 and 20) with three control outliers (see Figure 1f).

Subgroups

Figure 3 shows all the children with dyslexia grouped by their deviant performance on the different tasks. In total, 14 dyslexics had a sensorimotor impairment; eight of whom had a single sensory or motor impairment, five had two impairments and one had three impairments. However, when comparing those dyslexics with sensorimotor impairments to those without, no differences were found in literacy performance, either for all those with sensorimotor impairments ($t(19.8) = 1.758$, $p = .094$) or when divided into those with single ($t(15) = 1.931$, $p = .073$) or multiple impairments ($t(5.8) < 1$). This was also true for phonology performance. It should also be noted that nine dyslexics had no sensorimotor impairments whatsoever, and 13 controls had one or more impairment.

In total, 11 dyslexics were not classified as extremely poor performers on the phonology factor, as defined by our criterion of 1.65 SDs below the control mean (i.e. within the bottom 5th percentile). Differences between these dyslexics with better phonology, the remaining

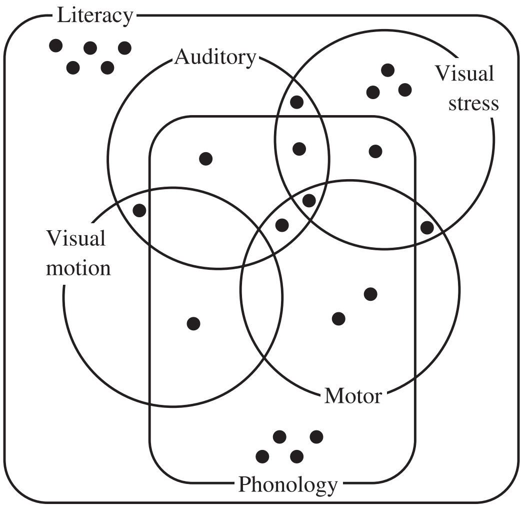

Figure 3 *Classification of dyslexic outliers by literacy, phonology, visual motion, visual stress, auditory and motor impairments. Those included inside each area are outliers whereas those outside an area are not.*

dyslexics with poor phonology and the control group were studied. While the 'poor' phonology dyslexics were significantly worse than controls on all phonology subtests of the PhAB ($t > 4$, $p < .001$; except alliteration fluency), the 'good' phonology dyslexics were still worse than controls on rhyme ($t(30) = 2.162$, $p = .039$), spoonerisms ($t(30) = 2.059$, $p = .048$), picture naming ($t(30) = 2.119$, $p = .043$) and digit naming ($t(30) = 3.485$, $p = .002$) and on the overall phonology factor ($t(30) = 2.627$, $p = .013$). Furthermore, in the 'good' subgroup, there was a correlation between literacy and phonology performance ($r = 0.664$, $p = .026$). This indicates that those dyslexic children who were not outliers on the phonology factor still had significant phonological problems that were related in severity to their literacy problems. No other differences were found between these subgroups. These children can therefore be seen as less severe, better compensated phonological dyslexics.

Correlates of reading and phonological ability

In order to study the extent to which the different impairments were able to predict each other and literacy ability, correlations between summary factors were performed. As can be seen from Figure 4, phonology is a good predictor of literacy skill, as correlations between the literacy and the phonology factor hold both in the whole sample ($r = 0.764$, $p < .001$) and within each of the groups (control $r = 0.451$, $p = .040$; dyslexic $r = 0.595$, $p = .003$). Although there are correlations between the motor factor and both the literacy ($r = 0.384$, $p = .010$) and phonology factors ($r = 0.377$, $p = .012$), these are visibly due to the same two dyslexic outliers with very poor performance in all three domains (literacy and motor $r = 0.196$, $p = .213$; phonology and motor $r = 0.223$, $p = .156$, after removing those two subjects). No other correlations were significant.[4]

A multiple linear step-wise regression was performed to investigate which factors (from phonology, motion, auditory, motor and visual stress) could predict the variance in literacy performance, corrected for age and IQ. The phonology factor accounted for 60% of the variance in literacy performance and none of the other factors were found to be significant predictors of literacy. All children fell within the 95% confidence interval for this regression factor against literacy, apart from one control and one dyslexic child. Literacy skill, in these children, could therefore be explained by phonological skill, to the exclusion of all the other factors.[5]

Discussion

The present findings largely replicate those of Ramus, Rosen *et al.* (2003) by showing that sensory and motor impairments affect only a subset of dyslexics and cannot by themselves explain the phonological deficit and reading disability. Furthermore, as the present study was carried out on a larger and more representative sample of dyslexic children, it alleviates suspicions that our previous results seriously underestimated sensorimotor

[4] Further correlations between IQ- and age-corrected factors, after partialling out variance in reading skill, were performed as a partial replication of the analysis used by Talcott *et al.* (2000). To quote these authors, 'reading ability was removed because the reciprocal nature of the causal relationship between reading and phonological awareness may obscure the relationship between our sensory and phonological processing measures' (p. 2955). Following Talcott *et al.*, a new phonology factor was computed, composed of non-word reading and spoonerisms; however, as an orthographic choice task had not been included in the test battery, this was replaced with spelling as a measure of orthographic knowledge. Unlike the study by Talcott *et al.*, who found partial correlations between phonology and the auditory FM at 2 Hz task and between orthography and motion coherence, no such correlations were found either in the whole sample (phonology and FM at 2 Hz: $r = -0.054$; orthography and motion: $r = 0.077$) or within each group (phonology and FM at 2 Hz: control $r = -0.100$, dyslexic $r = 0.054$; orthography and motion: control $r = 0.162$, dyslexic $r = 0.205$). This indicates that, in our sample at least, phonological skill cannot be predicted by auditory skill, even after variance in reading skill has been removed.

[5] This regression was performed again but with more conservative factors for literacy (reading and spelling) and phonology (rhyme, rhyme fluency, and digit and picture naming), taking into account the possibly circular nature of the relationship between certain phonological awareness and literacy tasks. As before, phonology was the only predictor of literacy skill, accounting for 60% of the variance.

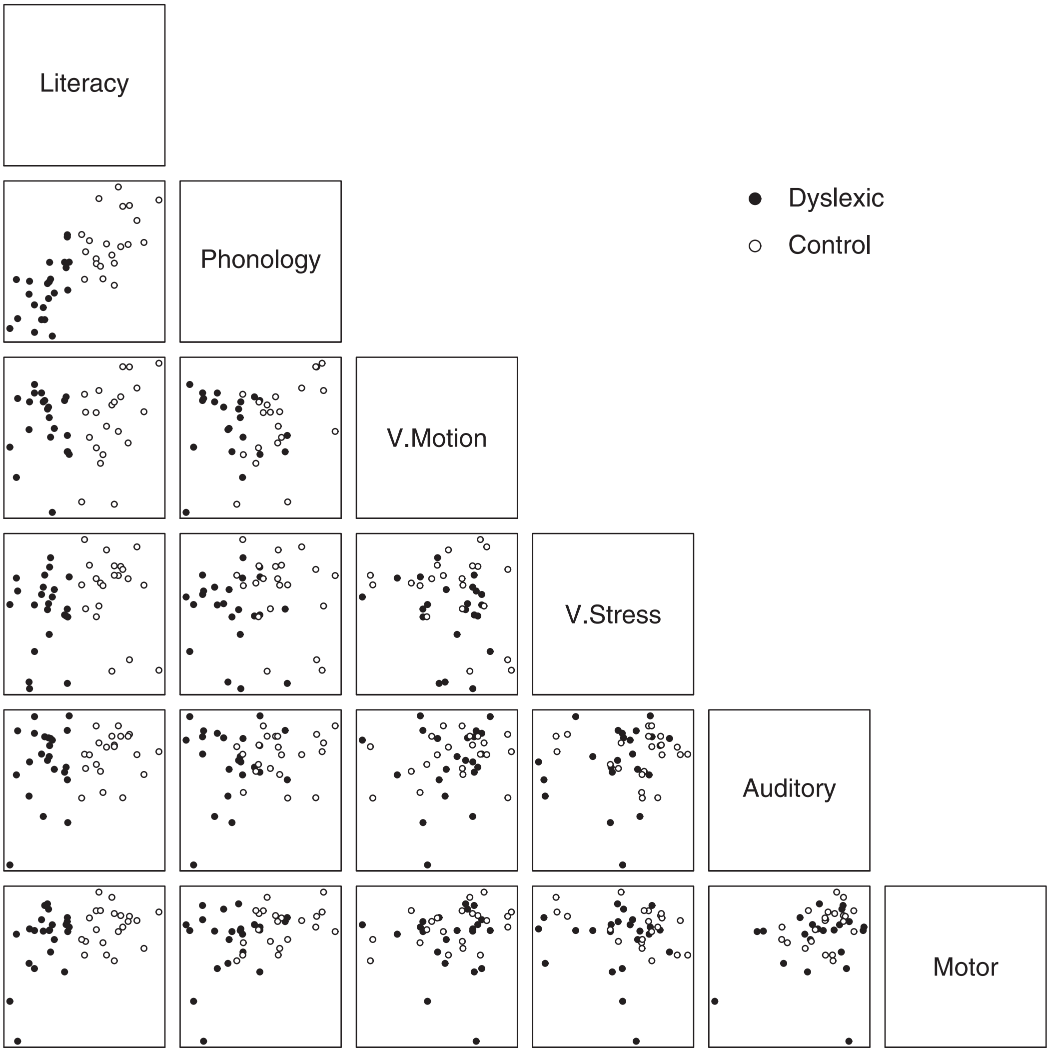

Figure 4 *Summary of correlations between literacy, phonology, visual motion, visual stress, auditory and motor factors. Good scores for each plot are in the top right corner, poor scores in the bottom left. The x-axis is denoted by the label above for each plot, y-axis by the label to the right.*

disorders due to selecting an atypical group of highly-compensated and proficient dyslexic adults.

Can an auditory deficit explain dyslexia?

According to the temporal auditory processing theory (Tallal, 1980) and to the magnocellular theory (Stein, 2001), an auditory deficit is the underlying cause of the phonological deficit, which should predict that all individuals with a phonological deficit have an auditory deficit, and that there is a true correlation between auditory and phonological skills. Our results do not support these predictions. On the contrary, we found that only a minority of children with a clear phonological deficit also had an auditory deficit (four out of 12). Out of these four auditorily impaired children, only one was amongst the six worst phonological performers, so these data do not even support the weaker hypothesis that an auditory deficit might explain at least the most severe phonological cases. More generally, auditory skill was not found to predict phonological skill in any way.

It should be noted that here, speech perception tasks have been lumped indiscriminately into the auditory factor. This does not reflect an *a priori* conception on speech perception in dyslexia but simply the observation that, in our data, speech discrimination and categorization results are similar to those of non-speech discrimination tests: only a few individuals show abnormal performance, with little relationship with the classic phonological deficit. As in our previous study (Ramus, Rosen *et al.*, 2003), our current results therefore do not support the hypothesis that a perceptual deficit, even specific to speech sounds, explains difficulties in phonological awareness, rapid naming and verbal short-term memory, hence the reading impairment.

Can visual deficits explain dyslexia?

We have found no significant relationship between visual measures and reading ability. However, it could be that visual deficits account for some cases of reading disability, independently of the phonological deficit. In the past, this has proved difficult to evaluate, as visual impairments have often been shown to aggregate with the phonological deficit (e.g. Cestnick & Coltheart, 1999). Here, our analysis of individual data highlights six candidates for a possibly visual-based dyslexia without a phonological deficit: five with visual stress and one with poor coherent motion detection. Furthermore, these six cases are the only cases of non-phonological dyslexia with sensorimotor deficits. It is therefore possible that six dyslexic children within this sample of 23 may have their reading disability explained at least partly through visual factors.

Out of these six candidate visual dyslexics, five have visual stress, a disorder that is in fact not promoted as a theory of dyslexia as it affects many non-dyslexics and leads to severe reading retardation in only a few cases (Wilkins, 1995). It should also be noted that it is unrelated to magnocellular dysfunction (Simmers *et al.*, 2001), as confirmed again here. Only two dyslexics in total have high thresholds in coherent motion detection, which is argued to be a sign of magnocellular dysfunction, although this test more generally targets the dorsal visual pathway and is not a unique indicator of magnocellular function. It is also argued to be a cause of poor binocular control (Stein, 2001), although the causal link has not been demonstrated. Overall these data provide little support for the magnocellular theory of dyslexia in general, but some support to the idea that visual dorsal stream dysfunction (with or without a magnocellular origin) may explain reading disability in a small proportion of dyslexics.

Can cerebellar deficits explain dyslexia?

In the cerebellar theory, there are two routes from the cerebellum to reading impairment: one via poor motor/articulatory skill to the phonological deficit, and another via poor automaticity directly to the reading impairment. This therefore predicts relationships between cerebellar measures and both phonological and reading measures. Here, we found no evidence for such relationships. Furthermore, most of the children in our sample showed phonological and literacy impairments without any impairment on the motor tasks. Therefore our data do not generally support the cerebellar theory of dyslexia.

This holds, of course, provided that our tasks suitably sampled cerebellar function. According to proponents of the cerebellar theory, poor performance on balance and manual dexterity tasks are indeed to be taken as good indicators of cerebellar dysfunction (Fawcett *et al.*, 1996). Arguably, we may have underestimated cerebellar dysfunction by not including non-motor cerebellar tests (time estimation, automaticity). However, in our previous work such tests were found to be rather less sensitive than motor tasks (Ramus, Pidgeon & Frith, 2003; Ramus, Rosen *et al.*, 2003). Furthermore, one might also argue that we have overestimated cerebellar dysfunction, to the extent that there are other possible causes of motor impairment.

Beyond the more general claims of the cerebellar theory of dyslexia, individual data analysis can be used to ask whether motor or cerebellar impairment may explain at least some cases of reading disability. Out of five dyslexic children with motor impairments, four also have a phonological impairment. For these children (participants 10, 15, 17 and 20), it is impossible to know whether their phonological deficit is caused by a cerebellar dysfunction, or simply associated with a motor impairment (given that a motor impairment is clearly not necessary for a phonological deficit to arise). As for the other child (participant 2), who was in the normal range of phonological skills (although below average), one might want to consider him as a poor-automaticity dyslexic. However, he also had visual stress and therefore his condition cannot be explained uniquely by the motor factor. Overall, the evidence for a motor/cerebellar role in dyslexia is scant.

Can a phonological deficit arise in the absence of any sensorimotor impairments?

Just as in our previous study (Ramus, Rosen *et al.*, 2003), we find that certain dyslexics (participants 12, 13, 14, 18) can have a phonological deficit without any auditory, visual or motor impairment. This provides further evidence that the phonological deficit need not be secondary to any other deficit, and indeed can be the primary, language-specific, deficit for at least a subset of dyslexics. Given that auditory, visual and motor variables generally fail to explain any significant variance in phonological skill, even within the concerned subgroups, the most parsimonious generalization is that, in all subjects who have a phonological deficit, it is the primary deficit, causally unrelated to sensorimotor disorders.

Of course, it could be that, earlier in their life, these participants had sensorimotor impairments which eventually disappeared in some cases (including the most severe). Given that phonological acquisition occurs largely during the first year of life, only a longitudinal study starting at birth could investigate this possibility. The only one currently available showed that 6-month-olds at risk of dyslexia have, on average, a deficit in categorizing one speech contrast (Leppänen, Richardson, Pihko, Eklund, Guttorm, Aro & Lyytinen, 2002; Richardson, Leppänen, Leiwo & Lyytinen, 2003), but this study did not investigate the underlying basic auditory abilities. Early motor and speech articulation abilities, on the other hand, were found entirely identical between the at-risk and the control groups (Lyytinen, Ahonen, Eklund, Guttorm, Laakso, Leinonen, Leppänen, Lyytinen, Poikkeus, Puolakanaho, Richardson & Viholainen, 2001; Viholainen, Ahonen, Cantell, Lyytinen & Lyytinen, 2002), contrary to the predictions of the cerebellar theory. For magnocellular and auditory processing theories of dyslexia, the hope clearly lies in future longitudinal data which might confirm the hypothesis of early sensory deficits. Meanwhile, one must judge them according to the available data.

Can a phonological deficit explain all cases of dyslexia?

The present study has replicated once more the ubiquitous finding that dyslexics are very significantly impaired on a large array of phonological measures. Analysis of individual data in this sample reveals that 12 out of 23 dyslexics have a phonological deficit beyond the 5th control percentile. This is both far more than any other deficit observed in this study, and small with regard to the presumed universality of the phonological deficit. However, a number of observations can be made. First, it is likely that most dyslexics in this study, having previously been diagnosed by an educational psychologist, have obtained extra assistance, namely specific training on phonological skills and phonics. Consequently, phonological skill is the only ability measured in this study on which most dyslexics are likely to have received extra training as compared to the controls. For this reason we are likely to have significantly underestimated the discrepancy between dyslexics and controls on the phonological measure. It is therefore expected that a number of dyslexics who lie outside the Phonology circle in Figure 3, and who have been considered as non-phonological dyslexics for the purpose of the preceding discussion, should in fact be considered as phonological dyslexics, which further diminishes the role we have found for sensorimotor factors. It can also be noted that those dyslexics who fall within the normal range (above the 5th percentile) on phonological skill still had a significant phonological impairment, which suggests that they may well be less severe or better compensated, but nevertheless the causal relationship between phonology and literacy always holds (unlike that observed with sensorimotor measures).[6]

On the basis of these considerations, we therefore argue that a primary phonological deficit can in fact explain a far greater proportion of the present dyslexic sample than is suggested by the strict discrepancy criterion. Nevertheless, it is unlikely that the phonological deficit can explain 100% of dyslexics. In the present sample, subjects 2, 4, 6, 16, 19 and 24 are the most likely candidates for a non-phonological type of dyslexia, indeed one that is based on visual impairments and, in particular, visual stress. Between the phonological deficit and visual impairment, therefore, the vast majority of dyslexics can be accounted for. Indeed, only five are unexplained who appear to have neither phonological nor sensorimotor impairment; our data are simply insufficient to uncover the precise origin of their reading impairment.

Consistency with previous studies

Considering that this study finds no significant group difference on any of the sensorimotor measures (except balance), and no significant correlation between any of the sensorimotor measures and phonology or literacy, it can be thought to be at odds with many previously published studies which have reported such significant effects. Could it be that we lacked statistical power to detect the effects? This is most unlikely, since we chose our tasks from those reported to produce the largest effects, and many studies with far fewer subjects have found significant effects. In fact the inconsistency is a far more general fact about the dyslexia literature, as a large number of studies have also failed to find significant sensorimotor impairments in dyslexia (see review in Ramus, 2003). Furthermore, a number of recent studies showing individual data have confirmed that only a restricted subset of dyslexics have sensorimotor disorders; this is true in the auditory domain (Griffiths, Hill, Bailey & Snowling, 2003; Muneaux *et al.*,

[6] We acknowledge the reciprocal nature of the relationship between reading skill and phonological awareness, which has been discussed many times in the literature (see Castles & Coltheart, 2004, for a recent discussion). However, the well-established influence of reading on phonological awareness has never disproved the influence of PA on reading, which is supported by considerable converging evidence. Furthermore our measures of phonological skills do not reduce solely to phonological awareness; they also include verbal short-term memory and rapid lexical retrieval, which are not reciprocally related to reading. See footnote 4 for an analysis taking the possible circularity into account.

2004), visual domain (Birch & Chase, 2004; Schulte-Korne, Bartling, Deimel & Remschmidt, 2004; Sperling, Lu, Manis & Seidenberg, 2003; Wilmer, Richardson, Chen & Stein, 2004) and motor domain (McPhillips & Sheehy, 2004) (see Roach, Edwards & Hogben, 2004, for a recent interpretation of these findings).

If one accepts our estimate that auditory, visual and motor impairments each affect between 30 and 50% of dyslexics (Ramus, 2003), it is likely indeed that some studies are bound to find significant group differences and correlations while others not. Across studies, the significance of statistical tests will vary according to the number of subjects, recruitment biases and simply chance. In many studies, children are recruited through clinics or special needs schools (for instance, our earlier study where we found significant motor impairments in dyslexic children; Ramus, Pidgeon & Frith, 2003). For good reasons, these institutions may tend to attract the most severe cases of dyslexia, including cases with multiple cognitive deficits and comorbid disorders. In the present study, most of the dyslexics were schooled in mainstream institutions, which may on the contrary constitute a bias toward 'purer' dyslexics. At any rate, for our present argument it does not really matter whether significant group differences are found or not. What matters is the reliability of the observation that, across all studies reporting individual data, sensorimotor impairments are always found in a minority of dyslexics, regardless of sample size and recruitment bias.

Furthermore, it could be argued that the present study fails to take into consideration developmental processes by not including a reading-age control group (Goswami, 2003); but this must be judged according to the hypotheses being tested. Here we are evaluating the presence of sensory and motor disorders in a group of dyslexic children. A reading-age matched group would allow us to control for the possibility that a reading deficit can have negative effects on the development of perceptual and motor abilities. Certainly this possibility cannot be overlooked, especially regarding the influence of phonology on auditory perception, and of reading skills on visual perception. We do not believe that such effects could explain *all* the sensorimotor deficits, but if they did occur, then this would mean that the incidence of sensorimotor deficits is being inflated in the present comparison. Indeed, reading-age matched controls, being younger children, would inevitably have worse, or equal (but certainly not better) performance on the sensorimotor tasks than the age-matched controls. This would make the dyslexic group even *less* deviant (probably indistinguishable) on sensorimotor measures with respect to that reading-age control group. This is indeed the pattern observed in studies of auditory processing including a reading-age control group, where the dyslexic group differs significantly in auditory skill only with the chronological-age control group, not with the reading-age control group (Goswami *et al.*, 2002; Muneaux *et al.*, 2004). Quite wisely, these authors have refrained from commenting on the absence of the latter difference (as they wanted to provide evidence *for* an auditory deficit). Pushing this wisdom to its logical conclusion, we have refrained from including a reading-age control group. To summarize, our comparison with a chronological-age control group can only overestimate the incidence of sensorimotor disorders, and is therefore conservative with respect to our conclusions. If we were to perform a comparison with a reading-age control group, this could only reinforce our conclusion (perhaps spuriously) that sensorimotor deficits affect only a small subset of this group of dyslexic children.

Clinical implications

There is no point training the auditory abilities of children who have no auditory deficit, the binocular control of children who have no visual impairment, the balance of children who have no balance problem, and the phonological skills of children who have no phonological deficit. It is therefore high time that putative treatments for dyslexia focus on impairments actually observed in particular individuals, rather than claim to cure all dyslexics indiscriminately. Furthermore, attention must be paid to which of the impairments are likely to be the cause of the reading disability, and which are likely to be simply associated. Our interpretation of the present study and of the dyslexia literature in general suggests that phonological treatment should be directed to the majority of dyslexics who have a clear phonological deficit, while visual treatments should be directed to the minority of dyslexics who do have visual deficits (and the visual treatment should be appropriate to the particular type of visual deficit, since there may be several). On the other hand, from the study of auditory and motor impairments in dyslexia, we find little reason to expect that auditory and motor treatments would have any beneficial effects on reading, other than placebo and non-specific effects. And indeed the efficacy of such treatments remains to be proven (Agnew, Dorn & Eden, 2004; Gillam, Froeme Loeb & Friel-Patti, 2001; Hook, Macaruso & Jones, 2001; Snowling & Hulme, 2003; Stein, 2003).

Conclusion

There appear to be two broad classes of impairments that can lead to specific reading disability: visual and phonological. In a small proportion of cases, dyslexia may be explained by visual impairment, specifically visual stress. In the majority of dyslexics, the reading impairment seems to be directly and exclusively due to a specifically linguistic phonological deficit, which cannot be accounted for by auditory or motor impairments. Furthermore, there is an undeniable association between phonological dyslexia and a sensorimotor syndrome including auditory, visual and motor disorders, which certainly points at some common underlying biological factor (Ramus, 2002, 2004), but does not directly explain the reading disability.

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