The relationship between motor control and phonology in dyslexic children

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Running head: Motor control and phonology in dyslexic children.

Abstract

Background: The goal of this study was to investigate the automaticity/cerebellar theory of dyslexia. We tested phonological skills and cerebellar function in a group of dyslexic 8-12 year old children and their matched controls. Tests administered included the Phonological Assessment Battery, postural stability, bead threading, finger to thumb and time estimation. **Results:** Dyslexic children were found to be significantly poorer than the controls at all tasks but time estimation. About 75% of dyslexics were more than one standard deviation below controls in phonological ability, and 50% were similarly impaired in motor skills. However, at least part of the discrepancy in motor skills was due to dyslexic individuals who had additional disorders (ADHD and/or DCD). The absence of evidence for a time estimation deficit also casts doubt on the cerebellar origin of the motor deficiency. About half the dyslexic children didn't have any motor problem, and there was no evidence for a causal relationship between motor skills on the one hand and phonological and reading skills on the other. **Conclusion:** This study provides partial support for the presence of motor problems in dyslexic children, but does not support the hypothesis that a cerebellar dysfunction is the cause of their phonological and reading impairment. **Keywords:** dyslexia, reading, phonology, cerebellum, automaticity, motor control.

Introduction

Over the last twenty years, researchers have converged on the idea that developmental dyslexia (henceforth, dyslexia) results from a specific impairment of phonological representations (Bradley & Bryant, 1983; Snowling, 1987; Snowling, 2000; Vellutino, 1979). According to this "phonological" theory (see Figure 1), a dysfunction in some peri-sylvian areas of the left hemisphere of dyslexic individuals is the cause of a deficit of phonological representations or processes. In alphabetic systems where orthographic rules are not entirely consistent, this provokes difficulty with the learning of grapheme-phoneme rules, hence with reading. The phonological deficit is also manifested in many tasks involving speech sounds.

Figure 1 here

However, parallel work has emphasised that dyslexics may have more general deficits in the auditory, visual and motor domains. This work gave rise to a temporal auditory processing theory of dyslexia (Tallal, Miller, & Fitch, 1993), a visual/magnocellular theory (Lovegrove, Bwoling, Badcock, & Blackwood, 1980; Stein & Walsh, 1997), and an automaticity/cerebellar theory (Nicolson & Fawcett, 1990), respectively. Those three theories are compatible with the idea that a phonological deficit is a direct cause of dyslexia, but challenge the specificity of such a deficit. More precisely, it is hypothesised that the phonological defect itself results from a more general auditory impairment (Tallal et al., 1993) or from a motor (articulatory) dysfunction (Nicolson, Fawcett, & Dean, 2001); the visual deficit is also proposed as an additional source of reading difficulties (Stein & Walsh, 1997). The present paper will focus on the cerebellar theory of dyslexia, attempting to replicate the finding that dyslexic children are impaired on a range of tasks designed to tap the cerebellum, and to provide more insight on the relationship between cerebellar function, phonology and reading. We begin with a brief review of the cerebellar theory of dyslexia.

The cerebellar theory of dyslexia: review and questions

The cerebellar theory (Figure 2) is primarily based on the observation that dyslexic children often show a range of motor impairments, mostly manifested in overall clumsiness, poor manual dexterity, balance and coordination (Denckla, 1985; Haslum, 1989; Wolff, Cohen, & Drake, 1984). Nicolson and Fawcett (1990) proposed that such motor impairments might be the result of mild cerebellar dysfunction, and set out to test cerebellar function more comprehensively in dyslexic children. They showed that dyslexics were significantly impaired on a large number of motor tasks of a cerebellar battery. Depending on the task, between 40 and 100% of dyslexic children had a performance more than 1 SD below that of a same-age control group (Fawcett, Nicolson, & Dean, 1996; Nicolson & Fawcett, 1995). One study provided evidence for cerebellar impairment outside the motor domain: dyslexics were shown to be poorer at a time estimation task (Nicolson, Fawcett, & Dean, 1995), assumed to tap the timing functions of the cerebellum (Ivry & Keele, 1989).

Figure 2 here

Another key concept proposed is that of automaticity, the ability to learn and perform a task automatically, without conscious monitoring (like cycling or driving). The hypothesis is that automaticity crucially depends on the cerebellum, and that its impairment in dyslexic individuals may play a role in the aetiology of reading difficulties, as well as of more general learning problems. Evidence for poor automaticity in dyslexic children was provided using a dual-task paradigm (Nicolson & Fawcett, 1990), as well as various learning tasks (Nicolson & Fawcett, 1994; Nicolson & Fawcett, 2000).

Brain imaging studies also provide some insight into dyslexics' cerebellum. Anatomical studies found metabolic abnormalities (Rae et al., 1998), decreased grey matter density (Brown et al., 2001; Eliez et al., 2000), and excessive asymmetry (Leonard et al., 2001). Functional imaging showed reduced activation in a motor sequencing task (Nicolson et al., 1999), in a reading task (Brunswick, McCrory, Price, Frith, & Frith, 1999) and in a word and non-word repetition task (McCrory, Frith, Brunswick, & Price, 2000). Of course, all these studies also found differences between controls and dyslexics in many other areas besides the cerebellum.

Unfortunately, few research groups have attempted to replicate Nicolson and Fawcett's basic findings. This implies that the support for their theory is highly dependent on the particular cohort of dyslexic children that they have followed over the years in Sheffield. One study reported a partial replication, using a balance/dual task (Yap & van der Leij, 1994). But another one reported a failure to replicate using the very same task (Wimmer, Mayringer, & Landerl, 1998), and subsequent attempts at finding motor/automaticity impairments in dyslexic children were unsuccessful (van Daal & van der Leij, 1999; Kronbichler, Hutzler, & Wimmer, in press). Findings of poor time estimation in dyslexics were also found hard to replicate (Stringer & Stanovich, 1998). Finally, a study investigating implicit learning of sequences found that dyslexics showed automatic learning to the same extent as controls (Kelly, Griffiths, & Frith, in press). It is quite clear that more studies are needed in order to objectively assess the reality of a cerebellar impairment in dyslexia. This is the first goal of the present study.

Wimmer et al. (1998) suggested that the reason for the inconsistency between studies might have to do with the presence of attention deficit/hyperactivity disorder (ADHD) in some dyslexic children. Indeed, there is evidence for motor problems in ADHD, and there is a high degree of co-morbidity between dyslexia and ADHD. One study of a representative epidemiological sample of 409 children took into consideration 42 children scoring below the 10^{th} percentile in reading comprehension: 6 of them (14%) were also classified as ADHD and 12 others (29%) were classified as sub-threshold ADHD (Kadesjö & Gillberg, 2001). In another study of a more clinically defined group of 162 children with learning/attention problems, 71 (44%) had a loosely-defined reading disorder, 30 of which (42%) were also classified as ADHD (Kaplan, Wilson, Dewey, & Crawford, 1998). Wimmer et al. (1998) and van Daal and van der Leij (1999) hypothesised that automaticity impairments might be found only in such co-morbid individuals, implying that they were in significant proportion within the samples studied by Nicolson and Fawcett (1990) and Yap and van der Leij (1994), whereas they had been screened out in their own respective studies. This hypothesis was supported by a new study showing balance problems only in dyslexic/ADHD co-morbid children, not in pure dyslexics (Wimmer, Mayringer, & Raberger, 1999).

These results echo similar earlier findings on manual dexterity tasks (Denckla, Rudel, Chapman, & Krieger, 1985).
In the same line of argument, it should be noted that developmental co-ordination disorder (DCD; see DSM-IV, 1994, p. 56) is also co-morbid with both dyslexia and ADHD. Indeed, Kadesjö and Gillberg (2001) found that 47% of their ADHD children also had DCD, and Kaplan et al. (1998) found that 69% of their ADHD and 63% of their dyslexic children also had DCD. Since the cerebellar tasks used on dyslexic children are in the motor domain, it seems even more likely that co-morbidity of dyslexia with DCD might contribute to group differences in those tasks. Therefore, a second goal of this study will be to ask whether poor performance in cerebellar tasks is due to co-morbidity.

Finally, assuming that findings of a cerebellar dysfunction in dyslexia are reliable, the question remains of whether such a defect can explain the reading impairment. A recent review of the cerebellar theory offered an explicit causal model of the involvement of the cerebellum in dyslexia (Nicolson et al., 2001). Between the cerebellum and the various manifestations of dyslexia, three distinct causal pathways are hypothesised: 1) a general motor skill impairment would directly affect writing; 2) its manifestation in speech articulation would affect phonological skills, hence reading; 3) an automaticity impairment would make the acquisition of visual word forms more difficult, which would have consequences both in reading and spelling (see Figure 2). The third goal of the present study is to assess these hypothetical causal links in the light of the results obtained.

Method

Participants

Twenty-two children were recruited from a special school for dyslexic children and a centre of the Dyslexia Institute on the basis of a previous formal diagnosis of developmental dyslexia by an educational psychologist. Additional inclusion criteria were that they be aged between 8 and 12 years old, have a full-scale IQ greater than 80, reading and spelling scores below 110, and have no reported basic auditory dysfunction. Out of those 22 children, 7 had an additional diagnosis of ADHD, 1 a diagnosis of DCD, and 2 both ADHD and DCD, as documented in their institution's files, on the basis of standard tests such as Conner’s questionnaire and the Movement ABC (Note that these tests were not necessarily performed systematically in every child). Individuals with co-morbid ADHD and DCD were indeed actively sought alongside "pure" dyslexics, in order to address the co-morbidity hypothesis mentioned above.

Twenty-two control children between 8 and 12 years old were recruited from a mainstream school with pupils of similar socio-economic status. Post-hoc examination of their psychometric scores helped ensure that they were matched with the dyslexic group in terms of age, performance IQ, and that they had no particular reading or learning disability. Two participants had to be excluded from the study, thereby reducing the cohort to 20: one had a performance IQ of 147 and was impossible to match with any of the dyslexic children; another one had signs of mental retardation (FSIQ: 57).

It should be noted that we did not recruit a reading-age control group. Cerebellar tests tap neurological and morphological maturation and therefore only require a chronological-age control group. Phonological tests, which would normally require a reading-age control group, were drawn from a standardised battery, making it possible to compare standard scores between children of different reading abilities but of similar general ability.

Procedure

Tests were administered individually by the second author in a quiet conference room at the child's school/institution, except for a few control children who were tested at their home. Dyslexic children, who already had IQ scores, were tested in one single session. Control subjects were tested in two separate sessions, one for the WISC, and one for the other tests.

Psychometric measures

Intelligence was measured using the WISC-III^{UK} (Wechsler, 1992) and reading and spelling ability was measured using the WRAT3 (Wilkinson, 1993). All dyslexic children had been administered the WISC by the school/institution less than one year before the study, and most of them also had recent WRAT scores available; those scores were therefore copied without re-testing. For dyslexic children who did not have recent WRAT scores and for all control children, the necessary tests were administered at the time of the study.

Phonological Assessment Battery

Except for a few dyslexics who already had recent scores available, all children were administered the Phonological Assessment Battery (PhAB) (Frederickson, Frith, & Reason, 1997). The battery includes the following tests (all preceded with appropriate instructions and practice trials):

Alliteration: the child is told three words (e.g., ship fat fox) and must say aloud the two words that start with the same phoneme (10 items).

Rhyme: same as alliteration but the two words share the same rhyme (e.g., made hide fade) (21 items).

Naming speed: time to name 50 pictures (2 measures) and 50 digits (2 measures on different orderings of the pictures).

Spoonerisms: in the first part of the test the child is asked to substitute the initial phoneme of a word with another phoneme (cot with a /g/ gives got) (10 items). In the second part the child must swap the initial phonemes of two words (sad cat gives cad sat) (10 items).

Alliteration fluency: the child is asked to say as many words as possible that begin with a given phoneme (time limit: 30 sec; 2 measures with different phonemes).

Rhyme fluency: same as alliteration fluency, but with words that have a given rhyme.

Semantic fluency: same as alliteration fluency, but with words that share a given semantic property (things to eat; animals). This is meant to be a control tasks for dyslexic children, as it does not particularly tap phonological abilities.

Non-word reading (20 items).

Cerebellar tests

All children were administered the 4 following tests: finger to thumb, bead threading, postural stability and time estimation, assumed to tap cerebellar function and directly taken from Nicolson and Fawcett's studies. Obviously, poor performance on any one

of these tasks might result from other dysfunctions than cerebellar. They were nevertheless highlighted as cerebellar indicators in previous work on the cerebellum (Dow & Moruzzi, 1958; Ivry & Keele, 1989). We suppose that joint poor performance on all these tasks would be the best indicator of an underlying cerebellar dysfunction.

Finger to thumb: This test, drawn from the Dow and Moruzzi (1958) battery, was administered as in Fawcett et al. (1996). Subjects place the index finger of one hand onto the thumb of the other hand and vice-versa. Then, keeping the top thumb and finger together, they rotate one hand clockwise and the other anti-clockwise until the finger and thumb touch again, and so on. The task is demonstrated and subjects train until they complete the movement fluently 5 times. They are then asked to perform 10 such movements as fast as possible. The measure is the time taken for 10 movements.

Bead threading: Subjects are provided with a string and 15 wooden beads from the Dyslexia Screening Test (DST) (Fawcett & Nicolson, 1996). After demonstrating the task, subjects are asked to thread the 15 beads as fast as possible, holding the string in the dominant hand. The task was administered as in Fawcett and Nicolson (1995), but here the measure is the time taken to thread the 15 beads, rather than the number of beads threaded in 1 minute (to avoid ceiling effects).

Postural stability: This is another test drawn from Dow and Moruzzi (1958) and Fawcett et al. (1996). Subjects are asked to stand up straight, blindfolded, with feet together and arms alongside. They are pushed in the lower back (opposite the navel) and must try to stay as still as they can. Pushing is performed using the balance tester from the DST (Fawcett & Nicolson, 1996): it consists of a collar sliding along a shaft, with the friction of the collar generating a constant force at the extremity of the shaft for about 2 seconds. The friction was adjusted on a kitchen scale before each test, so that the force was 2.5 kg. There were three trials per child, administered and scored as specified in the DST manual from 0 to 6: 0 rock solid, 1 slight sway, 2 rises up on to toes, 3 small step forward/marked sway, 4 marked step forward, 5 two controlled steps forward, 6 several steps or marked loss of balance.

Time estimation: This task is identical to that used by Nicolson et al. (1995), which was itself inspired by Ivry and Keele (1989). It is meant to tap the timing functions of the cerebellum. In each trial, two tones are presented successively, and the task is to say whether the second one is longer or shorter than the first one. The standard stimulus, always presented first, is a 1200 ms-long pure tone of frequency 392 Hz. Twenty-two comparison tones had respective durations of 400, 700, 800, 900, 950, 1000, 1050, 1100, 1140, 1160, 1180, 1220, 1240, 1260, 1300, 1350, 1400, 1450, 1500, 1600, 1700 and 2000 ms. The two tones were separated by a 1000-ms silence interval. Each trial was repeated three times, therefore amounting to 66 test trials, presented in a random order. The test block was preceded by a practice block of 8 trials (using only the 8 extreme comparison tones), during which feedback was provided. No feedback was provided during the test block. The experiment was programmed on a PC using E-Prime 1.0 and the stimuli were presented through headphones at about 75 dB SPL. After each pair of sounds, children had to say "shorter" or "longer" and the response was entered on the keyboard for them. Following Nicolson et al. (1995), we also included loudness estimation as a non-cerebellar control task. This experiment follows exactly the same design as time

estimation, except that all tones are 1000 Hz and 1000-ms long and differ only in loudness. Comparison tones had respective amplitudes 4%, 8%, 12%, 16%, 20%, 26%, 32%, 38%, 46%, 56% and 70% greater or smaller than the standard stimulus. The calibrated sound pressure level was around 67 dB for the standard tone. Children had to say whether the second tone was louder or softer than the first one.

Results

Psychometric tests

Table 1 summarises the data obtained. An ANOVA was run to assess between-group differences. The two groups were found to be very well matched in age, full-scale IQ and performance IQ ($F(1,41)<1$). There was a very slight trend for the dyslexics to have a higher verbal IQ than the controls ($F(1,41)=1.4$, $p=.24$). Although this trend is not statistically significant, it may seem surprising since in most studies it is in the opposite direction. However, this is not too worrying, since if this trend is real, it can only make any difference that will be found in literacy and phonological skills more convincing.
As expected, the two groups were also found to be very significantly different in their reading ($F(1,41)=72.8$, $p<0.001$) and spelling ($F(1,41)=78.4$, $p<0.001$) scores, with almost no overlap at all.

Table 1 here

To further investigate the issue of verbal IQ, we computed the WISC index scores. These factors summarise clusters of sub-tests that presumably tap common cognitive resources. They are Verbal Comprehension, Perceptual Organisation, Freedom from Distractibility and Processing Speed. Here, we could not compute the latter because a component sub-test (symbol search) had not been administered to all participants. We therefore only compared performance on Coding, which is the other component sub-test for this factor. Scores are summarised in Table 1.

An ANOVA revealed that the two groups were not significantly different in terms of Perceptual Orientation and Coding ($F(1,41)<1$). However, the dyslexics were significantly poorer on the Freedom from Distractibility index ($F(1,41)=8.1$, $p=0.007$), which actually indexes verbal short-term memory (component sub-tests: digit span and arithmetic). This difference therefore reflects the well-known finding that dyslexics are poorer at verbal shorter-term memory, which is the main reason why their verbal IQ is often lower. Here, this happens to be counteracted by the better performance of a number of dyslexics in Verbal Comprehension, as reflected by a non-significant trend ($F(1,41)=3.1$, $p=0.09$). Since Verbal Comprehension sums up 4 out of the 5 verbal IQ sub-tests, this explains why our dyslexics were found to have a slightly higher verbal IQ than controls.

Phonological Assessment Battery

Table 2 summarises the scores obtained by the two groups in each phonological test. The overall picture is that dyslexics have rather severe problems with the phonological tasks. This shows most dramatically in non-word reading, spoonerisms, rhyme detection and alliteration fluency ($F(1,41)>16$ for each variable, $p<0.001$).

Table 2 here

Alliteration detection also demonstrates a significant difference ($F(1,41)=11.7$, $p=0.001$), although this test is limited by the fact that many subjects, including dyslexics, reached the ceiling score. This problem was recognised in the standardisation of the PhAB and standard scores above 100 were therefore not provided for the age range of interest (Frederickson et al., 1997). For this reason, the results of this test will not be used in subsequent analyses.

Picture and digit naming revealed smaller differences between the two groups ($F(1,41)=4$, $p=0.051$ and $F(1,41)=4.4$, $p=0.04$ respectively). In the case of these two variables, statistical significance would not survive a Bonferroni correction for multiple tests. An informal look at the individual data showed that only a subgroup of the dyslexics had slow naming speed, the others performing at normal level.

Rhyme fluency showed only a marginally significant difference ($F(1,41)=3.2$, $p=0.08$ without correction). Finally, semantic fluency did reveal a significant difference between the two groups ($F(1,41)=5$, $p=0.03$), which is more surprising since this task is not supposed to tap phonological abilities, and the dyslexic children otherwise had rather high verbal IQ scores (including vocabulary). However, here again statistical significance would not survive a correction for multiple tests.

Since many of these tests are expected to tap the same underlying cognitive abilities, a good way to get an overall picture of phonological ability is to conduct a factor analysis. The extraction method used was principal component analysis. Two factors were extracted, which we call PhAB1 and PhAB2. PhAB1 accounts for 51.9% of the variance, and PhAB2 for 18.5% of additional variance. Their loadings on the different variables show that PhAB1 amounts to an average of all the phonological variables, whereas PhAB2 opposes the two speeded naming tasks to phonological awareness tasks. An ANOVA on the two factors with Group as independent variable shows that PhAB1 captures most of the variance between the two groups (Group effect: $F(1,40)=36.5$, $p<0.001$), whereas PhAB2 only captures within-group variance ($F(1,40)<1$). For the purpose of further analyses, we will therefore take PhAB1 as the best single factor indexing phonological ability relevant to dyslexia.

PhAB1 appears to discriminate well between the dyslexic and the control group. If we set the threshold for a phonological deficit at one standard deviation below the control mean, then 17 dyslexics out of 22 fall under this criterion, but only 3 controls out of 20. This is even more remarkable since all these dyslexic children are being taught in a specialised institution, where they get specific phonics training. In spite of this training, most dyslexic children still show measurable phonological problems.

Cerebellar tests

For bead threading and finger to thumb, the best performance of the two trials of each task was recorded as the final result. Three dyslexic subjects were unable to perform the finger to thumb task (1 had dyslexia + ADHD, 1 dyslexia + DCD, 1 dyslexia + ADHD + DCD). For the purpose of further analyses, they were assigned the same time as the worst successful performer (who had dyslexia + ADHD; his best performance was 29.6 sec. for 10 finger to thumb oppositions). For postural stability, the median of the three trials was taken as the final result. This is because we felt that on any trial, a subject might be able to resist the push, or on the contrary, might lose

their balance by chance; taking the median therefore allows one to disregard outlying trials.

Table 3 shows the data obtained for the cerebellar tasks. A Mann-Whitney test was used to compare the groups because the distributions were not normal (Shapiro-Wilk's test). Significant differences were found in each motor task (postural stability:$U=134$, $p=0.02$; bead threading: $U=135$, $p=0.032$; finger to thumb:$U=121$, $p=0.013$). A number of dyslexic children are clearly out of the controls' range, although it is quite remarkable that the worst performers at one task are not the worst performers at the other (see correlations below). On the other hand, about half the dyslexic children are within the control range, although not amongst the very best performers.

Table 3 here

For time estimation, the classification function (% of "shorter" responses as a function of the duration of the comparison tone) of each subject was fitted with a logistic function. The parameters of the logistic function were then used to estimate the threshold duration difference at which each subject was 75% correct. The same procedure was followed for loudness estimation with the percentage of "softer" response. The logistic regression revealed that all subjects had slopes significantly different from 0, except for one dyslexic subject, in the time estimation task (whose responses are therefore not different from chance). Thereafter we refer to this subject as MM. Data is missing for three control subjects in time estimation and two of these in loudness estimation. As shown in Table 3, the two groups were not significantly different in either task (time: $U=139$, $p=0.18$; loudness: $U=142.5$, $p=0.13$; again the distributions were not normal).

Most of the controls and dyslexics overlap in the region of low thresholds. However, there are a number of dyslexic outliers. As expected, the most extreme outlier in time estimation is MM, the subject whose slope is not different from 0. His threshold of 916ms is therefore not very meaningful. There is one more outlier, and a few other "borderline" dyslexics in time estimation. Curiously, there are also three dyslexic outliers in loudness estimation, although this task is not supposed to tap the cerebellum. Furthermore, these are not the same subjects who are impaired on time and on loudness estimation, suggesting that outliers cannot simply be explained by an inability to perform psychophysical tasks or by basic auditory dysfunction. However, the high attentional demands of the task may have played a role in the children's willingness to perform conscientiously.

In order to better understand the relationship among the different cerebellar tasks, we ran bivariate correlations between the five of them. The only significant correlations we obtained were: postural stability vs. loudness estimation ($r=0.37$, $p=0.03$, but the significance doesn't hold after a Bonferroni correction); postural stability vs. time estimation ($r=0.49$, $p=0.001$); and bead threading vs. finger to thumb ($r=0.65$, $p<0.001$). Furthermore, the correlation between postural stability and time estimation is due only to subject MM, who is the worst performer in both tasks. When he is removed from the analysis, the correlation totally vanishes ($r=0.11$, $p=0.5$). It is furthermore notable that MM showed very good performance in both bead threading and finger to thumb. The conclusion from this correlation analysis is that bead threading and finger to thumb seem to be tapping the same underlying ability, i.e.,

manual dexterity. However, we find no reliable relationship between manual dexterity, postural stability and time estimation, which goes against the idea that poor performance in all those tasks reflects the same underlying deficit, that is, a cerebellar dysfunction.

We also ran a factor analysis of the cerebellar tasks in order to identify the main sources of variance. Only four factors were entered into the principal component analysis, loudness estimation being discarded since it is not supposed to be a cerebellar task. Two factors were extracted, the first one, Cereb1, accounting for 45.6% of the variance, and the second one, Cereb2, accounting for 35.4% of additional variance. Cereb1 loads on Postural stability, Bead threading and Finger to thumb, thereby indexing motor skills, whereas Cereb 2 loads mainly on Postural stability and Time estimation.

As with the phonological battery, we ran an ANOVA on the two factors with Group as independent variable: it seems that the first factor Cereb1 picks up the variance between groups ($F(1,37)=13.6$, $p=0.001$) (indeed, it loads on the three tasks where there were significant group differences), whereas Cereb2 mainly captures within group variance, and that due to subject MM ($F(1,37)<1$). For further analyses, we will therefore keep Cereb1 as the main index of performance in the cerebellar tasks.

Overall, the motor skills factor Cereb1 discriminates reasonably well between the two groups. If we set the threshold for a motor deficit at one standard deviation above the control mean, then 13 dyslexics out of 22, but only 1 control out of 17, fall under this criterion.

Discussion

These results replicate Fawcett et al.'s (1996) findings that dyslexic children are, on average, significantly impaired on motor control tasks such as postural stability, bead threading and finger to thumb. However, the incidence of such impairments within the dyslexic group is rather lower in the present study than in theirs: for a similar age group (around 10), Fawcett et al. (1996) reported a similarly defined incidence of 7/12 for finger to thumb (9/22 here), and 12/12 for postural stability (11/22 here). With the more powerful factor analysis taking into account impairments across several tasks, the incidence rises to 13/22 (59%) in the present study, still far from the 80% claimed by Nicolson et al. (2001). Furthermore, like Stringer and Stanovich (1998), we failed to replicate Nicolson et al.'s (1995) finding of a time estimation deficit in dyslexics, except for perhaps 2 outliers. Since reliable deficits were found only in the motor domain, it therefore seems more parsimonious to explain them simply through a motor impairment than through a cerebellar dysfunction, which would presumably also impact on time estimation.

One might worry that the poorer performance of dyslexics on motor tasks might be due to the fact that the proportion of boys is higher in the dyslexic group (see Table 1), and boys generally lag girls in motor development. Indeed, the five dyslexics with the worst Cereb1 scores are boys. Nevertheless, 3 dyslexic girls are also out of the normal range. Furthermore, there is no sign of any advantage for girls within the control group, so one would need to explain why a gender lag would be evident only in the dyslexic group. To further examine this possibility, we conducted a univariate ANOVA on Cereb1 with both Gender and Group as factors. Whereas Group came out

as a significant factor ($F(3,38)=9.8$, $p=0.004$), Gender did not ($F(3,38)<1$). Furthermore there was no Group X Gender interaction ($F(3,38)<1$). We therefore conclude that the difference found between dyslexic and control children cannot be explained by the gender imbalance.

The next question to address is whether this motor impairment we found in a little more than half our subjects can be explained by co-morbidity with other developmental disorders. Out of 13 dyslexic children we identified as more than one standard deviation above the control mean in the Cereb1 motor skills factor, 5 are purely dyslexic, 6 also have ADHD, one has DCD and one has DCD + ADHD. Furthermore, the 4 most severe cases on this scale are 2 with ADHD, one with DCD and one with both. Out of the 10 co-morbid children we had in the study, only 2 of them (one ADHD, one ADHD + DCD) fell within the normal range on the Cereb1 scale, whereas 7 pure dyslexics did. To look at it in yet another way, 5 pure dyslexics out of 12 (42%) and 8 co-morbid dyslexics out of 10 (80%) are poorer than controls at motor tasks. Therefore, observation of this limited sample suggests that co-morbid dyslexics are more likely to have a motor impairment than pure cases, and also tend to have the most severe forms of motor impairment. The presence of 5 pure dyslexics with poor motor skills may suggest that motor control is a genuine problem in dyslexia. However, we cannot fully rely on the absence of ADHD or DCD diagnoses in these pupils' files, since it is likely that such additional diagnoses were not performed systematically, but only for the most disruptive or obviously clumsy children. At this stage, it is therefore not possible to say whether Nicolson and Fawcett's findings might be entirely explained by co-morbidity, but the present data suggest that if part of their sample had other co-morbid disorders, this would have increased the observed effects in the motor tasks, both in incidence and in severity.

Finally, we also wanted to address the hypothesis of Nicolson et al. (2001), that a cerebellar deficit can be the underlying cause of dyslexia. More specifically, they proposed 3 routes through which a cerebellar dysfunction might provoke the symptoms of dyslexia. First, a motor skill impairment would impact on writing skills. Unfortunately, we have too little data on our subjects' writing skills to empirically assess this hypothesis. Nevertheless, it is quite plausible that the manual dexterity difficulties we observed would have an effect on these children's writing speed and legibility.

The second route is that a cerebellum-based deficit in articulatory skill would cause the phonological deficit. This predicts a monotonic relationship between performance in the cerebellar tasks and in the phonological tasks. The correlation between our Cereb1 and PhAB1 factors was indeed found to be significant ($r=-0.38$, $p=0.02$). However, correlation does not entail causation. Because the dyslexic group is, on average, poorer at both phonological and motor tasks, a correlation is to be expected even if there is no causal relationship between motor and phonological performance: in that case, it would merely reflect the difference between the two groups. On the other hand, if there is a genuine causal link, then one would expect that the correlation would hold irrespective of group membership. However, this is not the case here: the correlation does not hold within either group (dyslexics: $r=0.08$, $p=0.74$; controls: $r=-0.23$, $p=0.36$). Therefore, phonological ability does not generally decrease along with motor skills.

It could be argued that a fairer test of the hypothesis would be to look at the more direct relationship between articulatory skill and phonological skill. Although we had no specific measure of articulatory skill, rapid automatic naming can be seen as directly dependent on articulatory skill, although it also depends on phonological retrieval. Therefore one might want to look at the relationship between naming speed and other phonological tasks. The factor analysis of the PhAB already seemed to indicate that naming and phonological awareness tasks picked up different sources of variance. To further explore the issue, we conducted two separate factor analyses: one for the two rapid naming tasks, yielding one factor made of the sum of the two scores and accounting for 83.9% of the variance, and one for the phonological awareness tasks (rhyme, spoonerisms, non-word reading, alliteration fluency, rhyme fluency), yielding one factor accounting for 63.4% of the variance and loading almost equally on all the variables. These two factors were found to be significantly correlated ($r=0.41$, $p=0.007$). However, as before, this correlation is spurious and does not hold within group (dyslexics: $r=0.36$, $p=0.1$; controls: $r=0.18$, $p=0.45$). Our data therefore do not support the idea of a causal link between general motor skill, articulatory skill and phonological awareness.

In the third route proposed, the cerebellum-based automaticity deficit directly causes difficulty with the acquisition of visual word forms, thereby impairing reading and spelling. Again, one would then predict a monotonic relationship between cerebellar function and reading and spelling ability. Indeed, both correlations are significant (with reading: $r=-0.36$, $p=0.03$; with spelling: $r=0.36$, $p=0.02$). Again, the correlation with reading appears to be due only to the difference between the two groups, but does not hold within group (dyslexic group: $r=0.16$, $p=0.47$; control group: $r=0.03$, $p=0.91$), and the same is true with spelling (dyslexic group: $r=0.35$, $p=0.11$; control group: $r=-0.35$, $p=0.17$; note that the correlations are in opposite directions between the two groups). So reading ability does not seem to decrease monotonically with motor skills. The point is probably best appreciated when compared with the correlation between the PhAB1 factor and reading. Phonological competence is very significantly correlated with reading ability ($r=0.79$, $p<0.001$), and this does hold within group (dyslexic group: $r=0.5$, $p=0.02$; control group: $r=0.61$, $p=0.004$). So in this case there is a genuine linear relationship between phonological and reading ability, irrespective of children's overall level of ability and group membership.

General Discussion

Beyond the present data, the idea that motor skill is a precursor of phonological and reading skill flies in the face of a larger body of evidence. As pointed out by Ivry and Justus (2001) in their commentary on Nicolson et al. (2001), the cerebellar theory of dyslexia is intimately linked to the motor theory of speech (Liberman, Cooper, Harris, & MacNeilage, 1963). Although we cannot review here all the arguments for and against the motor theory, let us just point out that the causal model proposed by Nicolson et al. (2001) crucially relies on one assumption of the motor theory that is possibly the most controversial and widely criticised (Fourcin, 1975b), and was subsequently abandoned in a revision of the theory (Liberman & Mattingly, 1985): it is the assumption that the acquisition of internal representations of speech (i.e., phonology) relies on explicit speech articulation. The first piece of counter-evidence was provided by a famous patient studied by Lenneberg (1962) and Fourcin (1975a). This patient suffered from a severe congenital dysarthria which made his speech almost totally unintelligible. Nevertheless, language comprehension developed

normally in this child, implying that he had acquired the ability to adequately process the sounds of English. Furthermore, literacy was also acquired without problem. More evidence of an absence of impact of articulatory skill on language comprehension, reading and spelling was subsequently reported (Cromer, 1980; Hatfield & Walton, 1975). A more recent case of a congenitally speechless child (oral apraxia) was reported by Cossu (in press), who showed that, in addition to normal reading and writing skills, the child also had normal verbal memory and meta-phonological skills. In the light of this evidence, it appears unlikely that an articulatory deficit, whether of a cerebellar origin or not, might have any notable effect on the phonology and reading of dyslexic children.

As Fawcett et al. (1996) reported, a significant proportion of dyslexic children perform poorly in a variety of motor tasks. However, this effect is at least partly due to co-morbidity with other developmental disorders, such as ADHD and DCD. Furthermore, we failed to find a significant time estimation deficit in dyslexic children, even in those who had a motor deficit. This casts doubt on the hypothesis that their poor motor performance has a cerebellar origin. A significant proportion of dyslexic children have absolutely normal motor skills and time estimation, which is at odds with the hypothesis that cerebellar or motor dysfunction might be the cause of dyslexia. This is further confirmed by the fact that there is no monotonic relationship between motor skills on the one hand and phonology and reading on the other, beyond the overall group differences. On the other hand, phonological skills are greatly affected in a large proportion of dyslexic children (77%), even though this may be underestimated by the fact that they received specific tuition including phonological training. And performance in the phonological tasks is linearly related to performance in reading, confirming the direct causal role of a phonological deficit in dyslexia.
In conclusion, this study supports the consensual view that a phonological deficit directly causes the reading impairment in dyslexia, and does not support the view that the phonological deficit itself arises from a more general cerebellar/automaticity/motor impairment. However, it may be the case that dyslexics, as well as children with other developmental disorders such as ADHD, are more susceptible to additional motor skill impairments than control children. Even if these motor defects do not play a causal role in the aetiology of reading impairments or attention deficits, it still remains to be explained why their incidence is increased in those populations. This is a challenge that developmental research has yet to meet.

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Tables

Table 1. Psychometric measures

Group	N (boys/girls)	Age	FSIQ	VIQ	PIQ	Reading ***	Spelling ***	VC	PO	FD **	Coding
Controls	20 (10/10)	9.8 (1.3)	102 (9.7)	102 (8.8)	101.5 (9.8)	113.7 (8.8)	113.9 (11.2)	100.9 (9.4)	102.3 (11.6)	103 (9.7)	9.65 (2.16)
Dyslexics	22 (16/6)	9.9 (1.2)	103.6 (10)	105.5 (10.4)	100.8 (11.4)	87.9 (10.6)	86.9 (8.5)	106.9 (12.4)	102.4 (12.3)	94.1 (10.5)	9.14 (3.03)

N: number of subjects.
Age converted into decimals.
FSIQ, VIQ, PIQ respectively full-scale, verbal and performance intelligence quotient as measured by WISC III^{UK}.
Reading and spelling: standard scores from WRAT3.
WISC index scores VC: Verbal Comprehension; PO: Perceptual Orientation; FD: Freedom from Distractibility.
Standard deviations in parentheses.
**: Difference significant at the $p<0.01$ level.
***: Difference significant at the $p<0.001$ level.

Table 2. Phonological Assessment Battery standard scores

Group	Alliteration **	Rhyme ***	Spoonerisms ***	Non-word reading ***	Picture naming	Digit naming *	Alliteration fluency ***	Rhyme fluency	Semantic fluency *
Controls (N=20)	98.60 (4.36)	108.00 (13.15)	112.30 (14.15)	117.80 (10.73)	103.85 (11.89)	105.15 (13.81)	111.90 (10.21)	112.95 (11.44)	111.30 (15.78)
Dyslexics (N=22)	91.91 (7.70)	93.86 (9.62)	91.41 (10.87)	96.68 (9.85)	95.41 (14.98)	96.18 (13.85)	99.09 (8.70)	107.00 (10.20)	102.68 (8.45)

Standard deviations in parentheses.
*: Difference significant at the p<0.05 level.
**: Difference significant at the p<0.01 level.
***: Difference significant at the p<0.001 level.

Table 3. Cerebellar tasks

Group	Postural stability *	Bead threading *	Finger to thumb *	Time estimation (N= 17 for controls)	Loudness estimation (N=18 for controls)
Controls (N=20)	1.00 (.79)	50.4 (7.4)	8 (3.1)	147 (29)	6.2 (0.89)
Dyslexics (N=22)	1.68 (.84)	63.7 (20.9)	13.5 (8.4)	202.6 (167.8)	7.3 (2.98)

Standard deviations in parentheses.
*: Difference significant at the p<0.05 level.

Figures

Figure 1. A schematic causal model of the phonological theory of dyslexia.
Three levels of description are distinguished: biological, cognitive and behavioural, with possible contributions from the environment. Boxes symbolise dysfunction at any of these levels, and arrows causal links between them.

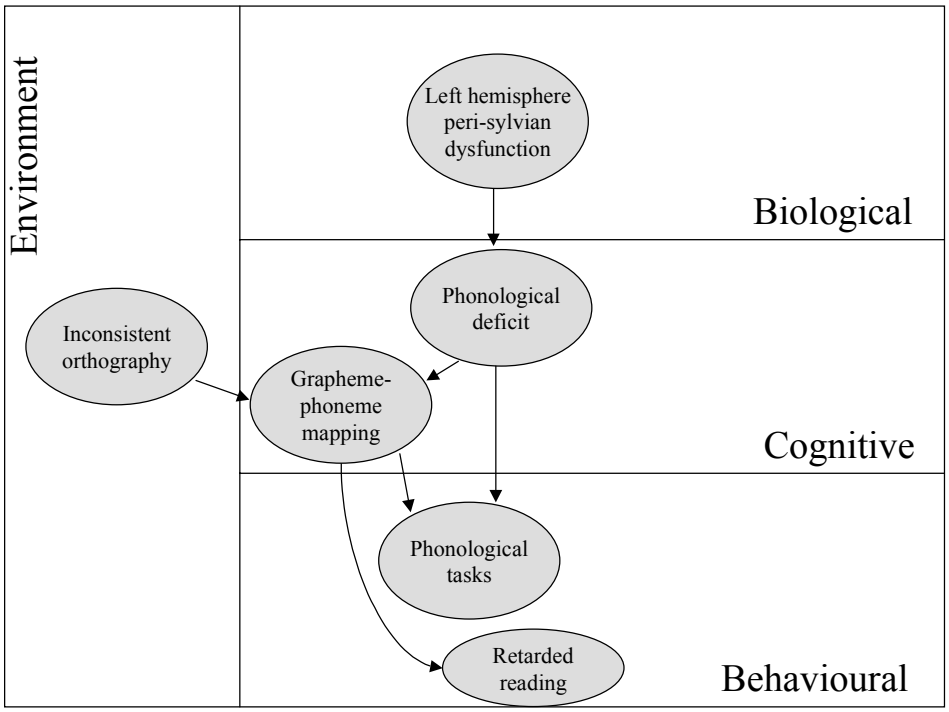

Figure 2. A schematic causal model of the cerebellar theory of dyslexia.