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Theories of developmental dyslexia: insights from a multiple case study of dyslexic adults

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Summary

A multiple case study was conducted in order to assess three leading theories of developmental dyslexia: (i) the phonological theory, (ii) the magnocellular (auditory and visual) theory and (iii) the cerebellar theory. Sixteen dyslexic and 16 control university students were administered a full battery of psychometric, phonological, auditory, visual and cerebellar tests. Individual data reveal that all 16 dyslexics suffer from a phonological deficit, 10 from an auditory deficit, four from a motor deficit and two from a visual magnocellular deficit. Results suggest that a phonological deficit can appear in the absence of any other sensory or motor disorder, and is sufficient to cause a literacy impairment, as demonstrated by five of the dyslexics. Auditory disorders, when present, aggravate the phonological deficit, hence the literacy impairment. However, auditory deficits cannot be characterized simply as rapid auditory processing problems, as would be predicted by the magnocellular theory. Nor are they restricted to speech. Contrary to the cerebellar theory, we find little support for the notion that motor impairments, when found, have a cerebellar origin or reflect an automaticity deficit. Overall, the present data support the phonological theory of dyslexia, while acknowledging the presence of additional sensory and motor disorders in certain individuals.

Keywords: dyslexia; audition; vision; magnocellular function; motor control

Abbreviations: ADHD = attention-deficit hyperactivity disorder; CoP = centre of foot pressure; FM = frequency modulation; IQ = intelligence quotient; IRI = inter-response interval; ISI = interstimulus interval; jnd = just noticeable difference; SLI = specific language impairment; SPL = sound pressure level; WAIS = Wechsler Adult Intelligence Scale; WMI = Working Memory Index; WRAT = Wide Range Achievement Test

Introduction

Developmental dyslexia is traditionally defined as a discrepancy between reading ability and intelligence in children receiving adequate reading tuition. Since the definition is entirely behavioural, it leaves open the causes for reading failure. It is now well established that dyslexia is a neurological disorder with a genetic origin, which is currently being investigated. The disorder has lifelong persistence, reading retardation being merely one of its manifestations. Beyond this consensus, and despite decades of intensive research, the underlying biological and cognitive causes of the reading retardation are still hotly debated. Indeed, there are no less than three major theories of dyslexia. The goal of the present study is to produce evidence to decide between these theories.

The major theories of developmental dyslexia

We begin by providing a neutral overview of the different theories of dyslexia, as described by their proponents. Note that there are different versions of each theory in the literature, which we are not able to represent in detail. Instead, we have chosen to describe the currently most prominent version of each theory.

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The phonological theory

The phonological theory postulates that dyslexics have a specific impairment in the representation, storage and/or retrieval of speech sounds. It explains dyslexics' reading impairment by appealing to the fact that learning to read an alphabetic system requires learning the grapheme–phoneme correspondence, i.e. the correspondence between letters and constituent sounds of speech. If these sounds are poorly represented, stored or retrieved, the learning of grapheme–phoneme correspondences, the foundation of reading for alphabetic systems, will be affected accordingly (Bradley and Bryant, 1978; Vellutino, 1979; Snowling, 1981; Brady and Shankweiler, 1991). While theorists have different views about the nature of the phonological problems, they agree on the central and causal role of phonology in dyslexia. The phonological theory therefore postulates a straightforward link between a cognitive deficit and the behavioural problem to be explained. At the neurological level, it is usually assumed that the origin of the disorder is a congenital dysfunction of left-hemisphere perisylvian brain areas underlying phonological representations, or connecting between phonological and orthographic representations.

Support for the phonological theory comes from evidence that dyslexic individuals perform particularly poorly on tasks requiring phonological awareness, i.e. conscious segmentation and manipulation of speech sounds. However, evidence for poor verbal short-term memory and slow automatic naming in dyslexics also points to a more basic phonological deficit, perhaps having to do with the quality of phonological representations, or their access and retrieval (Snowling, 2000). Anatomical work (Galaburda *et al*., 1985; Geschwind and Galaburda, 1985) and functional brain imaging studies support the notion of a left perisylvian dysfunction as a basis for the phonological deficit (Paulesu *et al*., 1996, 2001; Shaywitz *et al*., 1998; Brunswick *et al*., 1999; McCrory *et al*., 2000; Pugh *et al*., 2000; Temple *et al*., 2001; Shaywitz *et al*., 2002).

In order to better differentiate the phonological theory from the others, we discuss here only the strong version of the theory: that the cognitive deficit is specific to phonology. Indeed, challengers of the phonological theory do not dispute the existence of a phonological deficit and its contribution to reading retardation; rather, they uphold that the disorder is much more extended, having its roots in general sensory, motor or learning processes, and that the phonological deficit is just one aspect or consequence of the more general disorder.

The rapid auditory processing theory

The most obvious way to challenge the specificity of the phonological deficit is to postulate that it is secondary to a more basic auditory deficit. This is the claim of the rapid auditory processing theory, which specifies that the deficit lies in the perception of short or rapidly varying sounds (Tallal, 1980; Tallal *et al*., 1993). Support for this theory arises from evidence that dyslexics show poor performance on a number of auditory tasks, including frequency discrimination (McAnally and Stein, 1996; Ahissar *et al*., 2000) and temporal order judgement (Tallal, 1980; Nagarajan *et al*., 1999) (see reviews by Farmer and Klein, 1995; McArthur and Bishop, 2001). Abnormal neurophysiological responses to various auditory stimuli have also been demonstrated (McAnally and Stein, 1996; Nagarajan *et al*., 1999; Kujala *et al*., 2000; Temple *et al*., 2000; Ruff *et al*., 2002). The failure to correctly represent short sounds and fast transitions would cause further difficulties in particular when such acoustic events are the cues to phonemic contrasts, as in /ba/ versus /da/. There is indeed also evidence that dyslexics may have poorer categorical perception of certain contrasts (Mody *et al*., 1997; Adlard and Hazan, 1998; Serniclaes *et al*., 2001). In this view, the auditory deficit is therefore the direct cause, in the course of development, of the phonological deficit, and hence of the difficulty in learning to read. The original version of the auditory theory made no particular claim at the biological level, but we will see below that this is now specified within the magnocellular theory.

The visual theory

The visual theory (Lovegrove *et al*., 1980; Livingstone *et al*., 1991; Stein and Walsh, 1997) reflects another long-standing tradition in the study of dyslexia, that of considering it as a visual impairment giving rise to difficulties with the processing of letters and words on a page of text. This may take the form of unstable binocular fixations, poor vergence (Cornelissen *et al*., 1993; Stein and Fowler, 1993; Eden *et al*., 1994), or increased visual crowding (Spinelli *et al*., 2002). The visual theory does not exclude a phonological deficit, but emphasizes a visual contribution to reading problems, at least in some dyslexic individuals. At the biological level, the proposed aetiology of the visual dysfunction is based on the division of the visual system into two distinct pathways that have different roles and properties: the magnocellular and parvocellular pathways. The theory postulates that the magnocellular pathway is selectively disrupted in certain dyslexic individuals, leading to deficiencies in visual processing, and, via the posterior parietal cortex, to abnormal binocular control and visuospatial attention (Stein and Walsh, 1997; Hari *et al*., 2001). Evidence for magnocellular dysfunction comes from anatomical studies showing abnormalities of the magnocellular layers of the lateral geniculate nucleus (Livingstone *et al*., 1991), psychophysical studies showing decreased sensitivity in the magnocellular range, i.e. low spatial frequencies and high temporal frequencies, in dyslexics (Lovegrove *et al*., 1980; Cornelissen *et al*., 1995), and brain imaging studies (Eden *et al*., 1996).

The cerebellar theory

Yet another view is represented by the automaticity/cerebellar theory of dyslexia (Nicolson and Fawcett, 1990; Nicolson *et al*., 2001) (henceforth referred to as the cerebellar theory). Here the biological claim is that the dyslexic's cerebellum is mildly dysfunctional and that a number of cognitive difficulties ensue. First, the cerebellum plays a role in motor control and therefore in speech articulation. It is postulated that retarded or dysfunctional articulation would lead to deficient phonological representations. Secondly, the cerebellum plays a role in the automatization of overlearned tasks, such as driving, typing and reading. A weak capacity to automatize would affect, among other things, the learning of grapheme–phoneme correspondences. Support for the cerebellar theory comes from evidence of poor performance of dyslexics in a large number of motor tasks (Fawcett *et al*., 1996), in dual tasks demonstrating impaired automatization of balance (Nicolson and Fawcett, 1990), and in time estimation, a non-motor cerebellar task (Nicolson *et al*., 1995). Brain imaging studies have also shown anatomical, metabolic and activation differences in the cerebellum of dyslexics (Rae *et al*., 1998; Nicolson *et al*., 1999; Brown *et al*., 2001; Leonard *et al*., 2001).

The magnocellular theory

Finally, there is a unifying theory that attempts to integrate all the findings mentioned above. A generalization of the visual theory, the magnocellular theory (Stein and Walsh, 1997) postulates that the magnocellular dysfunction is not restricted to the visual pathways but is generalized to all modalities (visual and auditory as well as tactile). Furthermore, as the cerebellum receives massive input from various magnocellular systems in the brain, it is also predicted to be affected by the general magnocellular defect (Stein *et al*., 2001). Through a single biological cause, this theory therefore manages to account for all known manifestations of dyslexia: visual, auditory, tactile, motor and, consequently, phonological (for an attentional variant see Hari and Renvall, 2001). Beyond the evidence pertaining to each of the theories described previously, evidence specifically relevant to the magnocellular theory includes magnocellular abnormalities in the medial as well as the lateral geniculate nucleus of dyslexics' brains (Livingstone *et al*., 1991; Galaburda *et al*., 1994), poor performance of dyslexics in the tactile domain (Grant *et al*., 1999; Stoodley *et al*., 2000), and the co-occurrence of visual and auditory problems in certain dyslexics (Witton *et al*., 1998; Cestnick, 2001; van Ingelghem *et al*., 2001).

Although the auditory and visual theories have been presented here separately for historical and logical reasons, their supporters now agree that visual and auditory disorders in dyslexia are part of a more general magnocellular dysfunction. We will therefore not discuss the visual and auditory theories independently. Rather, we will restrict the discussion to a comparison between the phonological, cerebellar and magnocellular theories.

A critical look

The major weakness of the phonological theory is its inability to explain the occurrence of sensory and motor disorders in dyslexic individuals. Supporters of the phonological theory typically dismiss these disorders as not part of the core features of dyslexia. They consider their co-occurrence with the phonological deficit as potential markers of dyslexia, but do not see them as playing a causal role in the aetiology of reading impairment (e.g. Snowling, 2000).

The cerebellar theory also fails to account for sensory disorders, but its proponents entertain the idea of distinct cerebellar and magnocellular dyslexia subtypes (Fawcett and Nicolson, 2001). Another problem for the cerebellar theory is that the causal link postulated between articulation and phonology relies on an outdated view of the motor theory of speech, according to which the development of phonological representations relies on speech articulation. This view has long been abandoned in the light of cases of normal phonological development despite severe dysarthria or apraxia of speech (for a discussion see Liberman and Mattingly, 1985; Ramus *et al*., 2003). Finally, it remains uncertain what proportion of dyslexics are affected by motor problems. A number of studies have failed to find any (Wimmer *et al*., 1998; van Daal and van der Leij, 1999; Kronbichler *et al*., 2002), others have found motor problems only in a subgroup of dyslexics (Yap and van der Leij, 1994; Ramus *et al*., 2003), and it has been suggested that motor dysfunction is found only in dyslexic children who also have attention-deficit hyperactivity disorder (ADHD) (Denckla *et al*., 1985; Wimmer *et al*., 1999).

The magnocellular theory, unique in its ability to account for all manifestations of dyslexia, is undoubtedly attractive. Nevertheless, it also has its problems and has been facing growing criticism in recent years (e.g. Ramus, 2001). One line of criticism emphasizes a number of failures to replicate findings of auditory disorders in dyslexia (Heath *et al*., 1999; Hill *et al*., 1999; McArthur and Hogben, 2001). Other studies do find auditory deficits in dyslexics, but only in a subgroup, ranging from a few isolated individuals to 50% of the population studied (Tallal, 1980; Reed, 1989; Manis *et al*., 1997; Mody *et al*., 1997; Adlard and Hazan, 1998; Lorenzi *et al*., 2000; Marshall *et al*., 2001; Rosen and Manganari, 2001). Another line of criticism focuses on results inconsistent with the idea that the auditory deficit lies in 'rapid' auditory processing, and therefore with magnocellular function: indeed, with some tasks 'rapid' auditory processing is found to be intact, while with others 'slow' auditory processing is found to be impaired (Reed, 1989; McAnally and Stein, 1996; Adlard and Hazan, 1998; Schulte-Körne *et al*., 1998*b*; Witton *et al*., 1998; Nittrouer, 1999; Lorenzi *et al*., 2000; Rosen and Manganari, 2001; Share *et al*., 2002). It is also argued that auditory deficits do not predict

phonological deficits (Mody *et al*., 1997; Schulte-Körne *et al*., 1998*a*; Bishop *et al*., 1999; Marshall *et al*., 2001; Rosen and Manganari, 2001; Share *et al*., 2002). Criticism of the visual side of the magnocellular theory also focuses on failures to replicate findings of a visual deficit (Victor *et al*., 1993; Johannes *et al*., 1996), or on findings of such a deficit only in a subgroup (Cornelissen *et al*., 1995; Witton *et al*., 1998; Amitay *et al*., 2002), and on inconsistencies between predictions and empirical results. Most notably, visual impairments, when found, seem to be observed across a whole range of stimuli, not just those specifically tapping the magnocellular system (Skottun, 2000; Amitay *et al*., 2002; Farrag *et al*., 2002). There is also negative evidence regarding cross-modal sensory deficits (Heim *et al*., 2001). More generally, the idea that the magno-/parvocellular distinction can be extended to non-visual sensory systems remains controversial (personal communication, B. Skottun, 2002).

In summary, the phonological theory suffers from its inability to explain the sensory and motor disorders that occur in a significant proportion of dyslexics, while the magnocellular theory suffers mainly from its inability to explain the absence of sensory and motor disorders in a significant proportion of dyslexics. The cerebellar theory presents both types of problems.

Of course, it is possible that the three theories are true of different individuals. For instance, there could be three partially overlapping subtypes of dyslexia, each being an independent contribution to reading difficulties: phonological, auditory/visual, and cerebellar. Alternatively, it could also be that just one theory accounts for every case of dyslexia, and that the other manifestations observed are markers, i.e. they are associated without causation. In order to tease apart the many possible alternatives, we need to be able to answer such questions as: What proportion of dyslexics have a given deficit? Are there dissociations between certain deficits? Are there systematic associations between certain deficits? Unfortunately, the current literature does not contain answers to any of these questions. Indeed, virtually all studies have focused on just one or a few tasks within one modality, and most of them have only analysed group differences, making it impossible to assess what proportion of dyslexics are really affected by a deficit.

Three notable exceptions are worth mentioning. Witton *et al*. (1998) have shown significant differences between dyslexic and controls in frequency modulation (FM) detection at 2 Hz and coherent motion detection. The individual data reported suggest that four dyslexics out of 17 had abnormal performance in the visual task, nine out of 17 in the auditory task, and 15 out of 17 in non-word reading. The absence of phonological and cerebellar tasks prevents the assessment of what might explain the reading impairment of the seven dyslexics who have normal visual and auditory performance, and to analyse the relationships between all the variables and their predictive power with respect to reading.

Van Ingelghem and colleagues tested both visual and auditory gap detection in dyslexic children and found significant group effects for both (Van Ingelghem *et al*., 2001). They report that nine dyslexics out of 10 were impaired in the auditory task and seven out of 10 in the visual task. However, their criterion for being impaired was that the individual's threshold be above the 95% confidence interval for the control group, that is, for 10 individuals, >0.67 SD above the control mean. This makes it an extremely liberal criterion, since if the control group is normally distributed, ~25% of the controls should also meet it (individual control data not available). Again, cerebellar and phonological performance was not tested. This study is also potentially undermined by the fact that the two groups were not matched in non-verbal intelligence quotient (IQ), a factor that is known to affect performance significantly in psychophysical tasks (Ahissar *et al*., 2000).

It seems that only one study to date has assessed all the relevant modalities in a group of dyslexics (Kronbichler *et al*., 2002). The authors administered a battery of phonological tests and tests of auditory illusory movement perception, visual coherent motion detection, and peg moving. They report significant differences between the two groups in the phonological tests, but none in the auditory, visual or motor tasks. Unfortunately, no individual data are reported to allow assessment of whether some dyslexics could have sensory or motor disorders, and the relationships between the variables are not analysed. In all three studies, only one task for each modality was administered, leaving open the possibility that other, more sensitive tasks, might change the picture significantly.

The present study

Our aim was to produce data that would enable us to start answering questions concerning associations, dissociations and, eventually, causal relationships between sensory, motor, phonological and reading disorders. Our approach was that of a multiple case study: by having the most comprehensive neuropsychological profile for each individual, we sought to identify who had which combination of disorders and, crucially, who did not have a given disorder. We therefore created a battery of psychometric, phonological, auditory, visual and cerebellar tests to be administered to each subject. Within each domain, we selected several tasks that have, according to the literature, most consistently shown differences between dyslexics and controls.

Because we felt that dissociations between disorders would be the most informative, we selected a special dyslexic population, consisting of university students. Obviously, the few dyslexics who enter university are not representative of the whole population: they may be more intelligent, resourceful and socially privileged, and may have received better help with respect to reading. Most importantly, we hypothesized that they would be least likely to accumulate several types of disorders. For instance, if a phonological and a visual disorder can appear independently, an individual having both disorders should be less likely to succeed academically than an

individual with just one of them. By studying a high-achieving population, we therefore maximized our chances of finding pure cases of the different possible subtypes of dyslexia. For the same reason, we also minimized the chances of studying individuals with another comorbid developmental disorder, such as specific language impairment (SLI), ADHD and developmental coordination disorder, which would be an undesirable confound.

Methods

Subjects

Seventeen dyslexic university students at University College London (UCL) volunteered for this study. They had all received a formal diagnosis of developmental dyslexia by a qualified educational psychologist in secondary school or earlier, and most of them had a documented history of reading difficulties. They were initially contacted via UCL's Examination Section, where dyslexic students may apply for time concessions. With their agreement, their files were made available to us so that we could exclude at this stage all individuals who also suffered from another neurological or psychiatric disorder, with special attention to SLI, ADHD, developmental coordination disorder and autism. Additional inclusion criteria were checked after a first testing session; these were a full-scale IQ >100 and reading and spelling standard scores <110 on average. One dyslexic subject was excluded after the first session because his reading and spelling scores averaged 114.5, thereby reducing the sample to 16.

Seventeen control subjects were recruited from the same university. Inclusion criteria were: no known developmental, neurological or psychiatric disorder; full-scale IQ >100; and reading and spelling scores >100. One subject was excluded after the first session because his reading and spelling scores averaged 98.5 and he showed signs of phonological problems; this reduced the sample to 16.

It was checked *a posteriori* that the two groups were matched overall in age, sex and full-scale IQ. All the subjects gave informed consent according to the Declaration of Helsinki and the study was approved by the Joint UCL/UCL Hospitals Committee on the Ethics of Human Research.

A battery of psychometric, phonological, auditory, visual and cerebellar tests amounting to ~10 h of testing was administered to each individual in several sessions, lasting 1–2 h each.

Psychometric tests

Verbal intelligence and non-verbal intelligence were assessed using the Wechsler Adult Intelligence Scale (WAIS-III^{UK}; Wechsler, 1998). Reading and spelling were assessed using the Wide Range Achievement Test (WRAT3; Wilkinson, 1993), the National Adult Reading Test (NART; Nelson, 1991), concentrating mainly on rare and irregular words, and a reading speed test adapted from the Neale Analysis of Reading Ability (NARA; Neale, 1997). Non-word reading was also assessed, using 20 non-words from the Graded Nonword Reading Test (GNRT; Snowling *et al*., 1996). Each non-word was presented on a computer screen. Overall reading time was recorded as well as accuracy.

Screening for other disorders

To check for possible language impairments, two non-phonological language tests were administered. These two tests have been shown previously to be sensitive to subtle impairments of syntax in SLI children and adolescents (van der Lely, 1996*a*; van der Lely and Stollwerk, 1997).

Advanced Syntactic Test of Pronominal reference (ASTOP) (van der Lely, 1997). A sentence was played through headphones by a computer and a picture was displayed at the same time. The subject had to press one of two keys to indicate whether the sentence described the picture or not. The 96 items in this test assessed the understanding of pronominal reference and quantifiers in embedded phrases (such as 'Minnie the Minx says every dancer is pinching herself').

*Test of active and passive sentences (TAPS) (van der Lely, 1996*b*).* A sentence was played through headphones by a computer and four pictures were displayed at the same time. The subject had to press one of four keys to indicate which picture was best described by the sentence. The 48 items assessed the correct computation of agent–patient relationships in active and passive sentences (such as 'The car is hit by the lorry').

To determine the possible presence of attention deficit disorder, each subject completed the Brown attention deficit disorder questionnaire (Brown, 1996).

Phonological tests

Automatic picture naming

The subject was asked to name 50 pictures of five objects (hat, ball, table, door, box) as fast as possible. A second measure was taken with a different ordering of the 50 pictures. Total naming time was recorded irrespective of accuracy. This task is taken from the Phonological Assessment Battery (PhAB; Frederickson *et al*., 1997).

Automatic digit naming

This was the same as the automatic picture naming test, but with two lists of 50 digits.

Spoonerisms

Upon hearing a pair of words (like 'basket–lemon') via loudspeakers, the subject had to swap their initial phonemes

and pronounce the resulting pair of non-words ('lasket–bemon') in the correct order. The stimuli were 12 pairs of words from McCrory (McCrory, 2001), which were recorded on hard disk and played one at a time from a computer. Both accuracy and time taken to produce each pair (from offset of stimulus) were recorded.

Non-word repetition

Upon hearing a non-word through headphones, the subject had to repeat it immediately. The stimuli were 40 non-words from the Children's Test of Nonword Repetition (Gathercole and Baddeley, 1996), recorded on hard disk and played by computer.

Tests of auditory perception

All tests were performed in a quiet room using headphones which (except for audiological screening) were calibrated using a B&K 4157 ear simulator (Brüeland Kjaer, Naerum, Denmark). Masked thresholds and the syllable–formant discrimination task were run using special-purpose psychoacoustic hardware and Sennheiser HD 475 headphones. The other tasks were run on a laptop with Sony MDR-CD270 headphones.

Audiological screening

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

Backward and simultaneous masking

The masking tasks were modelled closely on corresponding ones described by Wright and colleagues (Wright *et al*., 1997), with identical stimuli but a different adaptive procedure. Thresholds were measured monaurally in the right ear using a two-interval, two-alternative forced-choice adaptive task tracking 79% correct using Levitt's (1971) procedure with modifications by Baker and Rosen (2001) to increase efficiency. On each trial, two 300 ms bursts of a bandpass masking noise [0.6–1.4 kHz at a spectrum level of 40 dB sound pressure level (SPL)] were presented with a 340 ms interstimulus interval (ISI). The 20 ms 1 kHz sinusoidal probe tone occurred along with one of the noise bursts. The listener indicated which of the noise bursts was associated with the probe by pressing one of two buttons on a response box. Feedback was given by lighting the correct button. The probe tone could occur either simultaneously with the masking noise (200 ms after masker onset; simultaneous masking) or with its onset 20 ms prior to the start of the masker (backward masking). All stimuli were gated on and off with 10 ms cosine-squared envelopes.

The probe tone was set to be clearly audible at the beginning of each test, its level decreasing by 8 dB after each correct response until the first reversal. Hereafter, the standard 3-down/1-up rule was implemented, with a decreased step size of 6 dB. Step size decreased by 2 dB after each successive reversal until it was 2 dB, at which point four further reversals were obtained. The final threshold value was estimated as the mean of the final four reversal points.

Absolute thresholds for perception of the probe tone were also acquired in a condition with no masking noise.

A minimum of two tests of threshold and simultaneous masking took place per subject, and four of backward masking (because there is greater within-subject variability in this condition). All tests of a condition took place consecutively, with reversal of the order from one subject to the next. Absolute thresholds were always tested between backwards and simultaneous masking, and further tests were run if two thresholds for a subject were not within 6 dB. Once this criterion was met, medians of all the tests run in each condition were taken as the final index of performance.

Formant discrimination in syllables and non-speech analogues

The ability of subjects to discriminate second-formant transitions in speech and non-speech sounds was assessed using the software package described by Carrell and colleagues (Carrell *et al*., 1999).

A *ba–da* continuum and the corresponding non-speech analogues were generated using the Klatt (1980) synthesizer in cascade mode with a 1 ms update interval. The 41 stimuli in each continuum differed only in second-formant (F2) onset frequency, which was varied in equal logarithmic steps.

The *ba–da* continuum was based on that specified by Mody and colleagues (Mody *et al*., 1997) but with only the lower two formants and with a monotone fundamental frequency at 125 Hz. The voicing source was turned off 235 ms into the signal and allowed to decay naturally so as to avoid transients. The total duration of each signal was 250 ms. Steady-state formant frequencies were 750 and 1200 Hz with bandwidths of 90 Hz for both. The first-formant (F1) transition was identical for all stimuli, beginning at 200 Hz and reaching 750 Hz after 35 ms. The second formant (F2) began at 825 Hz for *ba* and at 1500 Hz for *da*, reaching its steady-state value of 1200 Hz after 50 ms. Non-speech isolated-F2 stimuli were obtained simply by outputting from the synthesizer the waveforms from the F2 resonator on their own (a straightforward option in the Klatt synthesizer). Note that no plosive release bursts were included. Thus the crucial acoustic distinction was carried only by the F2 transition and was similar for the speech and the non-speech stimuli.

The discrimination task was based on a 4IAX (four-interval, two-alternative, forced-choice same–different) procedure. On each trial, two pairs of stimuli are heard, with a longer interval (900 ms) between the pairs than within

(300 ms). One pair of stimuli are identical, being two repetitions of the most extreme *ba*. In the other, the *ba* is paired with another stimulus on the continuum. The subject is required to indicate which pair of stimuli is different. At the beginning of the test, the *ba* is paired with an extreme *da*, but an adaptive procedure chooses the comparison stimulus so as to estimate the stimulus which is discriminable from the *ba* 69% of the time. Exactly the same procedure was applied to test discrimination of the non-speech analogues.

Subjects were not acquainted with the sounds being presented until the trials began, aside from a verbal explanation. Two consecutive measurements of the just noticeable difference (jnd) were acquired for each condition (*ba* and non-speech analogues), with the order alternated between subjects.

Phonemic categorization

Categorization functions were obtained for three speech sound continua using special-purpose software known as SPA (Speech Pattern Audiometry). Two of the continua varied place of articulation (*ba–da*, *date–gate*) and one varied voicing (*coat–goat*). The *ba–da* continuum was the highly schematic one described above.

Both *date–gate* and *coat–goat* were based, with minor modifications, on the 'combined-cue' synthetic continua developed by Hazan and Barrett (2000) using the Klatt (1980) synthesizer. Unlike the *ba–da* continuum, these were modelled closely on a particular speaker's tokens (an adult female speaker of standard southern British English). They are much more complex than typical formant-synthesized speech, and sound much more natural.

The *date–gate* continuum varied both the spectrum of the initial release burst and the starting frequencies of the second and third formants to signal the change in place of articulation. The *coat–goat* continuum varied voice onset time in 1 ms steps (the first formant onset frequency covaried with voice onset time, as it does naturally). Both continua consisted of 51 stimuli. Further details of their properties can be found in Hazan and Barrett (2000).

On each trial of the test, subjects heard a single stimulus and indicated which they had heard by clicking on one of two pictures on the computer screen (except for 'BA' and 'DA', which were spelled out in upper case letters, as here). Two independent adaptive tracks, with Levitt's (1971) rules as modified by Baker and Rosen (2001), were used to estimate the points on the continuum at which the stimuli were labelled as one word of the pair (e.g. 'coat') 29 and 71% of the time. The procedure terminated when there was a total of five reversals on each track, or a maximum of 50 trials. Tracks started at the endpoints of the continuum, and step size decreased from a large step to a smaller one over the first three reversals. In order to assist in the stability of the phoneme categories, continuum endpoints were randomly interspersed throughout the test on 20% of the trials. The categorization function was derived from all trials in a particular test, and summary statistics for slope and category boundary estimated by probit analysis. Shallower slopes indicate less sensitivity to variations in the particular acoustic feature used in the continuum.

Frequency modulation detection at 2 and 240 Hz

Stimuli were modelled closely on those used by Talcott and colleagues (Talcott *et al*., 2000). Each trial consisted of two 1 s tone bursts (20 ms rise/fall times) separated by an interstimulus interval of 500 ms. In each pair, one of the tones was a sinusoid of 1 kHz, whereas the other was frequency-modulated. Two modulation frequencies were used (2 and 240 Hz). For each modulation frequency, a continuum of 100 stimuli was constructed spanning a wide range of values of the modulation index (a maximum modulation index of 4.95 in steps of 0.1 for the 2 Hz modulation frequency and 0.02475 in steps of 0.0005 for 240 Hz). These correspond to maximum frequency deviations of 9.9 and 5.94 Hz, respectively, for the two continua. Stimuli were presented through headphones at ~75 dB SPL.

The discrimination task was run in the guise of an identification experiment using the SPA software described above, but without continuum endpoints randomly interspersed. Subjects indicated which tone was modulated by clicking on an appropriate graphic. Feedback was provided in the form of appropriate pictures (a happy face for correct responses and a sad face for incorrect ones). Probit analysis was used to fit cumulative Gaussian Functions to the psychometric functions, so as to obtain an estimate of the modulation that was detectable 75% of the time.

Temporal order judgement of long and short sounds

The temporal order judgement task was based on two sounds, readily identifiable without prior training as a car horn (periodic with a fundamental frequency of ~400 Hz) and an aperiodic dog bark. Starting from sounds accompanying a children's computer game, various manipulations of amplitude envelope and duration were used to create stimuli with a total duration of 115 ms each, with rise and fall times of 5 ms ('long' sounds). The two stimuli were then normalized to have the same root mean square level. The continuum of sounds consisted of 204 stimuli in which the stimulus onset asynchrony varied from +405 ms (horn leading dog) to –405 ms (dog leading horn) in 4 ms steps. Stimuli were allowed to overlap to the degree necessary to create the specified stimulus onset asynchrony values. 'Short' sounds were the same stimuli cut to 30 ms duration, thus minimizing stimulus overlap at short stimulus onset asynchrony values at the expense of less distinctive sound qualities. For testing, the same adaptive procedure and data analysis were employed as for FM detection, but the subjects indicated simply which

sound (dog or car horn) they heard first. Feedback as to the correctness of response was given after every trial.

Tests of visual perception

A more detailed description of these tests is available in supplementary material at http://www.lscp.net/persons/ramus/dyslexia02/supp.html.

Equipment

Experimental procedures and stimulus generation were controlled by a Macintosh computer (Apple Computer). Experiments were run under the MatLab programming environment (Mathworks, Natick, MA, USA). Software for display calibration and stimulus display contained elements of the VideoToolbox (Pelli, 1997) and PsychToolbox (Brainard, 1997) software packages. Stimuli were displayed on a 19 inch Sony Trinitron CRT monitor operating at a screen resolution of 1024 × 768 pixels with a frame refresh rate of 85 Hz. Subjects viewed the screen binocularly at a viewing distance of 228 cm for the acuity experiment and 114 cm for all other conditions. Under these conditions one pixel subtends 0.5 and 1.0 min of arc respectively. Subjects always fixated the centre of the screen, aided by the presence of a continuously visible fixation marker. Subjects made all responses on a numeric keypad clearly marked with available choices.

Experimental procedure

An adaptive psychophysical staircase procedure (QUEST; Watson and Pelli, 1983) was used to estimate thresholds. QUEST works by sampling a range of cue levels and using subjects' responses, in combination with a Bayesian estimator, to attempt to converge on the cue level yielding 83% correct performance on the task. Unless stated otherwise, runs consisted of blocks of 45 trials and at least three runs were undertaken for each data point. Feedback, in the form of an audible beep, was used to indicate errors. Each subject underwent at least three runs in each task and the median of all runs is reported.

Visual acuity

Subjects were presented with a Landolt C, centred on their point of fixation, at one of four orientations (0°, 90°, 180° or 270° rotation). The letters appeared white (100 cd/m^2) on a grey (50 cd/m^2 background. By convention, the thickness of the stroke forming the C is 1/5 of the letter diameter, as is the height of the gap. Subjects performed a single-interval 4AFC (four-alternative forced choice) task to report the orientation of the letter using the keypad. Stimuli were presented for a total of 500 ms and were smoothly ramped on and off with a Gaussian contrast envelop (σ = 200 ms) to minimize the contribution of transients at the stimulus onset and offset. Threshold sizes of the 'C' gap (expressed in arc min) were converted to produce a minimum angle of resolution (MAR). This was then converted to Snellen acuity (Snellen acuity in metres = minimum angle of resolution 6/6*).

Contrast sensitivity, magno- versus parvo-cellular

Perhaps the most direct way to assess magno-cellular (M) versus parvo-cellular (P) function is to measure differences in sensitivity to low-contrast stimuli designed to target each stream. A number of studies have interpreted such contrast sensitivity findings as supporting M deficits in dyslexics (e.g. Martin and Lovegrove, 1987; Slaghuis and Ryan, 1999) (for a critical review see Skottun, 2000; for other objections see Stuart *et al*., 2001). However, many such studies have been methodologically flawed either in terms of the spatial/temporal frequencies of stimuli employed or because, while some show poor dyslexic performance on M-specific stimuli, few establish normal performance with P-specific stimuli (Skottun, 2000). We sought to avoid these pitfalls and measured contrast sensitivity using a grating detection task.

Stimuli were Gabor patterns: cosinusoidal gratings spatially windowed by an isotropic Gaussian contrast envelope (σ = 1.0°) (Fig. 1A, B). We tested two combinations of spatial and temporal frequency: magnocellular-selective (M-selective) stimuli had a peak spatial frequency of 0.5 cycles per degree (c/°) and counter-phase flickered at the rate of 15 reversals/s, while parvocellular-selective (P-selective) stimuli had a peak spatial frequency of 8.0 c/° and did not counter-phase flicker. Spatial frequency values were chosen to span the point at which psychophysical detection switches from transient to sustained mechanisms (~1.5 c/°) (Legge, 1978). To further target the magnocellular pathway we followed Demb and colleagues (Demb *et al*., 1998) in making M-selective stimuli low-luminance, since it is known that the M-pathway response is dominant at mesopic/scotopic light levels (Purpura *et al*., 1988; Lee *et al*., 1997). M-selective stimuli therefore had a mean luminance of 5 cd/m^2 (range 0–10 cd/m^2) while P-selective stimuli varied around a mean luminance of 40 cd/m^2 (range 0–80 cd/m^2). Stimulus duration was 500 ms. In order to minimize the impact of onset and offset transients in P-selective conditions, the contrast of all stimuli was smoothly ramped on and off with a Gaussian contrast envelope (σ = 200 ms).

Subjects were presented with two intervals; one randomly selected interval contained a Gabor patch (with carrier in random phase), the other a blank field at background luminance. The subjects' task was then to indicate which interval contained the grating; this is a 2AFC (two-alternative forced-choice) task). The onset of each interval was indicated by an auditory cue, and intervals were separated by a 500 ms ISI. Contrast detection thresholds are presented as percentage Michelson contrast [$(L_{min} - L_{max})/(L_{min} + L_{max})$, where L_{min}

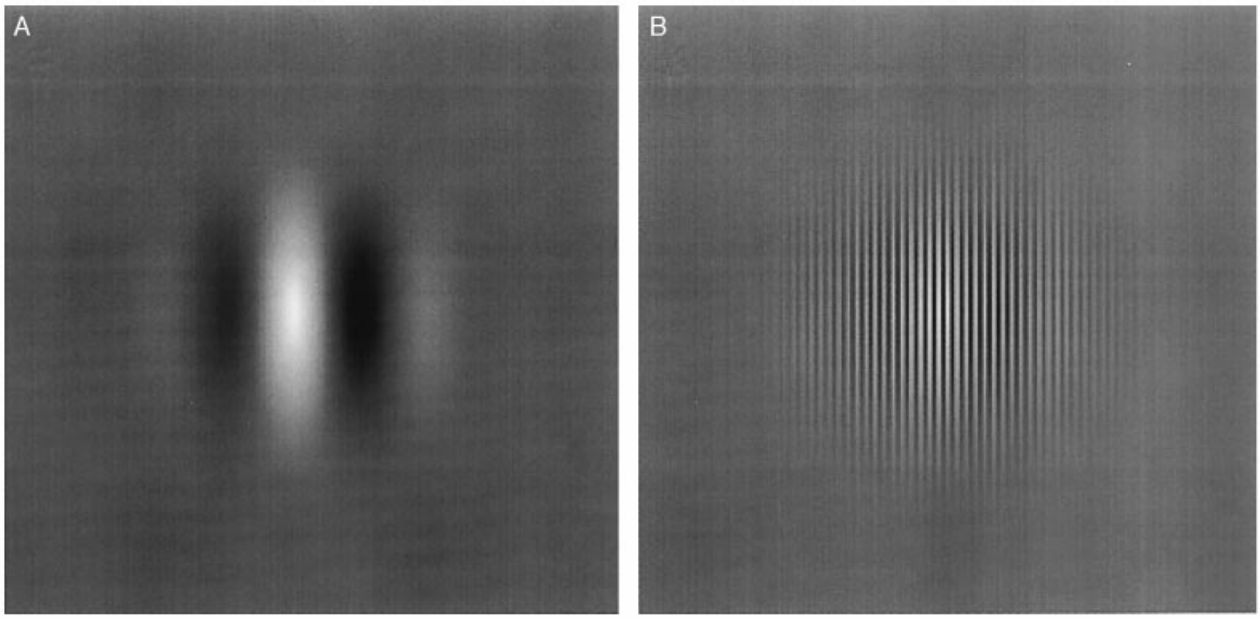

Fig. 1 Stimuli used for contrast sensitivity and speed discrimination. (**A**) Magnocellular-specific stimulus; (**B**) Parvocellular-specific stimulus.

and L_{max} are the luminances of the darkest and brightest parts of the display, respectively (in cd/m^2)].

Speed discrimination, magno- versus parvo-cellular

There is evidence that while poorer contrast sensitivity for M-selective stimuli may not reliably co-occur with dyslexia, poor speed discrimination might (Eden *et al*., 1996; Demb *et al*., 1998). We measured speed discrimination using versions of the stimuli similar to those we used to probe contrast detection (described in the preceding section) but with drifting carriers. The P- and M-selective stimuli were tested with reference speeds of 1.0 and 16.0°/s and contrasts of 20 and 80%, respectively. Speeds were selected not only to target transient and sustained mechanisms, but also to produce equivalent temporal frequencies in terms of carrier cycles/s (i.e. an M : P speed ratio of 16 : 1 and an M : P spatial frequency ratio of 1 : 16). Stimulus contrast was again enveloped using a temporal Gaussian function. However, in order to prevent subjects counting the number of bars passing rather than judging speed, the standard deviation of the envelope was varied uniformly and randomly between 160 and 240 ms. Neither class of stimulus flickered, but in all other respects (e.g. luminance differences) they were identical to the detection stimuli described above.

Subjects were presented with two intervals, both containing a Gabor patch with a carrier drifting randomly to the left or right. In one randomly selected interval the carrier moved at reference speed; in the other it moved slightly faster. Subjects indicated the interval in which the grating moved faster (2AFC). QUEST was used to estimate the percentage increase in speed over baseline required to perform this discrimination with 83% accuracy. Intervals were again separated by an ISI of 500 ms and, although all stimuli were clearly visible, were also audibly pre-cued.

Coherent motion detection

A number of studies have claimed that dyslexics are poorer at detecting coherent motion embedded in moving noise than normal controls (Cornelissen *et al*., 1995; Eden *et al*., 1996; Raymond and Sorenson, 1998; Witton *et al*., 1998; Everatt *et al*., 1999; Slaghuis and Ryan, 1999; Talcott *et al*., 2000), and it has further been claimed that poor coherent motion detection correlates with poor letter position encoding (Cornelissen *et al*., 1998). We sought to test these findings and broadly followed the methods of Witton and colleagues (Witton *et al*., 1998) for generating stimuli. Subjects were presented with an 8° × 8° field of 150 randomly positioned dots (each subtending 1 arc min), appearing white (100 cd/m^2) on a grey background (50 cd/m^2), and moving rapidly (11°/s) to the left or the right. Stimulus movies lasted for 900 ms and consisted of 19 distinct frames. Dots appeared for a maximum of four movie frames before being randomly replaced (limited lifetime elements) to minimize the possibility of subjects using tracking eye movements. Subjects performed a single-interval 2AFC task: to report whether the dots were moving, on average, to the left or the right. The difficulty of the task was manipulated (using QUEST) by replacing a proportion of the elements with dots moving in a random direction (with the same lifetime, speed, etc.). The threshold estimate corresponds to the minimum proportion of

coherently moving dots supporting 83% discrimination of direction.

Cerebellar tests

Each subject underwent a battery of tests measuring balance, motor coordination and timing, all involving the cerebellum to some degree. Obviously, poor performance in any of these tests could have causes other than cerebellar dysfunction, but it was hoped that, by bringing together a battery of varied tasks involving the cerebellum, difficulties across the whole battery would be a good indication of cerebellar dysfunction.

Balance/dual task

The subjects' static balance was assessed in four different conditions of increasing difficulty: (i) eyes open, feet apart; (ii) eyes closed, feet together; (iii) eyes closed, feet together, arms extended; (iv) eyes closed, feet together, arms extended and counting backwards. This last condition was inspired directly by Nicolson and Fawcett (1990); the presence of the secondary task is meant to evaluate the automaticity of the subject's balance. Because dyslexics might find it more difficult to count backwards (because of phonological problems), the difficulty of the task was calibrated as in Nicolson and Fawcett (1990): prior to the test session, the speed with which each subject was able to count backwards in 3 s was measured and used to determine the steps in which they should count during the balance dual task: in ones, in twos, in threes or in sevens. In each trial, subjects were instructed to stand as still as possible while measurements were made over a 40 s period. Each of the four conditions was repeated three times for each subject, and the order of the 12 resulting trials was counterbalanced across two groups of subjects.

In order to assess the subjects' stability more objectively than in previous studies, we measured body movements and the changes in position of the ground reaction force (centre of foot pressure, CoP). Movements of the body were measured using an opto-electronic motion analysis system (CODA mpx30; Charnwood Dynamics, Rothley, UK), which tracked in three dimensions infrared-emitting diodes attached to anatomical landmarks. Movements at the level of the neck and wrists were obtained from infrared-emitting diodes that were fixed to the skin over the C7 spinous process and over both ulnar styloid processes. Movements of the CoP between the feet and the ground were calculated from the distribution of forces measured from a force plate (Kistler type 9287; Kistler Instrumente, Winterthur, Switzerland). The force plate data were low-pass filtered (50 Hz cut-off frequency) before digitization. All data were sampled at 100 Hz. Body stability was assessed by calculating the total distance (path length) travelled by each infrared-emitting diode in three dimensions and by the CoP in two dimensions during each 40 s trial. To reduce the influence of noise on path length measures, the data were averaged over every 10 data points, which reduced the effective sampling rate to 10 Hz. The distances between successive data points were then calculated and summed to give total path length.

Bead threading

Subjects had to thread 15 beads as fast as possible, holding the string in the dominant hand. The dependent measure was total time taken, and was assessed twice. This task and the test material were taken from the Dyslexia Screening Test (Fawcett and Nicolson, 1996).

Finger-to-thumb

Subjects placed 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 rotated one hand clockwise and the other anticlockwise until the finger and thumb touched again, and so on. The task was demonstrated and subjects were trained until they completed the movement fluently five times. They were then asked to perform 10 such movements as fast as possible. The measure was the time taken for 10 movements, and was assessed twice. This test was drawn from the Dow and Moruzzi (1958) battery and was administered as described by Fawcett and colleagues (Fawcett *et al*., 1996).

Repetitive finger-tapping

Subjects were asked to press repeatedly and as fast as possible a button on a response box with the index finger of their dominant hand. One hundred presses were recorded and the dependent measure was the average interval between two presses. This task was adapted from Denckla and colleagues (Denckla *et al*., 1985).

Bimanual finger-tapping

Bimanual finger-tapping in synchrony with a metronome was recorded in three conditions: (i) left and right hand alternately at 2 Hz; (ii) left and right hand alternately at 5 Hz; (iii) asymmetrical rhythm (tap twice with the dominant hand then once with the other hand and so forth) at 4 Hz. In each condition, subjects first had to tap for 30 s in synchrony with the metronome, then the metronome stopped and they had to continue for 30 s at exactly the same pace. Subjects had to rest their hands on the table and move only the index fingers at the metacarpophalangeal joint. The metronome sound was produced by a computer, which also recorded the subjects' responses through a response box. Dependent measures were the average inter-response interval (IRI) and its standard deviation. Previous work suggested that adult dyslexics would show greater IRI variability in the fast (5 Hz) and asymmetrical conditions (Wolff *et al*., 1990; Wolff, 1993).

Table 1 *Psychometric tests (mean ± SD)*

	Age (years)	FSIQ	VIQ*	PIQ	VCI	POI	WMI***	PSI	ADD (*T*-score)
Controls (n = 16)	21.9 ± 2.2	124.8 ± 10.5	127.4 ± 9.8	116.5 ± 10.3	127.2 ± 11.3	115.2 ± 10.8	117.6 ± 11.8	115.1 ± 10.2	57.7 ± 8.1
Dyslexics (n = 16)	21.1 ± 1.4	122.7 ± 4.5	119.6 ± 7.3	122.2 ± 6.1	125.3 ± 9.4	121.5 ± 9	99.3 ± 7.1	111.9 ± 17.5	62.9 ± 11.1

FSIQ = full-scale IQ; VIQ = verbal IQ; PIQ = performance IQ; VCI = verbal comprehension index; POI = perceptual orientation index; WMI = Working Memory Index (WAIS); PSI = Processing Speed Index (WAIS). ADD = attention deficit disorder scale. *$P < 0.05$; ***$P < 0.001$.

Time versus loudness estimation

Time estimation is the only cerebellar task not involving motor control, and is therefore crucial in distinguishing the cerebellar hypothesis from a solely motor one. We used exactly the same task as Nicolson and colleagues (Nicolson *et al*., 1995), which was inspired by Ivry and Keele (1989).

In each time estimation trial, two tones were presented successively, and the task was to say whether the second one was longer or shorter than the first. The standard stimulus, always presented first, was a 1200 ms 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 silence interval of 1000 ms. Each trial was repeated three times, giving a total of 66 test trials, which were presented in random order. The test block was preceded by a practice block of eight trials (using only the eight extreme comparison tones), during which feedback was provided. No feedback was provided during the test block. The stimuli were presented by a computer through headphones at ~75 dB SPL. After each pair of sounds, subjects had to press [s] or [l] on the keyboard for 'shorter' or 'longer'.

The classification function (percentage 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 jn difference at which each subject was 75% correct.

Loudness estimation was a non-cerebellar control task. This experiment followed exactly the same design as time estimation, except that all tones were 1000 Hz and 1000 ms and differed 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 level was ~67 dB SPL for the standard tone. Subjects had to respond whether the second tone was louder or softer than the first one, pressing [s] or [l]. The same fitting procedure as for time estimation was followed for the percentage of 'softer' responses.

Procedure to assess deviance

Since one of the goals of this study was to determine in which domains a given dyslexic individual did and did not show abnormal performance, it was necessary to adopt a criterion for deviance. A common procedure is to set a threshold at n standard deviations of the mean of the control group. However, there is of course some arbitrariness in the choice of the value of n, and no value has been used consistently in the literature.

In the present study we chose n = 1.65 SD. In a normal distribution, this corresponds to the fifth percentile, which seems a reasonable threshold for deviance. However, because a control subject may occasionally show abnormal performance in one task, there is a risk that the control mean and standard deviations might be skewed by such points of data, which might make the criterion more stringent than intended. For this reason, we applied the criterion in two steps: (i) compute the control mean and standard deviation and identify control subjects who qualify for abnormal performance according to the 1.65 SD criterion (typically, this applied to 0 or 1 control subject for each measure); (ii) recompute the control mean and standard deviation excluding these control subjects, and identify dyslexics who are outside ±1.65 SD.

The application of this procedure to the results described below seemed to confirm that it successfully identified those dyslexic subjects whose performance was outside the range of most of the controls.

Results

Psychometric tests

Results are presented in Table 1. The two groups were adequately matched for sex (eight males and eight females in each group), handedness (two controls and one dyslexic left-handed) and full-scale IQ. Dyslexics scored significantly higher on one performance subtest of the WAIS, picture completion [$F(1,30) = 6.1$, $P < 0.05$]. On the other hand, they scored significantly lower in verbal IQ [$F(1,30) = 6.5$, $P = 0.016$], which is directly attributable to their significantly lower scores in two verbal subtests of the WAIS: digit span [$F(1,30) = 21$, $P < 0.001$] and letter–number sequencing [$F(1,30) = 14.9$, $P = 0.001$]. Furthermore, they were marginally poorer at arithmetic [$F(1,30) = 3.6$, $P = 0.069$]. The straightforward reason for these lower scores is that these three subtests load heavily on verbal short-term memory, which is known to be affected in dyslexics as part of their phonological deficit (Brady *et al*., 1983). The three scores are

Table 2 *Reading and language tests (mean ± SD)*

	Reading***	Spelling***	NART (CR/50)***	Reading speed (syl./s)**	GNRT acc. (CR/20)***	GNRT RT (s)***	ASTOP (% CR)	TAPS (% CR)	LITERACY (average *Z*-score) ***
Controls (n = 16)	113.9 ± 4.5	115.3 ± 4.7	35.7 ± 5.3	2.49 ± 0.21	19.1 ± 0.93	1.68 ± 0.21	0.94 ± 0.04	0.77 ± 0.12	0 ± 0.62
Dyslexics (n = 16)	103.7 ± 5.9	95.6 ± 7.1	25.4 ± 6.2	2.01 ± 0.50	16.4 ± 2.09	2.62 ± 0.72	0.95 ± 0.03	0.78 ± 0.06	–2.98 ± 1.56

CR = correct response; syl. = syllable; acc. = accuracy; GNRT = Graded Nonword Reading Test; RT = reaction time; ASTOP = Advanced Syntactic Test of Pronominal reference; TAPS = Test of Active and Passive Sentences; NART = National Adult Reading Test. $^{**}P < 0.01$; $^{***}P < 0.001$.

Table 3 *Phonological tests (mean ± SD)*

	Picture naming (s)**	Digit naming (s)***	Spoonerisms acc. (CR /12)**	Spoonerisms RT (s)**	CNREP (% CR)*	PHONOLOGY (average *Z*-score)***
Controls (n = 16)	54.5 ± 7.0	27.8 ± 4.6	11.3 ± 0.87	4.45 ± 1.21	0.92 ± 0.05	0 ± 0.42
Dyslexics (n = 16)	68.4 ± 15.4	42.9 ± 12.5	8.5 ± 2.9	9.96 ± 5.88	0.86 ± 0.06	–2.6 ± 1.49

acc. = accuracy; CR = correct response; RT = reaction time; CNREP = Children's Test of Nonword Repetition. $^{*}P < 0.05$; $^{**}P < 0.01$; $^{***}P < 0.001$.

subsumed by the Working Memory Index (WMI) of the WAIS, which was therefore also significantly different between the two groups [$F(1,30) = 28$, $P < 0.001$]. In the rest of the analyses, the WMI will be taken as an additional measure of phonological performance, since it is a sensitive measure of the ability to accurately receive, retain, manipulate and reproduce phonological representations.

Table 2 shows that dyslexics were significantly poorer than controls in all measures of literacy: WRAT reading [$F(1,30) = 30$, $P < 0.001$], WRAT spelling [$F(1,30) = 85.5$, $P < 0.001$], NART [$F(1,30) = 25.4$, $P < 0.001$], reading speed [$F(1,30) = 12.7$, $P = 0.001$], non-word reading accuracy [$F(1,30) = 22$, $P < 0.001$] and non-word reading time [$F(1,30) = 24.7$, $P < 0.001$]. This last measure is the time taken to produce each non-word, measured from the onset of display of the non-word to the offset of the non-word produced. Times to produce erroneous responses were not excluded, as there was no speed–accuracy trade-off.

In order to summarize literacy performance for the purpose of deviance analysis, we converted the six relevant variables into *Z*-scores, and averaged these *Z*-scores to produce a single variable called LITERACY, also shown in Table 2. The deviance analysis on LITERACY found that all but one dyslexic subject (J.G.) and just two control subjects (K.B. and C.C.) showed abnormal performance (one control subject excluded from control statistics). Subject J.G. still was 1.3 SD below the control mean. His file mentioned more severe literacy difficulties at the age of 12, suggesting that his good performance was due to adequate teaching and successful compensation strategies. He was therefore not excluded from the dyslexic group.

The two groups did not differ significantly on the two syntax tests. A deviance analysis on the average of the two tests did not single out any dyslexic subject (one control subject excluded). However, closer examination of each individual's file revealed that one subject (F.H.) had had phonological difficulties as a child and consequently received speech therapy between ages 5 and 7 years. This suggests that he may have qualified for a diagnosis of SLI. This will be discussed further in the light of his other results.

The two groups did not differ significantly on the score obtained from the ADD questionnaire [$F(1,30) = 2.26$, $P = 0.14$]. However, six dyslexics and one control were found to have a *T*-score that was both deviant according to our criterion (one control excluded) and above 65, the threshold for clinical significance for *T*-scores. Higher scores for dyslexics in this questionnaire are not entirely surprising since five questions out of 40 concerned reading or writing, and three concerned verbal short-term memory. We recomputed the ADD scores after excluding these questions. Two dyslexics (J.C. and O.N.) and one control (M.M.) remained with deviant scores, and will therefore be considered as potentially presenting an additional attentional disorder.

Phonological tests

Table 3 shows that dyslexics were significantly poorer than controls in all phonological tests: rapid picture naming [$F(1,30) = 10.7$, $P = 0.003$], rapid digit naming [$F(1,30) = 20.5$, $P < 0.001$], spoonerisms in both accuracy [$F(1,30) = 7.5$, $P = 0.01$] and production time [$F(1,30) = 13.4$, $P = 0.001$], and non-word repetition [$F(1,30) = 7.5$, $P = 0.01$]. In order to assess whether the dyslexics' poor performance in automatic naming might have been due to overall slowness, we computed a covariance analysis with group as independent variable, digit naming as dependent

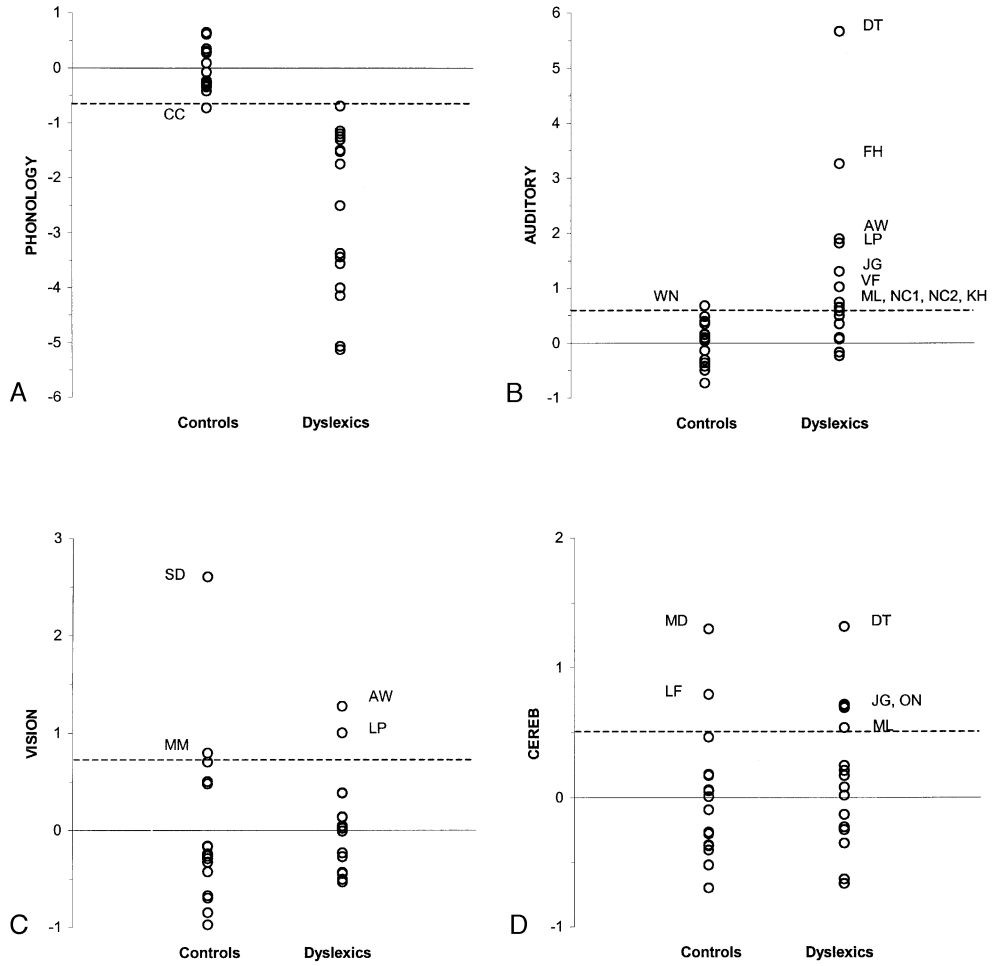

Fig. 2 Individual scores on summary factors for each domain. The solid line indicates the control mean and the dashed line the chosen deviance threshold (1.65 SD above the control mean after excluding deviant controls). Deviant individuals are identified, except for phonology, where all dyslexics are deviant. (**A**) phonology; (**B**) audition; (**C**) vision; (**D**) cerebellar function.

variable, and the Processing Speed Index of the WAIS as covariate (the Processing Speed Index summarizes performance on the symbol search and digit-symbol coding subtests). The Processing Speed Index effect was found to be significant [$F(1,29) = 7.1$, $P = 0.01$]. Nevertheless, the group effect was still highly significant [$F(1,29) = 21.5$, $P < 0.001$] even after differences in overall speed were taken into account. Similar results were obtained with picture naming [group effect, $F(1,29) = 10.9$, $P = 0.003$]. Poor performance in rapid automatic naming therefore reflects phonological difficulties beyond individual differences in overall speed.

In order to summarize phonological performance for individual analyses, we averaged the *Z*-scores of the first five variables in Table 3 plus the WMI. This new variable, PHONOLOGY, is also shown in Table 3, and individual scores are plotted in Fig. 2A. A deviance analysis on PHONOLOGY reveals that all dyslexics and one control (C.C.) have abnormal phonological performance (one control excluded). It can therefore be concluded that all the dyslexics in this sample suffer from a phonological deficit.

Auditory perception tests

Table 4 shows the results of the speech perception tests. For each subject we considered the average of the two thresholds measured per condition. Two values are given for syllable categorization results: the position of the boundary along the continuum and the jnd (the number of steps required for the categorization to shift from 50 to 75%). The jnd for the four-interval forced-choice *ba–da* discrimination is also given, together with that for the non-speech control condition.

The two groups did not differ significantly in any of the speech categorization tasks. However, there was a trend towards a difference for the *coat–goat* threshold [$F(1,30) = 3.5$, $P = 0.07$]; this was accounted for by five dyslexics who had inordinately high jnds, although they had phoneme boundaries within the control range.

For the *ba–da*/F2 discrimination task, there was no significant group difference, either in the speech or in the non-speech condition (with a single formant F2). A paired-samples *t*-test revealed that jnds were significantly lower in

Table 4 *Speech perception tests (mean ± SD)*

	ba–da jnd	*ba–da* boundary	*date–gate* jnd	*date–gate* boundary	*coat–goat* jnd	*coat–goat* boundary	*ba–da* discrimination jnd	*ba–da* F2 discrimination jnd
Controls (n = 16)	2.4 ± 1.4	30.5 ± 5.1	2 ± 1	37.5 ± 5.3	2.3 ± 0.8	27.5 ± 3.6	17.5 ± 4.6	15.6 ± 8.7
Dyslexics (n = 16)	3.1 ± 2.2	30.2 ± 4.5	2.4 ± 0.9	38.7 ± 3.9	3.3 ± 1.9	28.1 ± 4.2	19.6 ± 8.4	14.6 ± 6.6

Unit is the number of steps on the continuum (out of 41 for *ba–da* and out of 51 for *date–gate* and *coat–goat*).

Table 5 *Non-speech perception tests (mean ± SD)*

	Backward masking jnd (dB)	Simultaneous masking jnd (dB)	Absolute threshold jnd (dB)	FM detection jnd 2 Hz (modulation index)*	FM detection jnd 240 Hz (modulation index)	Temporal order (long) jnd (ms)**	Temporal order (short) jnd (ms)*
Controls (n = 16)	46.3 ± 10.1	76.3 ± 2.5	24.4 ± 5.4	1.01 ± 0.34	0.0042 ± 0.0027 (n = 14)	50.7 ± 24.7	34.7 ± 30.3
Dyslexics (n = 16)	53.3 ± 14	75.9 ± 4.2	26 ± 4.9	2.04 ± 1.97	0.0076 ± 0.0078	93 ± 53.5	106 ± 109

*$P < 0.05$; **$P < 0.01$. FM = frequency modulation.

Table 6 *Summary auditory variables (mean ± SD)*

	RAPID*	SLOW*	SPEECH*	NONSPEECH*	AUDITORY**
Controls (n = 16)	0 ± 0.44	0 ± 0.71	0 ± 0.51	0 ± 0.5	0 ± 0.38
Dyslexics (n = 16)	0.78 ± 1.07	1.53 ± 2.89	0.63 ± 0.82	1.13 ± 1.99	1.14 ± 1.5

*$P < 0.05$; **$P < 0.01$.

the non-speech than in the speech condition [$t(31) = 2.2$, $P = 0.035$], consistent with the reduced discriminability of speech stimuli within phoneme categories. A repeated measures analysis showed that this did not interact with the group factor [$F(1,30) < 1$].

Results of the non-speech tests are summarized in Table 5. For each subject we considered the median of the two to four thresholds measured per condition. The results of two control subjects in the FM 240 Hz task were rejected because of dysfunctional headphones. None of the control conditions (simultaneous masking, absolute thresholds, FM detection at 240 Hz) showed any significant group effect. There was a trend for a group difference in backward masking [$F(1,30) = 2.63$, $P = 0.11$], due to six dyslexics with thresholds >60 dB. There were significant group differences in FM detection at 2 Hz [$F(1,30) = 4.2$, $P = 0.048$] and in temporal order judgement with long stimuli [$F(1,30) = 8.26$, $P = 0.007$] and with short stimuli [$F(1,30) = 6.4$, $P = 0.017$]. In all conditions in which group differences were observed, they were attributable to the high thresholds of five to seven dyslexics.

There are several ways to assess the overall auditory performance of subjects in relation to dyslexia. According to the magnocellular theory, dyslexics should be poor at rapid auditory processing, i.e. tasks involving short sounds or fast transitions (Tallal *et al*., 1993). According to another view, those dyslexics who are impaired in the auditory modality are impaired only in tasks involving speech stimuli (as opposed to non-speech sounds) (Mody *et al*., 1997). In order to compare the two hypotheses, we computed several summary variables, which are presented in Table 6.

RAPID summarizes performance on all tasks involving short sounds or fast transitions; it is the average *Z*-score of *ba–da* jnd, *date–gate* jnd, *coat–goat* jnd, *ba–da* discrimination, *ba–da* F2 discrimination, backward masking, simultaneous masking, and temporal order judgement for short and long conditions (even in the long condition, stimulus onset asynchronies became short). Absolute threshold was not considered a rapid processing task, because when it was presented in isolation the short duration of the tone did not make it particularly difficult to detect. Neither was FM detection at 240 Hz, since at this frequency the modulations are not resolved by the auditory system. SLOW is the average *Z*-score of all the other jnds: absolute threshold and FM detection at 2 and 240 Hz.

SPEECH is the average *Z*-score of the tasks involving speech: the three syllable categorization tasks and *ba–da* discrimination. NONSPEECH is the average *Z*-score of all the other jnds: *ba–da* F2 discrimination, backward masking, simultaneous masking, absolute threshold, temporal order judgement for short and long conditions, and FM detection at 2 and 240 Hz.

Table 7 *Individual Z-scores for summary auditory variables for the dyslexic group and deviant controls*

	RAPID	SLOW	SPEECH	NONSPEECH	AUDITORY
Dyslexics					
A.J.		2.54			
A.W.	4.94		5.45	2.36	5.52
M.W.					
N.D.C.		3.20			1.95
S.M.			2.06		
K.H.			2.24		1.77
M.L.	2.42				2.23
D.M.					
V.F.	2.19		2.59		3.03
L.P.	4.22	5.12		5.26	5.31
F.H.	6.64	6.91	6.18	6.08	9.40
J.C.			4.21		
N.C.					1.98
O.N.					
D.T.	9.47	19.05	3.31	14.88	16.23
J.G.	1.89	5.70		3.59	3.83
Deviant controls					
M.D.		2.89		2.45	
R.G.			1.70		
W.N.	2.69		3.60		2.05
M.M.		2.08			

Only deviant values (>1.65) are shown.

Since some results in the literature are consistent neither with the rapid auditory processing theory nor with the speech-specific theory (e.g. poor performance on slowly varying non-speech sounds, Witton *et al*., 1998), we computed a more pragmatic variable, AUDITORY, summarizing all the tasks which (i) have shown poor performance in dyslexics in the literature, or (ii) should show poor performance in dyslexics according to at least one theory. This variable averaged the *Z*-scores of *ba–da* jnd, *date–gate* jnd, *coat–goat* jnd, *ba–da* discrimination, *ba–da* F2 discrimination, backward masking, temporal order judgement for short and long conditions, and FM detection at 2 Hz (i.e. the same as RAPID, without simultaneous masking and with FM 2 Hz).

All summary variables showed a significant group effect. A repeated measures analysis revealed no interaction between group and RAPID versus SLOW [$F(1,30) = 1.27$, $P = 0.27$], showing that dyslexics were not worse at tasks involving rapid auditory processing than at other tasks. Furthermore, a deviance analysis found abnormal performance in seven dyslexics and one control in RAPID and six dyslexics and one control in SLOW (one control excluded in each task). Overall, our results do not support the hypothesis that dyslexics are specifically impaired at rapid auditory processing. Similarly, there was no interaction between group and SPEECH versus NONSPEECH [$F(1,30) < 1$], showing that dyslexics were not worse at speech tasks than at non-speech tasks. A deviance analysis found abnormal performance in seven dyslexics and two controls in SPEECH (one control excluded) and five dyslexics in NONSPEECH. Our results therefore do not support the speech-specific hypothesis either.

The 'pragmatic' AUDITORY score showed the greatest difference between controls and dyslexics [$F(1,30) = 8.58$, $P = 0.006$], with 10 dyslexics out of 16 and one control showing abnormal performance (one control excluded). Individual scores are plotted in Fig. 2B. Unfortunately, no obvious construct seems to be able to capture what it is that all these auditory tasks have in common and that tasks such as simultaneous masking and FM detection at 240 Hz do not have. This remains true even if one considers only the most sensitive tasks, i.e. temporal order judgement for short and long conditions and FM 2 Hz. We therefore have to conclude, like Rosen and Manganari (2001), that an explanation for the auditory deficits observed in certain dyslexics has to be more sophisticated than just rapid auditory, or speech, processing.

This is further confirmed by looking at the individual scores for the summary auditory variables (Table 7). There seems to be no regularity whatsoever in the nature of the auditory deficits that dyslexics have. For instance, within the dyslexic group there are double dissociations between fast and slow auditory processing (A.W., M.L. and V.F. versus A.J. and N.D.C.), as well as between speech and non-speech perception (S.M., K.H., V.F. and J.C. versus L.P. and J.G.). Some dyslexics seem to have absolutely no auditory deficit (M.W., D.M., O.N.) and some have relatively focal problems (A.J., N.D.C., S.M.), while others are impaired across the board (F.H., D.T.).

In summary, we find that a significant proportion of dyslexics are impaired in the auditory domain. However, there is great heterogeneity in the nature of the problem.

Table 8 *Visual perception tests (mean ± SD)*

	Snellen acuity (m)	Contrast sensitivity magno (contrast)	Contrast sensitivity parvo (contrast)	Speed discrimination magno (% speed)	Speed discrimination parvo (% speed)	Coherent motion detection (% coherence)	VISION (average *Z*-score)
Controls (n = 16)	6/6.84 ± 1.2	1.45 ± 0.16	2.01 ± 0.23	140 ± 13	200 ± 84.5	60.4 ± 20.4	0 ± 0.87
Dyslexics (n = 15)	6/6.6 ± 1.14	1.48 ± 0.11	2.03 ± 0.15	146 ± 9.7	182 ± 26.5	54.7 ± 19.6	0.06 ± 0.53

Table 9 *Cerebellar tests (mean ± SD)*

	Bead threading (s)	Finger-to-thumb (s)	Time estimation jnd (ms)	Loudness estimation jnd (% amp.)	Repetitive finger-tapping IRI (ms)	BIMANUAL finger-tapping (average *Z*-score)	BALANCE (average *Z*-score)	CEREB (average *Z*-score)
Controls (n = 16)	40.8 ± 6.6	5.7 ± 1.3	84.3 ± 9.2	3.7 ± 0.14	186 ± 17	0 ± 0.74	0 ± 0.8	0 ± 0.51
Dyslexics (n = 16)	39.7 ± 3.4 (n = 13)	6.2 ± 1.5	81.8 ± 5.2	3.92 ± 0.66	194 ± 17	0.34 ± 1.08	–0.04 ± 0.8	0.15 ± 0.54

amp. = amplitude.

Depending on how one construes the auditory deficit, between seven and 10 dyslexics out of 16 were affected, compared with just one or two controls. Certain dyslexics, on the other hand, seemed to have entirely intact auditory abilities. This is consistent with all previous studies in which individual data have been examined. This conclusion holds even when using a far wider array of auditory tasks than in previous studies.

Visual perception tests

One dyslexic subject had to be excluded from this part of the study because he was blind in one eye. All subjects had a Snellen acuity above 6/9.3. Mean thresholds for the two groups are presented in Table 8. None of the variables showed a significant group effect (all P values >0.20).

In coherent motion detection, it appears that our subjects had much higher thresholds than in comparable published studies (e.g. Witton *et al*., 1998). The reason seems to be our use of smaller dots and the fact that the experiment was run under low-luminance conditions, both in the testing room and on the monitor. As the magnocellular system is particularly sensitive to low-luminance conditions, this should have increased the probability of observing magnocellular deficits. However, this increased the overall difficulty of the task so much that two subjects (one control, one dyslexic) were unable to perform it even at 100% coherence. This floor effect therefore prevents us from knowing whether some dyslexics were particularly impaired in this task. For this reason, this variable is not included in the deviance analysis.

We computed a summary variable, VISION, as the average *Z*-score of 'contrast sensitivity magno' and 'speed discrimination magno'. A deviance analysis on this variable found that just two dyslexics out of 15 and two controls had abnormal performance in the magnocellular conditions (one control excluded) (see individual data in Fig. 2C). This is consistent with previous studies in which individual data were examined; for instance, Cornelissen and colleagues found between five and 10 dyslexics out of 29 who were outside the range of most controls (Cornelissen *et al*., 1995), and Witton and colleagues found around four out of 17 (both in coherent motion) (Witton *et al*., 1998).

Cerebellar tests

Balance

The steps in which each subject counted backwards were determined so as to equate the difficulty of the tasks across the subjects. Among the controls, three counted in twos, nine in threes and four in sevens, while among the dyslexics, one counted in ones, 11 in twos and four in threes (χ^2 = 11.5, $P < 0.01$). This factor did not correlate with any measure of balance/dual task.

In each condition and measure, the three repeated measures per subject were averaged. There was a total of 12 variables across the four conditions (path lengths of the CoP and the C7 diode for the two conditions with arms alongside; CoP and C7 plus the two hands for the two conditions with arms extended). Means and standard deviations of all measures are summarized in supplementary material (http://www.lscp.net/persons/ramus/dyslexia02/supp.html). In order to assess group differences, a multivariate covariance analysis was performed with height and weight as covariates, as these factors might have had an influence on a subject's stability. In fact they did not have any significant effect on the measures. Furthermore, none of the measures was found to differ significantly between groups, even in the dual task condition [all $F(1,28) < 1$].

In order to summarize the balance results for further analyses, we averaged the *Z*-scores of these 12 variables into

Table 10 *Pearson correlations between summary variables across domains*

	LITERACY	PHONOLOGY	AUDITORY	VISION
PHONOLOGY	0.872***			
AUDITORY	–0.647***	–0.544**		
VISION	–0.108	–0.085	0.139	
CEREB	–0.048	–0.228	–0.35*	–0.316

*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$, no correction applied.

a single BALANCE score (Table 9). This new variable did not differ significantly between groups [$F(1,30) < 1$]. A deviance analysis found two dyslexics (O.N. and D.T.) and two controls with abnormal performance in BALANCE (one control excluded).

Bimanual finger-tapping

For each task, the mean and standard deviation of IRIs during the first 30 s (with metronome) and during the next 30 s (without metronome) are reported in supplementary material (http://www.lscp.net/persons/ramus/dyslexia02/supp.html). None of these variables showed any significant group effect. Following Wolff and colleagues (Wolff *et al*., 1990), we used only IRI standard deviations for subsequent analyses.

We summarized performance in finger-tapping by averaging the *Z*-scores of all IRI standard deviations to form the new variable BIMANUAL (Table 9). This variable did not differ significantly between the two groups [$F(1,30) = 1.1$, $P = 0.3$]. A deviance analysis found four dyslexics (M.L., V.F., N.C. and D.T.) and two controls with abnormal BIMANUAL scores (one control excluded).

Time/loudness estimation

In both tasks, the fit of the logistic regression was significant for all subjects (all P values <0.001). The jnds for duration and loudness differences were analysed. Neither variable showed a significant group difference. Two dyslexics (M.L. and V.F.) and two controls had abnormally high thresholds in time estimation (one control excluded). Curiously, five dyslexics (M.W., N.D.C., M.L., F.H. and D.T.) and one control were deviant on loudness estimation (one control excluded). Considering that M.L., V.F., F.H. and D.T. were already deviant on AUDIO, it seems that both time and loudness estimations tap some aspect of auditory function. Thus, it may not be very appropriate to interpret poor performance in time estimation as an indicator of cerebellar dysfunction.

Repetitive finger-tapping, finger-to-thumb and bead-threading

For bead-threading and finger-to-thumb, each task was performed twice, and only the best score was recorded. The bead-threading data for three dyslexic subjects were missing. Mean scores for each group are reported in Table 9. None of these cerebellar tests showed any significant difference between groups.

We computed a new variable, CEREB, averaging the *Z*-scores of all the cerebellar tests reported in Table 9 (including BALANCE and BIMANUAL but excluding loudness estimation, since this was only a control task). This variable did not differ significantly between the two groups [$F(1,30) < 1$]. A deviance analysis on this variable suggests that four dyslexics (M.L., O.N., D.T. and J.G.) and two controls had abnormal overall performance in the cerebellar tests (one control excluded) (see individual data in Fig. 2D). However, considering that just one of these four dyslexics (M.L.) was impaired in time estimation and that he was also impaired in loudness estimation and other auditory tasks, it is not quite clear whether the CEREB variable reflects cerebellar dysfunction at all, or whether it simply reflects some aspect of motor control. Similarly, only two dyslexics (O.N. and D.T.) had impaired balance, only one of them in the dual task (D.T.). This casts doubt on the idea of a general automaticity deficit. The present results are intermediate between reports of a high incidence (>50%) of motor/cerebellar disorders in dyslexics (Fawcett *et al*., 1996; Ramus *et al*., 2003) (but their criterion was ± 1 SD of the control mean) and reports of no such disorders (Wimmer *et al*., 1998; van Daal and van der Leij, 1999).

Further analyses

Relationship between auditory and phonological performance

The great heterogeneity of auditory performance observed in the dyslexic group, when compared with the relative homogeneity of the phonological deficit, would suggest that there is no meaningful relationship between the two domains. Yet there was a significant correlation between AUDITORY and PHONOLOGY ($r = –0.54$; $P = 0.001$) (Table 10). That is, AUDITORY accounted for 29.6% of the variance in PHONOLOGY. In order to be really meaningful, this correlation should hold within each group, since an overall correlation is predicted even without causation by virtue of the differences between the two groups along both dimensions. In fact, the correlation held within the control group ($r = –0.6$, $P = 0.01$) but not within the dyslexic group

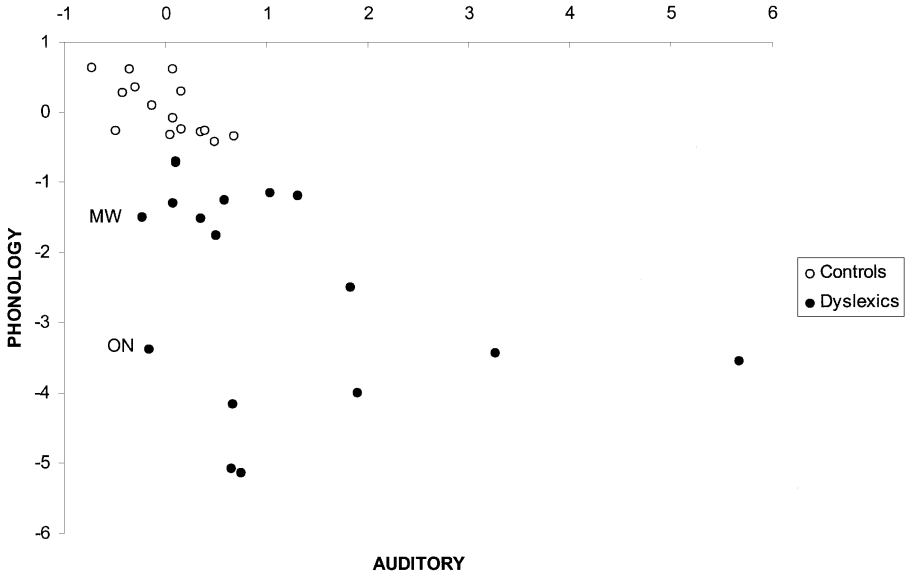

Fig. 3 Auditory versus phonological performance.

Table 11 *Pearson correlations between phonological and summary auditory scores.*

	RAPID	SLOW	SPEECH	NONSPEECH	AUDITORY
WMI	–0.269	–0.163	–0.331	–0.182	–0.289
Picture naming	0.345	0.548**	0.173	0.476**	0.488**
Digit naming	0.316	0.51**	0.157	0.44*	0.479**
Spoonerisms accuracy	–0.373*	–0.004	–0.386*	–0.153	–0.268
Spoonerisms RT	0.484**	0.242	0.344	0.363*	0.443*
CNREP	–0.361*	–0.144	–0.364*	–0.223	–0.352*

RT = reaction time; CNREP = Children's Test of Nonword Repetition. *$P < 0.05$; **$P < 0.01$, no correction applied.

(r = –0.3, P = 0.26), a rather surprising finding since the dyslexic group showed greater variability. The scatterplot (Fig. 3) seems to indicate that auditory performance does not really predict phonological performance, but rather that it places an upper limit on it. In other words, poor audition entails poor phonology, but the reverse is not true: some subjects had very poor phonology but excellent audition (e.g. O.N. and M.W.).

In order to further explore the relationship between auditory and phonological skills, we looked at the correlations between the phonological tasks and the summary auditory variables (Table 11). Obviously, the numbers in the present multiple case study do not allow powerful correlation analyses; indeed, if a Bonferroni correction were applied here, the only significant correlation would be between picture-naming and SLOW. Yet Table 11 provides interesting indications: that naming tasks seem to correlate with NONSPEECH and SLOW auditory processing, while spoonerism accuracy and non-word repetition correlate with SPEECH and RAPID auditory processing. (Note that the variables summarized in SPEECH are also included in RAPID, and those summarized in SLOW are also included in NONSPEECH, so these associations are expected by design.) Verbal short-term memory (WMI), on the other hand, does not seem to correlate reliably with any of the auditory variables, suggesting that some aspects of phonology might be less affected by auditory problems. If such a pattern of correlations were to be confirmed in future studies, it would suggest, interestingly, that different types of auditory deficits might affect different aspects of phonology.

It is worth noting that the correlation between SPEECH and spoonerisms and non-word repetition may arise for two different reasons. The straightforward interpretation is that speech perception skills have a developmental impact on phonological skills, as measured by spoonerisms and non-word repetition. But is also likely that, whether or not speech perception affects phonology, it affects performance in these particular tasks. Indeed, difficulties discriminating, say, between /b/ and /d/ must make it more difficult to correctly repeat a non-word containing /b/ or /d/, and likewise for spoonerisms. So a correlation between SPEECH (and therefore RAPID) and phonological tasks involving speech perception is expected, even in the absence of developmental causation. However, this reasoning does not generalize easily

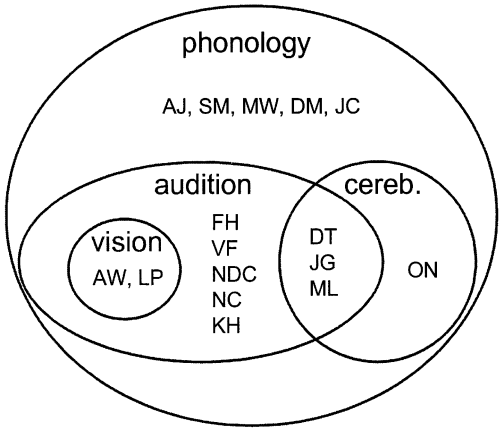

Fig. 4 Distribution of phonological, auditory, visual and cerebellar disorders in the sample of 16 dyslexic adults. Initials refer to individual dyslexic subjects.

to other correlations, e.g. between rapid automatic naming and SLOW.

In summary, the present results suggest that certain auditory deficits may act as aggravating factors for certain aspects of phonological performance, but do not seem strictly necessary for a phonological deficit to occur in the first place.

Role of vision and cerebellar function

CEREB was found to correlate weakly with AUDITORY ($r = 0.35$, $P = 0.05$), but this would not survive Bonferroni correction. Examination of the scatterplot suggests that the correlation is due to just one outlier (D.T., the worst performer in both domains), whose removal does indeed annihilate the effect ($r = 0.04$, $P = 0.84$). Therefore, CEREB does not seem to have any effect on the other variables. Neither does VISION (Table 10).

Overlap between the different disorders

Figure 4 summarizes the individual data across the different domains. As we have seen before, 16 dyslexics out of 16 had poor performance in phonology, 10 in audition, four in cerebellar function and two in magnocellular vision. There is some overlap between cerebellar and auditory disorders. In the present sample, as also reported by Witton and colleagues (Witton *et al*., 1998), visual disorders were confined to a subset of the auditorily affected dyslexics. Finally, five dyslexics seemed to be entirely unaffected by any sensory or motor/cerebellar disorder, i.e. they seemed to have a purely phonological dyslexia.

Predictors of literacy

The fact that five of the dyslexics seemed to have a phonological deficit without any sensory or motor disorder suggests that a pure phonological deficit is sufficient to cause a reading impairment. The question therefore arises whether the sensory or motor disorders observed in some individuals make an additional contribution to reading problems.

This question was investigated by running a stepwise multiple linear regression of LITERACY on PHONOLOGY, AUDITION, VISION and CEREB. The main predictor by far was PHONOLOGY, accounting for 76.1% of the variance [$F(1,30) = 95.4$, $P < 0.001$]. AUDITORY was found to account for an additional 4.2% of the variance [$F(1,29) = 6.2$, $P = 0.02$] (when entered first, AUDITORY accounted for 41.8% of the variance). Finally, CEREB was found to account for an additional 4.8% of the variance [$F(1,28) = 9.1$, $P = 0.005$]. However, the coefficient of CEREB does not have the predicted sign; indeed, the greater (poorer) the CEREB score, the greater (better) the LITERACY residuals. We see no explanation of this relationship other than chance. We therefore conclude that CEREB does not actually contribute to the variance in LITERACY. VISION was not a significant predictor in the regression, contrary to the hypothesis that visual problems might be an additional factor of reading impairment.

How might auditory performance affect literacy, in addition to its impact on phonological performance? One possible link is via spelling. Indeed, WRAT spelling was the only literacy task that involved speech perception. Therefore, speech perception problems may affect the spelling of unknown words (non-words, for practical purposes). One would then expect that spelling is the literacy task that AUDITORY is most correlated with. This was indeed the case ($r = –0.609$, $P < 0.001$), although by very little (with WRAT reading: $r = –0.607$, $P < 0.001$). Of course, reading and spelling are themselves highly correlated ($r = 0.82$, $P < 0.001$), so little difference could be expected. The only other direct link we can think of between audition and literacy is that all the reading tasks involve speaking aloud, which itself may require auditory feedback for efficient self-correction. Presumably these two weak links are sufficient to explain the 4.2% of additional variance.

Possible role of additional developmental disorders

As we recalled in the Introduction, some researchers have proposed that auditory and motor/cerebellar deficits are found only in dyslexics who have an additional developmental disorder—SLI and ADHD respectively. In the present study, we specifically tried to avoid such comorbid cases. However, two dyslexics had abnormally high scores on the ADD questionnaire: J.C. and O.N. O.N. happens also to be an outlier on the CEREB variable, but J.C. seems to be a pure phonological case. We have also mentioned earlier that, according to his file, subject F.H. might be a case of mild SLI. He happens to have had the second worst AUDITORY score. No other indication of any additional developmental disorder

was found in the present sample. Our results suggest that, although comorbid developmental disorders may increase the likelihood of observing sensory/motor disorders in dyslexic individuals, this is not the whole story. A good number of our subjects have sensory or motor problems without having any sign or history of SLI or ADHD (for a similar conclusion see also Ramus *et al*., 2003).

Discussion

As in most previous studies of dyslexia, we found that the most significant cognitive problem of dyslexic individuals lies in phonological skills. Our analysis of individual data even shows that all the dyslexics in our sample were so affected. Obviously, this does not preclude the existence of reading-impaired people whose problem is not phonological. It remains perfectly possible that other, less frequent disorders can provoke reading impairments entirely independently of phonology; this might be the case in visual stress, for instance (Wilkins, 1995).

We found that a significant number of dyslexics in our sample (10 out of 16) had auditory problems. This is a rather higher incidence than in previous studies, where it ranged from 0 to 50%, with typically one-third of dyslexics affected. Previous studies are actually consistent with the results we obtained on any particular auditory task considered separately. The higher incidence found here results from the administration of a greater number of tasks than in any previous study (12 measures per individual) and from the compounding of all the relevant variables to make a more sensitive measure of auditory performance. However, it is not the case that these auditory problems can be characterized as a rapid auditory processing deficit, as predicted by the magnocellular theory, and neither is it the case that they can be reduced to a speech perception deficit; actually, no coherent construct seems to be able to characterize the patterns observed. Rather, it seems that, within each individual, the pattern of good and poor auditory performance is more or less random, and this pattern varies considerably across subjects. Nevertheless, auditory performance does have a significant impact on phonological skills, accounting for 30% of the variance. In other words, dyslexics who have an auditory impairment have, to a certain extent, an aggravated phonological deficit.

As a speculation, we mention an alternative, perhaps more parsimonious possibility: that the scattered auditory problems would be due to a failure in top-down processes. Indeed, phonological processes might provide top-down control through expectancies that enhance low-level auditory perception. At least in the visual domain, the ubiquity of such top-down enhancement in sensory hierarchies is increasingly demonstrated in single-cell recordings and brain imaging studies (Friston and Büchel, 2000; Lamme and Roelfsema, 2000; O'Connor *et al*., 2002).

We also found that motor problems were present in certain dyslexics (four out of 16), even in the absence of measurable comorbid ADHD. However, the results obtained on time estimation and the balance/dual task do not militate in favour of a cerebellar origin or a general automaticity deficit (this is consistent with Stringer and Stanovich, 1998; Wimmer *et al*., 1998; Ramus *et al*., 2003). Finally, our data raise the question whether motor problems play any causal role in dyslexia. Contrary to the predictions of the cerebellar theory, we found no influence of motor/cerebellar performance either on phonology or on literacy. This might be due to the low prevalence of motor/cerebellar problems in the present sample (four out of 16), but this is consistent with another study in which the prevalence was higher (Ramus *et al*., 2003).

Only two of the dyslexics in our sample seemed to have visual problems of a magnocellular nature. This is in line with other studies in which individual data also showed a relatively low incidence of visual deficits. This low incidence, together with the fact that the two visually impaired dyslexics also have auditory and phonological problems, makes it impossible, using the present data, to assess whether visual performance may have an independent contribution to reading impairment.

The generalizability of the present study may be intrinsically limited by the particularities of the population studied, which is not representative in several respects: sex, achievement and age. Because we selected an equal number of males and females, whereas dyslexia is thought to be more frequent in males, one could argue that our sample was biased towards the female pattern, which may be a milder form of dyslexia. To test this hypothesis, we ran analyses of variance with sex and group as independent variables and LITERACY, PHONOLOGY, AUDITORY, VISION and CEREB as dependent variables. We found no main effect of sex on any of the variables (all P values >0.10), and a significant sex $\times$ group interaction only on CEREB [$F(1,27) = 5.5$, $P = 0.027$], revealing that males were more impaired than females in the dyslexic group but not in the control group. Therefore, our sex ratio may have led us to slightly underestimate motor problems in the dyslexic group compared with the general dyslexic population.

Having selected high-achieving adult dyslexics is another obvious source of bias, which may have decreased the incidence of each disorder and the overlap between disorders. This implies again that the incidence reported for each disorder in the present sample is not to be generalized to the whole dyslexic population. At this stage, it should be recalled that the main goal of this study was not to establish the respective incidence of the different deficits associated with dyslexia, but to assess the extent to which they were associated or could be dissociated. In this respect, we found that motor difficulties seem dissociable from auditory and visual deficits, and, most importantly, that a phonological deficit can arise in the absence of auditory, visual and motor impairments.

These conclusions might be moderated by the age bias: indeed, it is in principle conceivable that sensory and motor

impairments are always present in dyslexic children, and that they somehow disappear through development in certain individuals. If this were the case, our cases of pure phonological dyslexia might just be an illusion due to sensory-motor recovery. How likely is this possibility? Most studies supporting the magnocellular theory have been run on adults (because of the constraints of psychophysical tasks), with positive findings and no suggestion that they might be more positive in children. Conversely, many negative findings of auditory or visual deficits were from studies on children. Finally, a recent study of dyslexic children aimed at replicating the present study without the sex, age and achievement biases has found similar results, i.e. a limited incidence of sensory and motor disorders, with cases of pure phonological deficits (S. White, E. Milne, S. Rosen, P. C. Hansen, J. Swettenham, U. Frith, F. Rasmus, unpublished results). Thus, it appears that sensory-motor deficits do not play a greater role in explaining dyslexia in children than they do in adults. Of course, it remains possible that auditory or motor deficits act much earlier in infancy, setting phonological acquisition off-track, then recovering in most cases before school age (note that this is not a plausible scenario for visual deficits, since if they recovered before school age, little impact would be expected on reading). Such a hypothesis can only be tested in longitudinal studies starting at birth. Differences in auditory and speech perception between at-risk and control infants have indeed been documented (Leppanen *et al*., 1999; Pihko *et al*., 1999; Molfese, 2000; Guttorm *et al*., 2001; Richardson *et al*., 2003). However, methodological limitations have made it impossible to consider infants' individual performance, and therefore these studies cannot address the possibility that some dyslexic infants have intact auditory processing. Nevertheless, the twin study of SLI children conducted by Bishop and colleagues (Bishop *et al*., 1999) suggests that phonological deficits (assessed by non-word repetition) have a largely genetic origin, while auditory deficits (assessed by Tallal's repetition test) have not, and instead may be due to environmental influences. If this is to be extrapolated to dyslexia and to other measures of phonological and auditory processing, it may well be the case that auditory disorders are not necessary for a phonological deficit to arise.

Conclusion

The results of the present study support the phonological deficit theory of developmental dyslexia. A phonological deficit may not be a necessary cause of dyslexia, given the possibility of other independent (but rare) causes of reading impairment, but the present comprehensive study suggests that it is a sufficient cause. The phonological deficit can arise independently of any sensory or motor impairment. Nevertheless, a significant proportion of dyslexics suffer from additional auditory, visual or motor disorders. Auditory deficits, at least, may aggravate the phonological deficit, with consequences for reading impairment. The nature of the auditory deficits observed is not particularly consistent with the hypothesis of a rapid processing deficit related to a magnocellular dysfunction. Neither is the nature of motor/timing impairments particularly consistent with the hypothesis of an automaticity deficit or a cerebellar dysfunction. The nature of the phonological deficit and its relationship to auditory processing difficulties remains to be established. Why sensory and motor disorders are frequently associated with phonological deficits (and other developmental disorders) is still to be understood.

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