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: the phonological, the magnocellular (auditory and visual) and the cerebellar theories. 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, 4 from a motor deficit, and 2 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 5 of the dyslexics. Auditory disorders, when present, aggravate the phonological deficit, hence the literacy impairment. However, auditory deficits cannot be characterised 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; phonology; audition; vision; magnocellular function; motor control; cerebellum.

Abbreviations: ADHD = attention deficit/hyperactivity disorder; DCD = developmental coordination disorder; IRI = inter response interval; jnd = just noticeable difference; SLI = specific language impairment.

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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 (Grigorenko, 2001). The disorder has life-long persistence, with 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.

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 those sounds are poorly represented, stored, or retrieved, learning of grapheme-phoneme correspondences, the foundation of reading for alphabetic systems, will be accordingly affected (Bradley and Bryant, 1978; Brady and Shankweiler, 1991; Snowling, 1981; Vellutino, 1979). 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 peri-sylvian 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 and speech sounds. However, evidence for poor verbal short-term memory and slow automatic naming in dyslexics also points at 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 also support the notion of a left peri-sylvian dysfunction as a basis for the phonological deficit (Brunswick et al., 1999; McCrory et al., 2000; Paulesu et al., 2001; Paulesu et al., 1996; Pugh et al., 2000; Shaywitz et al., 2002; Shaywitz et al., 1998).

In order to better differentiate the phonological theory from the others, we only discuss here the strong version of the theory: that is, one that contends that the cognitive deficit is specific to phonology. Indeed, challengers of the phonological theory don't 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 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 indeed the claim of *the rapid auditory processing theory*, which specifies moreover 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: tone discrimination, temporal order judgement, backward masking... 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, like in /ba/ versus /da/. There is indeed also evidence that dyslexics may have poorer categorical perception of certain contrasts (Adlard and Hazan, 1998). In this view, the auditory deficit is therefore the direct cause, in the course of development, of the phonological deficit, hence of the difficulties 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 (Livingstone et al., 1991; Lovegrove et al., 1980; 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. The visual theory makes its central claim at the biological level. It is based on the division of the visual system into two distinct pathways that have different roles and properties: the magnocellular and the 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 (Stein and Walsh, 1997) . Evidence for the theory 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 (Cornelissen et al., 1995; Lovegrove et al., 1980), as well as unstable binocular fixations (Eden et al., 1994; Stein and Fowler, 1993). The visual theory does not exclude a phonological deficit, but emphasises an additional visual contribution to reading problems, at least in some dyslexic individuals.

Yet another view is represented by *the automaticity/cerebellar theory* of dyslexia (Nicolson and Fawcett, 1990; Nicolson et al., 2001) (henceforth, the cerebellar theory). Here the biological claim is that dyslexics' cerebellum would be 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. Second, the cerebellum plays a role in the automatisation of overlearned tasks, such as driving, typing, reading... A weak capacity to automatise would affect, among other things, the learning of grapheme-phoneme correspondences. Support for the cerebellar theory comes from evidence of poor dyslexic performance in a large number of motor tasks (Fawcett et al., 1996), in dual tasks demonstrating impaired automatisation 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 dyslexics' cerebellum (Brown et al., 2001; Leonard et al., 2001; Nicolson et al., 1999; Rae et al., 1998).

Finally, there is a unifying theory, that attempts to integrate all the findings mentioned above. A generalisation of the visual theory, *the magnocellular theory* (Stein and Walsh, 1997) postulates that the magnocellular dysfunction is not restricted to the visual pathways, but generalised to all modalities (visual, 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. Beyond the evidence pertaining to each of the previously described theories, evidence specifically relevant to the magnocellular theory include magnocellular abnormalities in the medial as well as the lateral geniculate nucleus of dyslexics' brains, 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 (van Ingelghem et al., 2001; Witton et al., 1998).

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 as independent. Rather, we will restrict the discussion to a comparison between the phonological, the cerebellar and the 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 those 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 (see Liberman and Mattingly, 1985; Ramus et al., in press, for a discussion). Finally, it remains uncertain what proportion of dyslexics are affected by motor problems. A number of studies have failed to find any (Kronbichler et al., 2002; van Daal and van der Leij, 1999; Wimmer et al., 1998), others have found motor problems only in a subgroup of dyslexics (Ramus et al., in press; Yap and van der Leij, 1994), 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 the recent years (e.g., Ramus, 2001). One line of criticism emphasises 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 (Adlard and Hazan, 1998; Lorenzi et al., 2000; Manis et al., 1997; Marshall et al., 2001; Mody et al., 1997; Reed, 1989; Rosen and Manganari, 2001; Tallal, 1980). 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 (Adlard and Hazan, 1998; Lorenzi et al., 2000; McAnally and Stein, 1996; Nittrouer, 1999; Reed, 1989; Rosen and Manganari, 2001; Schulte-Körne et al., 1998b; Witton et al., 1998). It is also argued that auditory deficits do not predict phonological deficits (Bishop et al., 1999; Marshall et al., 2001; Mody et al., 1997; Rosen and Manganari, 2001; Schulte-Körne et al., 1998a). Finally, some authors suspect that auditory deficits are restricted to those dyslexic individuals who also have additional language impairments (Heath et al., 1999; McArthur and Hogben, 2001). Criticism of the visual side of the magnocellular theory also focuses on failures to replicate findings of a visual deficit (Johannes et

al., 1996; Victor et al., 1993) and on inconsistencies between predictions and empirical results; most notably, visual impairments, when found, seem to be observed across the whole range of spatial frequencies, not just that characteristic of the magnocellular system (Farrag et al., 2002; Skottun, 2000).

In summary, the phonological theory suffers from its inability to explain sensory and motor disorders 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, with each 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, that is, 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 on frequency modulation detection at 2 Hz and coherent motion detection. The individual data reported suggests that 4 dyslexics out of 17 had abnormal performance in the visual task, 9 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 7 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 et al. (2001) tested both visual and auditory gap detection in dyslexic children and found significant group effects for both. They report that 9 dyslexics out of 10 were impaired in the auditory task and 7 out of 10 in the visual task. However, their criterion for being impaired is that the individual's threshold be above the 95% confidence interval for the control group, that is, for 10 individuals, beyond about .67 standard deviation above the control mean. This makes it an extremely liberal criterion, since if the control group is normally distributed, about 25% of the controls should also meet it (individual control data not available). Again, cerebellar and phonological performance were not tested. This study is also potentially undermined by the fact that the two groups were not matched in non-verbal IQ, a factor that is known to significantly affect performance 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). These authors administered a battery of phonological tests, 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 is reported to assess whether *some* dyslexics could have sensory or motor disorders, and the relationships between the variables are not analysed. Finally, in all three studies, only one task for each modality was administered, leaving open the possibility that other, more sensitive tasks, might significantly change the picture.

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 is that of a multiple case study: by having the most comprehensive neuropsychological profile for each individual, we may be able to identify who has which combination of disorders and, crucially, who *does not* have a given disorder. We have therefore created a battery of psychometric, phonological, auditory, visual and cerebellar tests, to be administered to each subject. Within each domain, we have 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, socially privileged, and may have received better help with respect to reading. Most importantly, we hypothesised 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 with just one of them. By studying a high-achieving population, we therefore maximise our chances of finding pure cases of the different possible subtypes of dyslexia. For the same reason, we also minimise the chances of studying individuals with another co-morbid developmental disorder (SLI, ADHD, DCD (developmental coordination disorder)), which would be an undesirable confound.

Methods

Subjects

Seventeen dyslexic university students at 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, DCD or autism. Additional inclusion criteria were checked after a first testing session: a full-scale IQ above 100, and reading and spelling standard scores below 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 above 100, reading and spelling scores above 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, thereby reducing the sample to 16.

It was checked a posteriori that the two groups were overall matched 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.

Test battery

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

Psychometric tests

Verbal and non-verbal intelligence were assessed using the Wechsler Adult Intelligence Scale (WAIS-IIIUK; Wechsler, 1998). Reading and spelling were assessed using the Wide Range Achievement Test (WRAT3; Wilkinson, 1993), the National Adult Reading Test, concentrating mainly on rare and irregular words (NART; Nelson, 1991), 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 were previously shown to be sensitive to subtle impairments of syntax in SLI children and adolescents (van der Lely, 1996a; 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 respond whether the sentence described the picture. The 96 items in this test assessed the understanding of pronominal reference and quantifiers in embedded phrases (like: “Minnie the Minx says every dancer is pinching herself”).

Test of active and passive sentences (TAPS) (van der Lely, 1996b): A sentence was played through headphones by a computer and four pictures were displayed at the same time. The subject had to point at the picture best described by the sentence. The 48 items assessed the correct computation of agent/patient relationships in active and passive sentences (like: “The car is hit by the lorry”).

To check on the possible presence of Attention Deficit Disorder, each subject completed the Brown ADD questionnaire (Brown, 1996).

Phonological tests

Automatic picture naming: The subject was to name 50 pictures of four objects (hat, ball, 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: same as above, but with 2 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 (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 CNRep (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 . Masked thresholds and the syllable/formant discrimination task were run using special-purpose psychoacoustic hardware and Senheiser 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 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 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 & 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 SPL) were presented with a 340-ms inter-stimulus interval. Along with one of the noise bursts occurred the 20-ms 1-kHz sinusoidal probe tone. 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 4 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 nonspeech analogues: The ability of subjects to discriminate second-formant transitions in speech and nonspeech sounds was assessed using the software package described by Carrell et al. (1999).

A /ba/-/da/ continuum and the corresponding nonspeech 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 *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. Nonspeech 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 nonspeech stimuli.

The discrimination task was based on four-interval two-alternative forced-choice same/different (4IAX) 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/ (or its nonspeech analogue). 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/ (or its nonspeech analogue), but an adaptive procedure chooses the comparison stimulus so as to estimate the stimulus which is discriminable from the /ba/ 69% of the time.

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

Phonemic categorisation: Categorisation 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) synthesiser. Unlike the /ba/-/da/ continnum, these were modeled closely on a particular speaker's tokens (an adult female speaker of standard Southern British English). They are much more complex than typical formant-synthesised 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 (VOT) in 1 ms steps (the first formant onset frequency covaried with VOT 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 & Rosen (2001), were used to estimate the points on the continuum at which the stimuli were labeled as one word of the pair (*e.g.*, 'coat') 29% and 71% of the time. The procedure terminated when there were a total of 5 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 3 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 categorisation 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 Hz and 240 Hz: Stimuli were modelled closely on those used by Talcott et al. (2000). Each trial consisted of two 1-s tone bursts (20 ms rise/fall times) separated by an inter-stimulus 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 Hz and 240 Hz. For each modulation frequency, a continuum of 100 stimuli was constructed spanning a wide range of the value 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 Hz and 5.94 Hz, respectively, for the two continua. Stimuli were presented over headphones at a level of about 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 form of appropriate pictures (a happy face for correct responses and a sad face for incorrect ones). Probit analysis was used to fit cumulative gaussians to the psychometric functions, so as to obtain an estimate of the modulation that was detectable 75% of the time.

Temporal order judgement (TOJ) of long and short sounds: The TOJ task was based on two sounds, readily identifiable without prior training as a car horn (periodic with a fundamental frequency of about 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 normalised to have the same rms level. The continuum of sounds consisted of 204 stimuli in which the stimulus onset asynchrony (SOA) 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 SOAs. 'Short' sounds were the same stimuli cut to 30 ms duration, thus minimising stimulus overlap at short SOAs, at the expense of less distinctive sound qualities. For testing, the same adaptive procedure and data analysis was 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

(NB: a more detailed description of those 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 Ltd). Experiments were run under the MatLab programming environment (Mathworks Ltd). 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" Sony Trinitron CRT monitor operating at a screen resolution of 1024 X 768 pixels with a frame refresh rate of 85Hz. 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 minutes 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 four-alternative forced choice (4AFC) 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 envelope (σ=200 ms.) to minimise 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 = 6/6*MAR).

Contrast sensitivity magno vs. parvo

Perhaps the most direct way to assess M- versus 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) (but see Skottun, 2000 for a critical review and ; Stuart et al., 2001 for other objections). 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 deg.; see Figure 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/deg) and counter-phase flickered at a rate of 15 reversals per second, while parvocellular-selective (P-selective) stimuli had a peak spatial frequency of 8.0 c/deg 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/deg; Legge, 1978). To further target the magnocellular pathway we followed Demb et al. (1998) in making M-selective stimuli *low-luminance*, since it is known that M-pathway response is dominant at mesopic/scotopic light levels (Lee, Smith, Pokorny and Kremers 1997; Purpura, Kaplan and Shapley 1988). M-selective stimuli therefore had a mean luminance of 5 cd/m^2 (i.e. 0-10cd/m^2)

while P-selective stimuli varied around a mean luminance of 40 cd/m^2 (i.e. 0-80cd/m^2). Stimulus duration was 500ms. In order to minimise 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.).

Figure 1 here

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 (2AFC). The onset of each interval was indicated by an auditory cue, and intervals were separated by a 500ms ISI. Contrast detection thresholds are presented as percent Michelson contrast [= (L_{min}-L_{max})/(L_{min}+L_{max}) where L_{min} and L_{max} are the luminance of the darkest and brightest parts of the display respectively (in cd/m^2)].

Speed discrimination magno vs. parvo

There is evidence that while poorer contrast sensitivity for M-selective stimuli may not reliably co-occur with dyslexia, poor speed discrimination might (Demb et al., 1998; Eden et al., 1996). We measured speed discrimination using versions of the stimuli similar we used to probe contrast detection (described in the last section) but with drifting carriers. The P and M-selective stimuli were tested with reference speeds of 1.0 and 16.0 deg/sec., and contrasts of 20% and 80% respectively. Speeds were selected both to target transient and sustained mechanisms, but also to produce equivalent temporal frequencies in terms of carrier-cycles per second (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 uniformly randomly varied 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 the 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 500ms 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; Everatt et al., 1999; Raymond and Sorenson, 1998; Slaghuis and Ryan, 1999; Talcott et al., 2000; Witton et al., 1998) 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 et al. (1998) for generating stimuli. Subjects were presented with an 8° X 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 deg./sec) to the left or the right. Stimulus movies lasted for 900ms and consisted of 19 distinct frames. Dots appeared for a maximum of 4 movie-frames before being randomly replaced (*limited life-time 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 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 those 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: subjects' static balance was assessed in four different conditions of increasing difficulty: eyes open, feet apart; eyes closed, feet together; eyes closed, feet together, arms extended; eyes closed, feet together, arms extended and counting backwards. This last condition was directly inspired from Nicolson & Fawcett (1990), according to which 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 (due to phonological problems), the difficulty of the task was calibrated as in Nicolson & Fawcett (1990): prior to the test session, the speed with which each subject was able to count backward in 3s was measured and used to determine the steps in which they should count during the balance dual task: in 1s, in 2s, in 3s or in 7s. For 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 twelve resulting trials was counterbalanced across two groups of subjects.

In order to assess 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, Leicestershire, U.K.), which tracked in three dimensions infrared-emitting diodes (IRED) attached to anatomical landmarks. Movements at the level of the neck and wrists were obtained from IREDs 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 AG, CH-8408 Winterthur, Switzerland). The force plate data were low-pass filtered (50 Hz cut-off frequency) before digitisation. All data were sampled at 100 Hz .Body stability was assessed by calculating the total distance (path length) travelled by each IRED 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 ten 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 is 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 anti-clockwise until the finger and thumb touched again, and so on. The task was demonstrated and subjects trained until they completed the movement fluently 5 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 is drawn from the Dow and Moruzzi (1958) battery and was administered as in 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 duration between two presses. This task is adapted from Denckla et al. (1985).

Bi-manual finger tapping: Bi-manual finger tapping in synchrony with a metronome was recorded in three different conditions: left and right hand alternately at 2 Hz; left and right hand alternately at 5 Hz; asymmetric 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 seconds in synchrony with the metronome, then the metronome stopped and they had to continue for 30 seconds at exactly the same pace. Subjects had to rest their hands on the table and move only the index fingers at the metacarpo-phalangeal joint. The metronome sound was produced by a computer which also recorded subjects' responses through a response box. Dependent measures are average inter-response interval (IRI) and its standard deviation. Previous work suggested that adult dyslexics would show greater IRI variability on the fast (5 Hz) and asymmetric conditions (Wolff, 1993; Wolff et al., 1990).

Time vs. loudness estimation: Time estimation is the only cerebellar task not involving motor control, and is therefore crucial to distinguish the cerebellar hypothesis from a solely motor one. We used exactly the same task as Nicolson et al. (1995), which was itself inspired by Ivry & 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 one. The standard stimulus, always presented first, was a 1200 ms-long pure tone of frequency 392 Hz. Twenty-two comparison tones had respective durations of 400, 700, 800, 900, 950, 1000, 1050, 1100, 1140, 1160, 1180, 1220, 1240, 1260, 1300, 1350, 1400, 1450, 1500, 1600, 1700 and 2000 ms. The two tones were separated by a 1000-ms silence interval. Each trial was repeated three times, therefore amounting to 66 test trials, presented in a random order. The test block was preceded by a practice block of 8 trials (using only the 8 extreme comparison tones), during which feedback was provided. No feedback was provided during the test block. The stimuli were presented by a computer through headphones at about 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 (% 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 just noticeable duration 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 long 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 sound pressure level was around 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 with the percentage of "softer" response.

Procedure to assess deviance

Since one of the goals of this study is to determine in which domains a given dyslexic individual does and does 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 beyond the mean of the control group, but there is of course some arbitrariness to the choice of the value of *n*, and no value has been consistently used in the literature.

In the present study we chose n=1.65 St.Dev. In a normal distribution, this corresponds to the 5th 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:

- First, compute the control mean and standard deviation and identify control subjects who qualify for abnormal performance according to the 1.65 St.Dev. criterion (typically, this applied to 0 or 1 control subject in each measure).
- Then, recompute the control mean and standard deviation excluding those control subjects, and identify dyslexics who are beyond 1.65 St.Dev.

The application of this procedure on 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 on sex (8 males and 8 females in each group), handedness (2 controls and 1 dyslexic left-handed) and full-scale IQ. Dyslexics scored significantly higher on one performance subtest of the WAIS, picture completion ($F(1,30)=6.1$, $p<.05$). On the other hand, they scored significantly lower in verbal IQ ($F(1,30)=6.5$, $p=.016$), which is directly attributable to their significantly lower scores in two verbal subtests of the WAIS: digit span ($F(1,30)= 21$, $p<.001$) and letter-number sequencing ($F(1,30)=14.9$, $p=.001$). Furthermore, they were marginally poorer at arithmetic ($F(1,30)=3.6$, $p=.069$). 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 subsumed by the Working Memory Index (WMI) of the WAIS, which is therefore also significantly different between the two groups ($F(1,30)=28$, $p<.001$). 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.

Tables 1 and 2 here

Table 2 shows that dyslexics were significantly poorer than controls in all measures of literacy: WRAT reading ($F(1,30)=30$, $p<.001$), WRAT spelling ($F(1,30)=85.5$, $p<.001$), NART ($F(1,30)=25.4$, $p<.001$), reading speed ($F(1,30)=12.7$, $p=.001$), non-word reading accuracy ($F(1,30)=22$, $p<.001$) and non-word reading time ($F(1,30)=24.7$, $p<.001$). The latter 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 summarise literacy performance for the purpose of deviance analysis, we converted the 6 relevant variables into z-scores, and averaged those 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 (JG) and just two controls (KB and CC) subjects showed abnormal performance (one control subject excluded from control statistics). Subject JG still was 1.3 St.Dev. 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 (TAPS and ASTOP). 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, FH, had phonological difficulties as a child and consequently received speech therapy between ages 5 and 7. This would suggest that he may have qualified for a diagnosis of SLI. This will be further discussed in the light of his other results.

The two groups did not differ significantly on the score obtained from the ADD questionnaire ($F(1,30)=2.26$, $p=.14$). However, 6 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 5 questions out of 40 concern reading or writing, and 3 concern verbal short-term memory. We recomputed the ADD scores after excluding those questions. Two dyslexics (JC and ON) and one control (MM) 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 dyslexics' poor performance in automatic naming might be due to overall slowness, we computed a covariance analysis with group as independent variable, digit naming as dependent variable, and the Processing Speed Index of the WAIS as covariate (the PSI summarises performance on the symbol search and digit-symbol coding subtests). The PSI effect was found to be significant ($F(1,29)=7.1$, $p=0.01$). Nevertheless, the group effect still was 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.

Table 3 here

In order to summarise 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 just one control (CC) have abnormal phonological performance (one control excluded). It can therefore be concluded that all the dyslexics in this sample suffer from a phonological deficit.

Figure 2 here

Auditory perception tests

Table 4 shows the results of the speech perception tests. For each subject we considered the average of the 2 thresholds measured per condition. Two values are given for syllable categorisation results: the position of the boundary along the continuum and the just noticeable difference (jnd; i.e., the number of steps required for the categorisation to shift from 50% to 75%). The jnd for the 4-interval forced choice ba-da discrimination is also given, together with the non-speech control condition.

Table 4 here

The two groups did not significantly differ in any of the speech categorisation tasks. However, there was a trend towards a difference for the coat-goat threshold ($F(1,30)= 3.5$, $p=.07$); this was accounted for by 5 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, neither in the speech nor in the non-speech condition (with a single formant F2). A paired-samples t-test revealed that jnds were significantly lower in the non-speech than in the speech condition ($t(31)=2.2$, $p=.035$), 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 summarised in Table 5. For each subject we considered the median of the 2 to 4 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 ($F(1,30)= 2.63$, $p=.11$), due to 6 dyslexics with thresholds over 60 dB. There were significant group differences in FM detection at 2 Hz ($F(1,30)= 4.2$, $p=.048$) and in temporal order judgement with long stimuli ($F(1,30)= 8.26$, $p=.007$) as well as with short stimuli ($F(1,30)= 6.4$, $p=.017$). In all conditions where group differences were observed, they were attributable to the high thresholds of 5-7 dyslexics.

Table 5 here

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, presented in Table 6.

RAPID summarises 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, temporal order judgement short and long conditions (even in the long condition, stimulus onset asynchronies became short). Absolute threshold was not considered a rapid processing task, since when 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 that frequency the modulations are not resolved by the auditory system. SLOW is the average z-score of all the other jnds: absolute threshold, FM detection at 2 and 240 Hz.

SPEECH is the average z-score of the tasks involving speech: the three syllable categorisation 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 short and long conditions, FM detection at 2 and 240 Hz.

Since some results present 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, summarising all the tasks which: 1) have shown poor performance in dyslexics in the literature, or 2) *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 short and long conditions, FM detection at 2 Hz (i.e., same as RAPID, without simultaneous masking, and with FM 2 Hz).

Table 6 here

All summary variables showed a significant group effect. A repeated measures analysis revealed no interaction between group and RAPID vs. SLOW ($F(1,30)=1.27$, $p=.27$), showing that dyslexics were not worse at tasks involving rapid auditory processing than at the others. Furthermore, a deviance analysis found abnormal performance in 7 dyslexics and 1 control in RAPID and 6 dyslexics and 1 control in SLOW (1 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 vs. NONSPEECH ($F(1,30)<1$), showing that dyslexics were not worse at speech than at non-speech tasks. A deviance analysis found abnormal performance in 7 dyslexics and 2 controls in SPEECH (1 control excluded) and 5 dyslexics in NONSPEECH. Our results therefore do not support the speech-specific hypothesis either.

The "pragmatic" AUDITORY score shows the greatest difference between controls and dyslexics ($F(1,30)=8.58$, $p=.006$), with 10 dyslexics out of 16 and 1 control showing abnormal performance (1 control excluded). Individual scores are plotted in Figure 2b. Unfortunately, no obvious construct seems to be able to capture what all these auditory tasks have in common, that tasks such as simultaneous masking or FM detection at 240 Hz do not have. This remains true even if one considers only the most sensitive tasks, i.e., TOJ short and long and FM 2Hz. We therefore have to conclude, like Rosen & 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 on 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 (AW, ML and VF vs. AJ and NDC), as well as between speech and nonspeech perception (SM, KH, VF and JC vs. LP and JG). Some dyslexics seem to have absolutely no auditory deficit (MW, DM, ON), some have relatively focal problems (AJ, NDC, SM), while others are impaired across the board (FH, DT).

Table 7 here

In summary, we find that a significant proportion of dyslexics are impaired in the auditory domain. However there is a great heterogeneity in the nature of the problem. Depending on how one construes the auditory deficit, between 7 and 10 dyslexics out of 16 are affected, compared with just one or two controls. Certain dyslexics, on the other hand, seem to have entirely intact

auditory abilities. This is consistent with all previous studies where individual data has 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 >.20).

Table 8 here

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 so much the overall difficulty of the task 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-scores of contrast sensitivity magno and speed discrimination magno. A deviance analysis on this variable found that just 2 dyslexics out of 15 and 2 controls had abnormal performance in the magnocellular conditions (1 control excluded) (see individual data in Fig. 2c). This is consistent with previous studies where individual data was examined; for instance, Cornelissen et al. (1995) found between 5 and 10 dyslexics out of 29 who were outside the range of most controls, and Witton et al. (1998) around 4/17 (both in coherent motion).

Cerebellar tests

Balance

The steps in which each subject counted backwards was determined so as to equate the difficulty of the tasks across the subjects. Among the controls, 3 counted in 2s, 9 in 3s and 4 in 7s, while among the dyslexics, 1 counted in 1s, 11 in 2s and 4 in 3s (chi-square=11.5, $p<.01$). 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 is a total of 12 variable across the 4 conditions (path lengths of the center of foot pressure (CP) and the C7 diode for the two conditions with arms alongside; CP, C7 plus the two hands for the two conditions with arms extended). Means and standard deviations of all measures are summarised 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 significantly differ between groups, even in the dual task condition (all $F(1,28)<1$).

In order to summarise the balance results for further analyses, we averaged the z-scores of these 12 variables into a single BALANCE score (see Table 9). This new variable did not differ significantly between groups ($F(1,30)<1$). A deviance analysis found 2 dyslexics (ON and DT)

and 2 controls with abnormal performance in BALANCE (1 control excluded).

Bi-manual finger tapping

For each task the mean and standard deviation of inter-response intervals, during the first 30 seconds (with metronome) and during the next 30 seconds (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 et al. (1990), we used only IRI standard deviations for subsequent analyses.

We summarised performance in finger tapping by averaging the z-scores of all IRI standard deviations to form the new variable BIMANUAL (see Table 9). This variable did not differ significantly between the two groups ($F(1,30)=1.1$, $p=.3$). A deviance analysis found 4 dyslexics (ML, VF, NC and DT) and 2 controls with abnormal BIMANUAL scores (1 control excluded).

Time/loudness estimation

In both tasks, the fit of the logistic regression was significant for all subjects (all p values $<.001$). Just noticeable differences for duration and loudness differences were analysed. Neither variable showed a significant group difference. Two dyslexics (ML, VF) and 2 controls had abnormally high thresholds in time estimation (1 control excluded). Curiously, 5 dyslexics (MW, NDC, ML, FH, DT) and 1 control were deviant on loudness estimation (1 control excluded). Considering that ML, VF, FH and DT were already deviant on AUDIO, it seems that both time and loudness estimation tap some aspect of auditory function. Thus, it may not be very appropriate to interpret poor performance at time estimation as an indicator of cerebellar dysfunction.

Table 9 here

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 was missing. Mean scores for each group are reported in Table 9. None of those cerebellar tests showed any significant difference between groups.

We computed a new CEREB variable 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 does not differ significantly between the two groups ($F(1,30)<1$). A deviance analysis on this variable suggests that 4 dyslexics (ML, ON, DT, JG) and 2 controls have abnormal overall performance in the cerebellar tests (1 control excluded) (see individual data in Fig. 2d). However, considering that just one of these 4 dyslexics (ML) was impaired in time estimation, and that he was also impaired on 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 (ON and DT) had impaired balance, and only one of them in the dual task (DT), casting 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., in press) (but their criterion was 1 St. Dev. beyond control mean) and reports of no such disorders (van Daal and van der Leij, 1999; Wimmer et al., 1998).

Further analyses

Relationship between auditory and phonological performance

The great heterogeneity of auditory performance observed in the dyslexic group, when compared to the relative homogeneity of the phonological deficit, would suggest that there is no meaningful relationship between the two domains. Yet, there is a significant correlation between AUDITORY and PHONOLOGY (r=-.54; p=.001; see Table 10). That is, AUDITORY accounts for 29.6% of the PHONOLOGY variance. In order to be really meaningful, this correlation should hold within each group, since an overall correlation is predicted even without causation, in virtue of the differences between the two groups along both dimensions. In fact, the correlation holds within the control group (r=-.6, p=.01), but not within the dyslexic group (r=-.3, p=.26), a rather surprising finding since the dyslexic group shows greater variability. The scatterplot (Figure 3) seems to indicate that auditory performance does not really predict phonological performance, but that it rather places an upper limit on it. In other words, poor audition entails poor phonology, but the reverse is not true: some subjects have very poor phonology but excellent audition (e.g., ON and MW).

Table 10 here

Figure 3 here

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 for powerful correlation analyses; indeed, if a Bonferroni correction was 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 spoonerisms accuracy and nonword repetition correlate with SPEECH and RAPID auditory processing. (NB: the variables summarised in SPEECH are also included in RAPID, and those summarised in SLOW are also included in NONSPEECH, so these associations are expected by design). Verbal short-term memory (WMI), on the other hand, doesn't 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 was confirmed in future studies, it would interestingly suggest that different types of auditory deficits might impact on different aspects of phonology.

Table 11 here

It is worth noting that the correlation between SPEECH and spoonerisms and nonword repetition may be due to two different reasons. The straightforward interpretation is that speech perception skills have a developmental impact on phonological skills, as measured by spoonerisms and nonword repetition. But is also likely that, whether or not speech perception affects phonology, it certainly affects performance in those particular tasks. Indeed, difficulties discriminating, say, between /b/ and /d/ must make it more difficult to correctly repeat a nonword 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 easily generalise 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 on 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 is found to weakly correlate with AUDITORY (r=.35, p=.05), but this would not survive a Bonferroni correction. Examination of the scatterplot suggests that the correlation is due to just one outlier (DT, the worst performer in both domains), whose removal does indeed annihilate the effect (r=.04, p=.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 summarises the individual data across the different domains. As we have seen before, 16 dyslexics out of 16 have poor performance in phonology, 10 in audition, 4 in cerebellar function and 2 in magnocellular vision. There is some overlap between cerebellar and auditory disorders. In the present sample, like in Witton et al. (1998), visual disorders are confined to a subset of the auditorily affected dyslexics. Finally, 5 dyslexics seem to be entirely unaffected by any sensory or motor/cerebellar disorder, i.e., they seem to have a purely phonological dyslexia.

Figure 4 here

Predictors of literacy

The fact that 5 of the dyslexics seem 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 have 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 ($F(1,30)=95.4$, $p<.001$). Then AUDITORY was found to account for an additional 4.2% of the variance ($F(1,29)=6.2$, $p=.02$) (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 ($F(1,28)=9.1$, $p=.005$). 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 way to explain this relationship other than chance. We therefore conclude that CEREB actually does not contribute to explaining LITERACY variance. 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, on top of 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 (nonwords, for practical purposes). One would then expect that spelling is the literacy task that AUDITORY is most correlated with. This is indeed the case ($r=-.609$, $p<.001$), although by very little (with WRAT reading: $r=-.607$, $p<.001$). Of course, reading and spelling are themselves highly correlated ($r=.82$, $p<.001$), 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 have 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 or ADHD respectively. In the present study, we specifically tried to avoid such co-morbid cases. However, two dyslexics had abnormally high scores on the ADD questionnaire: JC and ON. ON happens to also be an outlier on the CEREB variable, but JC seems to be a pure phonological case. We have also mentioned earlier that, according to his file, subject FH might be a case of mild SLI. He happens to have the second worst AUDITORY score. No other indication of any additional developmental disorder was found in the present sample. Our results suggest that although co-morbid 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 (see also Ramus et al., in press for a similar conclusion).

Discussion

As in most previous studies of dyslexia, we have 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 have found that a significant number of dyslexics in our sample (10/16) 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 what we obtain on any particular auditory task, considered separately. The higher incidence found here results from the administration of a greater number of tasks than 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 characterised as a rapid auditory processing deficit, as was predicted by the magnocellular theory; it is not the case either that they can be reduced to a speech perception deficit; actually, no coherent construct seems to be able to characterise 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 cortical hierarchies is increasingly demonstrated in single cell recordings and brain imaging studies (Friston and Büchel, 2000; Lamme and Roelfsema, 2000).

We have also found that motor problems are present in certain dyslexics (4/16), even in the absence of measurable co-morbid 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 Ramus et al., in press; Stringer and Stanovich, 1998; and Wimmer et al., 1998). Finally, our data beg the question whether motor problems play any causal role in

dyslexia. Contrary to the predictions of the cerebellar theory, we found an influence of motor/cerebellar performance neither on phonology nor on literacy. This might be due to the low prevalence of motor/cerebellar problems in the present sample (4/16), but this is consistent with another study where the prevalence was higher (Ramus et al., in press).

Only two of the dyslexics in our sample seemed to have visual problems of a magnocellular nature. This is in line with other studies where 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 generalisability 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 have selected an equal number of males and females whereas dyslexia is thought to be more frequent in males, one could argue that our sample is biased towards the female pattern, which may be a milder form of dyslexia. To test this hypothesis, we ran ANOVAs 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 >.10), and a significant sex × group interaction only on CEREB ($F(1,27)=5.5$, $p=.027$), 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, as compared to 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 generalised 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 have 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 pure phonological dyslexics might just be an illusion due to sensory-motor recovery. How likely is that possibility? We are not aware of cases of recovery of similar basic sensory or motor disorders, nor of a neurophysiological model of how such a recovery might occur. 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 forthcoming 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 [White et al., in preparation].

Conclusion

The results of the present study support the following view of developmental dyslexia. Notwithstanding the possibility of other independent (but rare) causes of reading impairment, the

cause of dyslexia is a phonological deficit. This deficit can arise independently of any sensory or motor impairment. Furthermore, a significant proportion of dyslexics suffer from additional auditory, visual or motor disorders. Auditory deficits, at least, may aggravate the phonological deficit, hence the 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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Table 1. Psychometric tests

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

FSIQ: Full-scale intelligence quotient; VIQ: verbal IQ; PIQ: performance IQ; VCI: Verbal comprehension index; POI: Perceptual orientation index; WMI: Working memory index; PSI: Processing speed index (WAIS).
ADD: Attention deficit disorder scale.
* $p<0.05$
*** $p<0.001$

Table 2. Reading and language tests

		Reading ***	Spelling ***	NART (correct resp. /50) ***	Reading speed (syl./sec) **	GNRT acc. (correct resp. /20) ***	GNRT RT (sec.) ***	ASTOP (% correct resp.)	TAPS (% correct resp.)	LITERACY (average z-score) ***
Controls	Mean	113.9	115.3	35.7	2.49	19.1	1.68	.94	.77	0
(N=16)	St.Dev.	4.5	4.7	5.3	.21	.93	.21	.04	.12	.62
Dyslexics	Mean	103.7	95.6	25.4	2.01	16.4	2.62	.95	.78	-2.98
(N=16)	St.Dev.	5.9	7.1	6.2	.50	2.09	.72	.03	.06	1.56

RT: reaction time.
** $p<0.01$
*** $p<0.001$

Table 3. Phonological tests

		Picture naming (sec.) **	Digit naming (sec.) ***	Spoonerisms acc. (correct resp. /12) **	Spoonerisms RT (sec.) **	CNREP (% correct resp.) *	PHONOLOGY (average z-score) ***
Controls	Mean	54.5	27.8	11.3	4.45	.92	0
(N=16)	St.Dev.	7.0	4.6	.87	1.21	.05	.42
Dyslexics	Mean	68.4	42.9	8.5	9.96	.86	-2.6
(N=16)	St.Dev.	15.4	12.5	2.9	5.88	.06	1.49

RT: reaction time.
* p<0.05
** p<0.01
*** p<0.001

Table 4. Speech perception tests

		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	Mean	2.4	30.5	2	37.5	2.3	27.5	17.5	15.6
(N=16)	St.Dev.	1.4	5.1	1	5.3	.8	3.6	4.6	8.7
Dyslexics	Mean	3.1	30.2	2.4	38.7	3.3	28.1	19.6	14.6
(N=16)	St.Dev.	2.2	4.5	.9	3.9	1.9	4.2	8.4	6.6

Unit: number of steps on the continuum, out of 41 for Ba-Da and 51 for Date-Gate and Coat-Goat.

Table 5. Non-speech perception tests

		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	Mean	46.3	76.3	24.4	1.01	.0042 (N=14)	50.7	34.7
(N=16)	St.Dev.	10.1	2.5	5.4	.34	.0027	24.7	30.3
Dyslexics	Mean	53.3	75.9	26	2.04	.0076	93	106
(N=16)	St.Dev.	14	4.2	4.9	1.97	.0078	53.5	109

* p<0.05
** p<0.01

Table 6. Summary auditory variables

		RAPID *	SLOW *	SPEECH *	NONSPEECH *	AUDITORY **
Controls	Mean	0	0	0	0	0
(N=16)	St.Dev.	.44	.71	.51	.5	.38
Dyslexics	Mean	.78	1.53	.63	1.13	1.14
(N=16)	St.Dev.	1.07	2.89	.82	1.99	1.5

* p<0.05
** p<0.01

Table 7. Individual z-scores on summary auditory variables (dyslexic group and deviant controls). Only deviant values (>1.65) are shown.

	RAPID	SLOW	SPEECH	NONSPEECH	AUDITORY
			Dyslexics		
AJ		2.54			
AW	4.94		5.45	2.36	5.52
MW					
NDC		3.20			1.95
SM			2.06		
KH			2.24		1.77
ML	2.42				2.23
DM					
VF	2.19		2.59		3.03
LP	4.22	5.12		5.26	5.31
FH	6.64	6.91	6.18	6.08	9.40
JC			4.21		
NC					1.98
ON					
DT	9.47	19.05	3.31	14.88	16.23
JG	1.89	5.70		3.59	3.83
			Deviant controls		
MD		2.89		2.45	
RG			1.70		
WN	2.69		3.60		2.05
MM		2.08			

Table 8. Visual perception tests

		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	Mean	6/6.84	1.45	2.01	140	200	60.4	0
(N=16)	St.Dev.	1.2	.16	.23	13	84.5	20.4	.87
Dyslexics	Mean	6/6.6	1.48	2.03	146	182	54.7	.06
(N=15)	St.Dev.	1.14	.11	.15	9.7	26.5	19.6	.53

Table 9. Cerebellar tests

		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	Mean	40.8	5.7	84.3	3.7	186	0	0	0
(N=16)	St.Dev.	6.6	1.3	9.2	.14	17	.74	.8	.51
Dyslexics	Mean	39.7 (N=13)	6.2	81.8	3.92	194	.34	-.04	.15
(N=16)	St.Dev.	3.4	1.5	5.2	.66	17	1.08	.8	.54

Table 10. Pearson correlations between summary variables across domains

	LITERACY	PHONOLOGY	AUDITORY	VISION
PHONOLOGY	.872***			
AUDITORY	-.647***	-.544**		
VISION	-.108	-.085	.139	
CEREB	-.048	-.228	-.35*	-.316

* $p<0.05$, ** $p<0.01$, *** $p<0.001$, no correction applied.

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

	RAPID	SLOW	SPEECH	NONSPEECH	AUDITORY
WMI	-.269	-.163	-.331	-.182	-.289
Picture Naming	.345	.548**	.173	.476**	.488**
Digit Naming	.316	.51**	.157	.44*	.479**
Spoonerisms accuracy	-.373*	-.004	-.386*	-.153	-.268
Spoonerisms RT	.484**	.242	.344	.363*	.443*
CNREP	-.361*	-.144	-.364*	-.223	-.352*

* $p<0.05$, ** $p<0.01$, no correction applied.

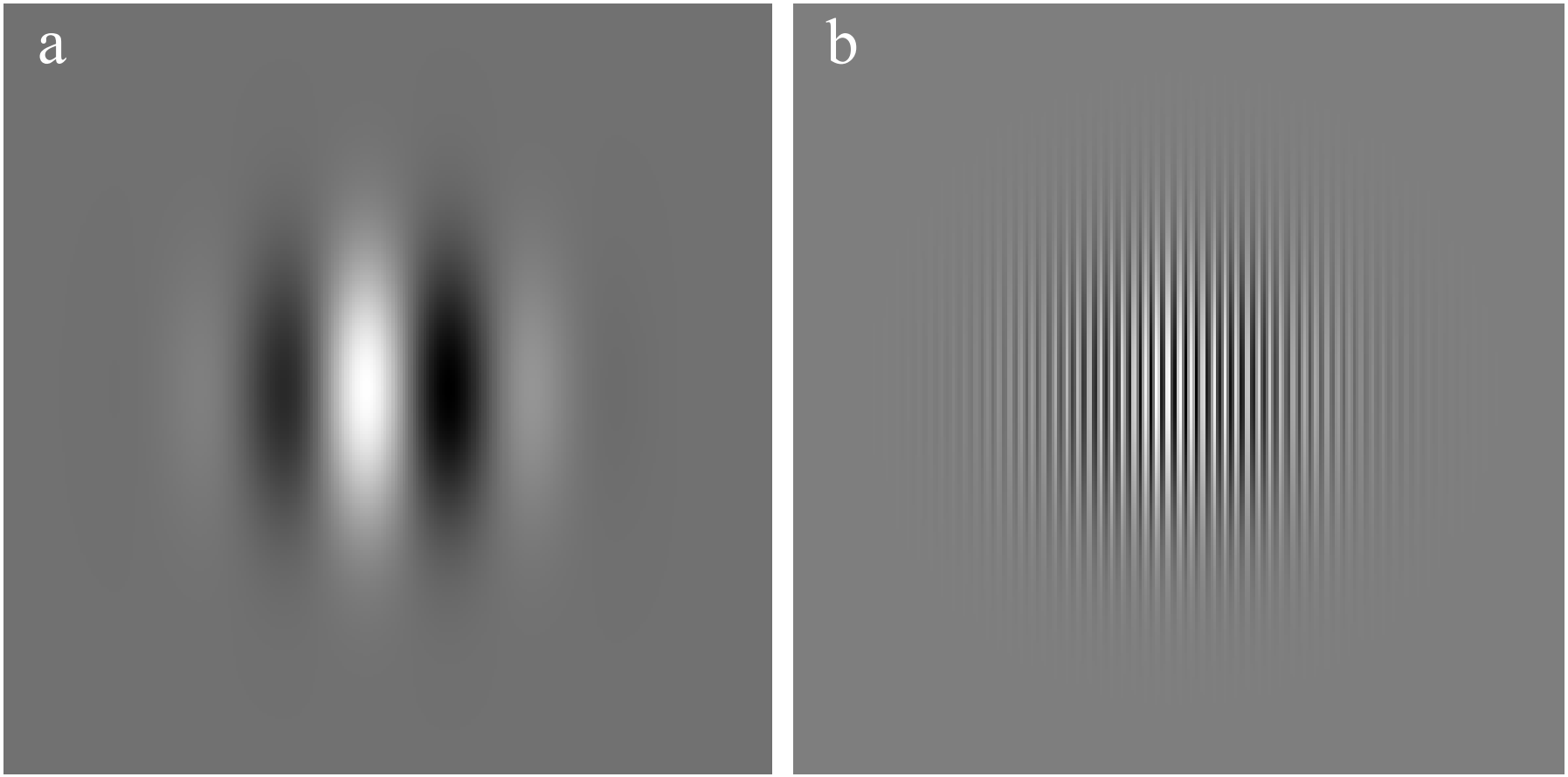


Fig. 1. Stimuli used for contrast sensitivity and speed discrimination. a) Magno-specific stimulus; b) Parvo-specific stimulus.

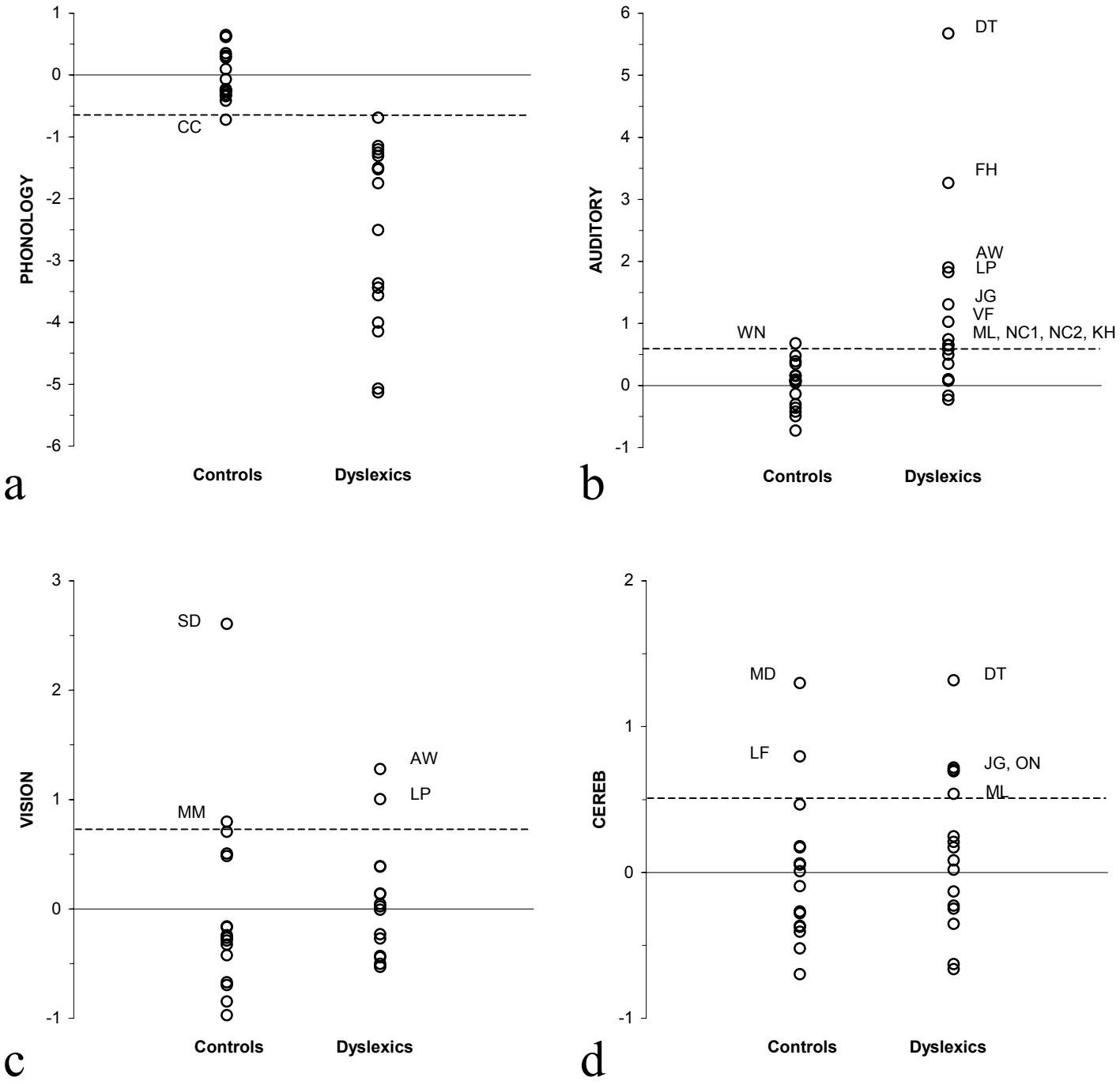
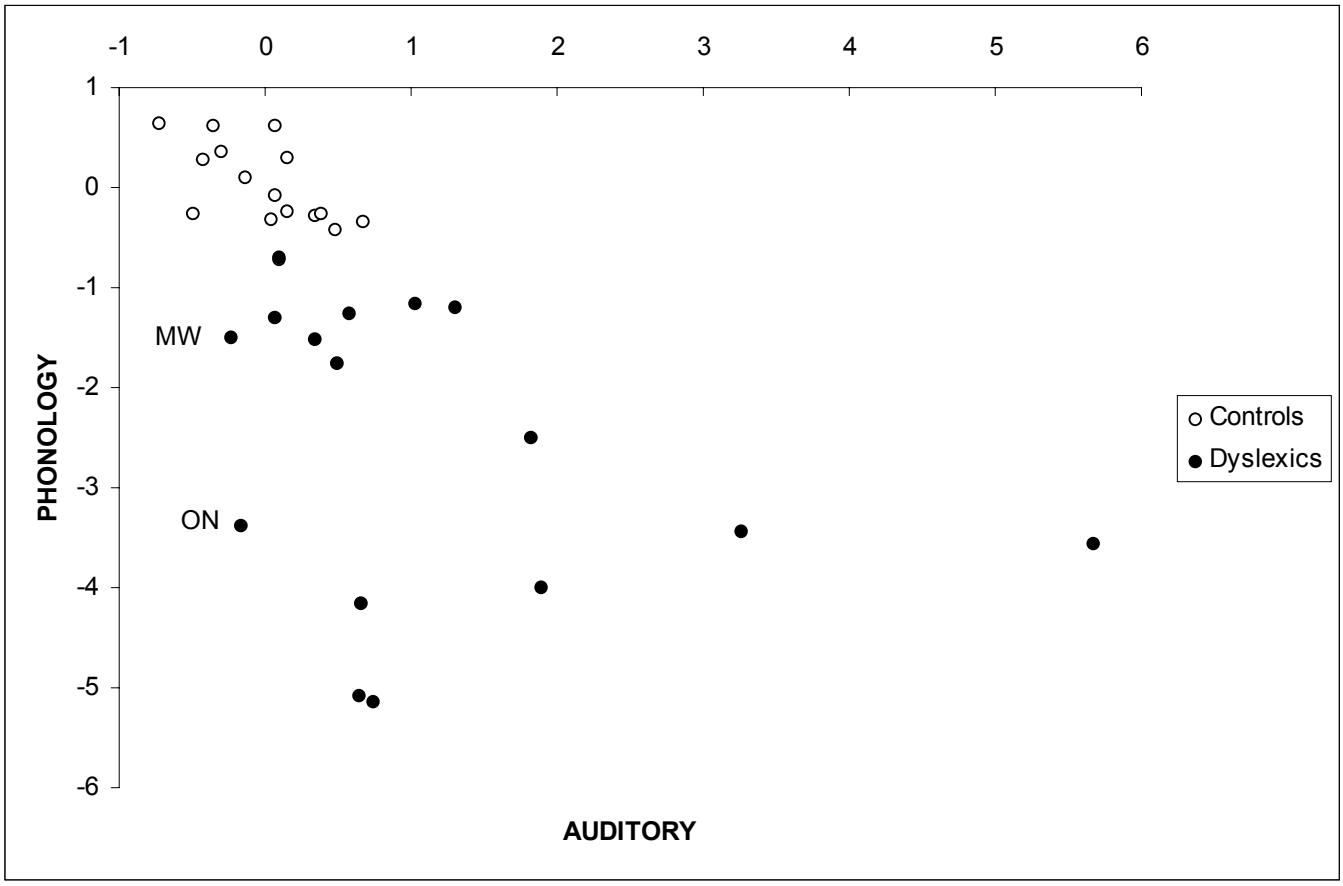


Fig. 2. Individual scores on summary factors for each domain.
The solid line indicates the control mean, and the dashed line the chosen deviance threshold (1.65 St. Dev. above the control mean, after excluding deviant controls). Deviant individuals are identified, except for phonology where all dyslexics are deviant. a: Phonology; b: Audition; c: Vision; d: Cerebellar function.

Fig. 3. Auditory vs. phonological performance.



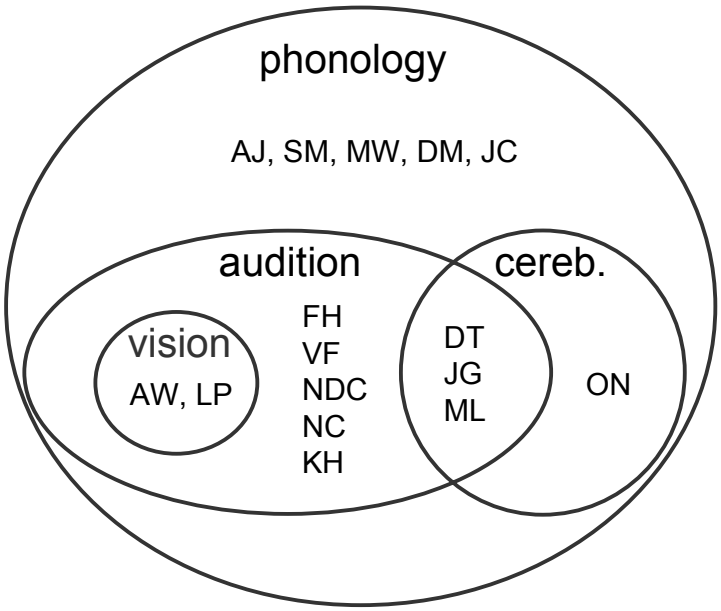


Fig. 4 Distribution of phonological, auditory, visual, and cerebellar disorders in the sample of 16 dyslexic adults. Initials refer to individual dyslexic subjects.