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Developmental dyslexia: specific phonological deficit or general sensorimotor dysfunction?

Franck Ramus

Dyslexia research now faces an intriguing paradox. It is becoming increasingly clear that a significant proportion of dyslexics present sensory and/or motor deficits; however, as this 'sensorimotor syndrome' is studied in greater detail, it is also becoming increasingly clear that sensory and motor deficits will ultimately play only a limited role in a causal explanation of specific reading disability.

Addresses
Laboratoire de Sciences Cognitives et Psycholinguistique (EHESS/ENS/CNRS), 54 boulevard Raspail, 75006 Paris, France
e-mail: ramus@lscp.ehess.fr

Current Opinion in Neurobiology 2003, **13**:212–218

This review comes from a themed issue on
Cognitive Neuroscience
Edited by Brian Wandell and Anthony Movshon

0959-4388/03/$ – see front matter

DOI 10.1016/S0959-4388(03)00035-7

Introduction

Developmental dyslexia is a failure to acquire reading skills that affects around 5% of children, despite adequate intelligence, education and social background. There is a wide consensus that it is a neurological disorder with a genetic origin. Yet, after decades of research, it seems surprising that theorists still have fundamental disagreements over the neurological and cognitive basis of the disorder. The dyslexia scene is currently occupied by no less than four major theories, which can be grouped within two antagonistic frameworks; moreover, each of these theories is supported by a whole body of empirical evidence.

On one side of the divide, theorists contend that the specific reading retardation characteristic of dyslexia is directly and exclusively caused by a cognitive deficit that is specific to the representation and processing of speech sounds: this is the phonological theory (Figure 1; [1–3]). At the level of the brain, this cognitive deficit would arise from a congenital dysfunction of certain cortical areas involved in phonology and reading [4–6]. On the other side of the divide, researchers agree with the idea of a phonological deficit but see it as secondary to a more basic auditory impairment, and as part of a general sensorimotor deficit. Separate theories have been developed on the basis of an auditory deficit [7], a magnocellular visual dysfunction [8] or a cerebellar/motor dysfunction [9•], but they have recently been unified under the general magnocellular theory of dyslexia [10•]. (There is also an attentional variant of this theory [11].) According to this view, there are two direct causes of reading retardation: phonological and visual deficits. The phonological deficit can be traced back to a more general auditory impairment, which has the same biological origin as the visual impairment, namely, a dysfunction of magno-cells in sensory pathways. This magnocellular dysfunction is also apparent in the tactile domain [12], and reaches the cerebellum via the posterior parietal cortex, causing further impairments, notably in the motor domain (Figure 2; [10•]). In this theory, therefore, dyslexia is seen as a general sensorimotor syndrome.

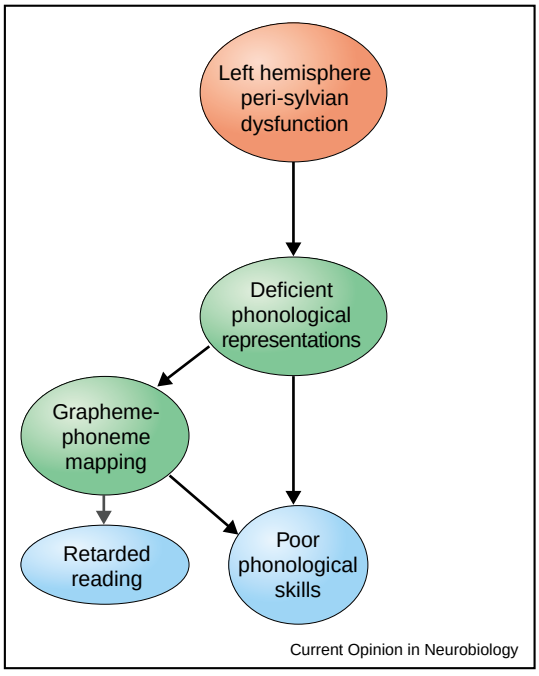
The phonological theory has been predominant for about 20 years. Gradually, more and more studies have emerged that provide evidence for auditory, visual and motor impairments in dyslexics, and give support to the sensorimotor theories. This trend was reflected in the last two reviews of dyslexia in this journal [13,14]. However, the past two years of research suggest that the tide may be turning again [15]. Here, I review recent studies of sensory and motor function in dyslexics, with a particular focus on those that challenge received wisdom in these areas.

Auditory processing in dyslexia

Many studies have further confirmed the presence of auditory deficits in the dyslexic population. Moreover, deficits are demonstrated across a wide range of auditory tasks, from Tallal's [7] classic temporal order judgement and repetition tests [16,17,18••,19,20••], to discrimination of frequency and intensity [21,22••,23••], gap detection [20••,24], detection of illusory movement [25•], detection of frequency and amplitude modulation [23••,26,27], categorical perception of phonemes and non-speech analogues [28,29•] and backward masking [30•,31••]. Three debates surround the study of dyslexics' auditory processing. First, what proportion of dyslexics are affected? Second, can the deficit be characterised in terms of 'rapid auditory processing'? And third, does the deficit in auditory processing explain the phonological deficit?

From the beginning of this type of research, it has been evident that only a fraction of dyslexics showed poor performance in the auditory tasks (45% in [7]). However, it is possible that this was due to the poor reliability of the tasks used, with low numbers of trials per subject leading to high measurement error and overlap between the groups [32]. Recent years have seen great improvements

Figure 1



The phonological theory of dyslexia. A specific phonological deficit of left peri-sylvian origin is postulated to be the direct cause of reading problems. Bubbles represent impairments at the neurological (red), cognitive (green) and behavioural (blue) levels; arrows represent causal connections.

in the methodology: adaptive psychophysical procedures with multiple measures of each threshold are now routinely used, so that individual data can be taken seriously. Yet, this has only reinforced the original observation; when collapsing the data across all the recent studies in which individual data was analysed or displayed, a total of 67/174 (39%) dyslexics had an observed auditory deficit [19,21,23••,24,26,28,30•,31••,33,34].

Most of the auditory studies have been taken to support the view that dyslexics' auditory processing is impaired specifically on short sounds and fast transitions: this is called the 'rapid' or 'temporal' auditory processing deficit [7]. Such a characterisation of the auditory dysfunction is consistent with the magnocellular theory, as magno-cells are particularly sensitive to high temporal frequencies [10•]. However, a closer look reveals major inconsistencies between the data and the theory; some deficits are found in tasks that don't tap rapid auditory processing, such as frequency discrimination [21,22••] or frequency modulation detection at 2 Hz [26]. On the other hand, the expected rapid processing deficits are often not observed. In fact, when inter-stimulus intervals were manipulated in a systematic manner, the auditory processing of dyslexics was not found to be poorer at short than at long intervals, and sometimes it was better [18••,20••,21,22••].

Finally, three separate studies have investigated dyslexics' auditory processing on a large array of psychophysical tests administered to the same subjects. They revealed that a subset of dyslexics do have difficulties with certain tests, but that the pattern of good and poor performance can in no way be characterised as a problem with rapid or temporal processing [23••,30•,31••]. Moreover, the pattern of performance varies widely across individuals. A coherent characterisation of dyslexics' auditory performance remains elusive.

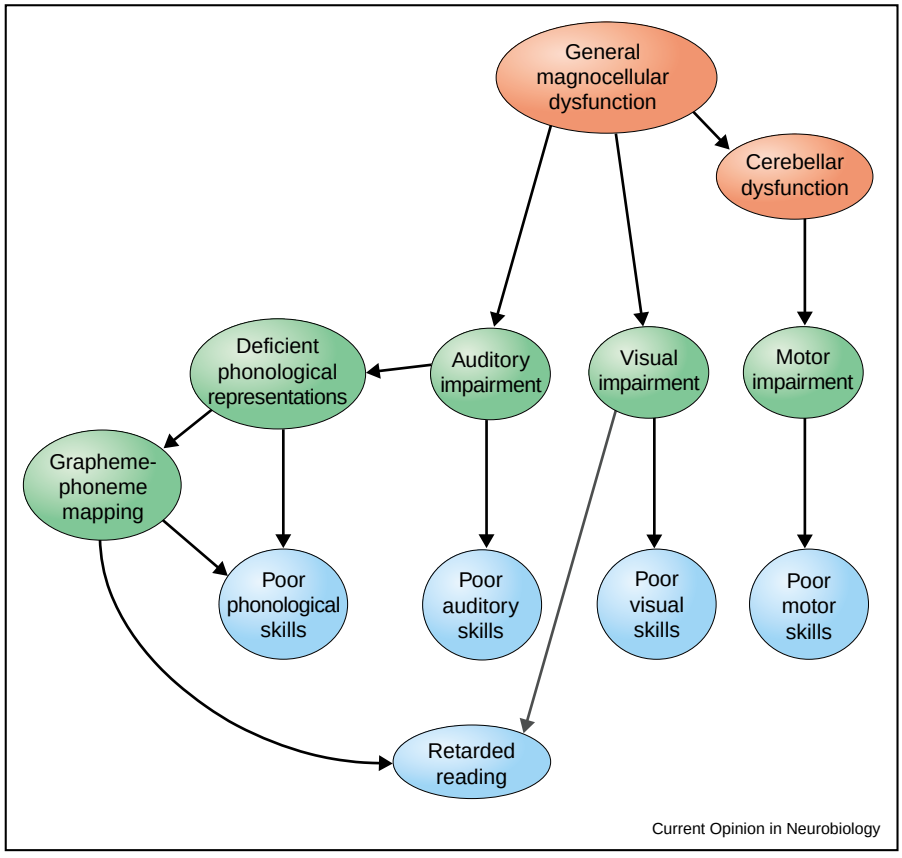
A causal connection?

This leads us to ask, when an auditory deficit is present in a dyslexic individual, is it responsible for the phonological deficit and/or for the reading disability? Supporters of the auditory processing theory have hypothesised that impaired perception of brief sounds and transitions would be particularly detrimental to speech perception, and hence would undermine the development of the child's phonological representations [35,36]. Evidence against this hypothesis was soon put forward [37]. Recent studies have now established that there is no reliable relationship between performance on rapid auditory processing tasks and speech categorisation and discrimination [29•,30•,31••]. Neither is there a reliable relationship between any auditory measure (speech or non-speech) and more general measures of phonological skill or reading ability [19,25•,33], even when assessed longitudinally [18••]. If anything, it seems that dyslexics that have the worst auditory impairments also have severely impaired phonology and reading, although the reverse is not necessarily true [23••,26,31••].

Interestingly, there have been claims that auditory training programs can improve the language and reading skills of dysphasic and dyslexic children [38–40]. Unfortunately, these studies have not protected themselves against placebo and Hawthorne effects by running double-blind randomised controlled trials (The Hawthorne effect refers to positive outcomes exclusively caused by being the subject of a trial). A few independent studies that have attempted to assess the effects of the controversial Fast Forword programme, which is based on an adaptive training of rapid auditory processing [39], have found it no more efficient than more traditional intervention programs, and they have challenged the role of the rapid auditory processing part of the training [41•–43•].

In summary, the auditory disorders that are observed in individuals with dyslexia are not particularly 'rapid' or 'temporal' in nature. These disorders are restricted to a subset of the dyslexic population, and have little influence on the development of phonology and reading. It therefore seems that the phonological deficit that is characteristic of dyslexia can arise in the absence of any auditory disorder, with the most severe auditory impairments nevertheless acting as aggravating factors.

Figure 2



The general magnocellular theory of dyslexia [10•]. A general magnocellular dysfunction is hypothesised to engender auditory, visual and cerebellar/motor deficits. The auditory deficit in turn causes a phonological deficit, thereby triggering the same cascade of events as predicted in the phonological theory. The visual magnocellular deficit is seen as another direct cause of reading problems. In the cerebellar theory [9•], the cerebellar/motor impairment is also thought to independently contribute to phonological and reading problems (not represented here).

Visual processing in dyslexia

The debate on visual deficits in dyslexia is articulated around three questions that are similar to those asked about the auditory deficit. First, do visual disorders cause reading difficulties? Second, do those visual disorders have a magnocellular origin? And third, what proportion of dyslexics are affected?

Even when excluding major ophthalmologic disorders, it seems plausible that more subtle visual deficits might have an impact on reading. Perhaps the clearest example is visual stress [44], a condition that provokes visual distortions and sometimes leads to impaired reading fluency. This condition can be improved by using coloured overlays or glasses [45,46]. Other visual problems that are often mentioned in the context of dyslexia include binocular fixation instability and poor vergence control [10•], increased visual crowding [47], and slight visuo-spatial attention deficits [48]. Although these are all plausible proximal causes of reading impairment, both their prevalence and their relationship to reading retardation remain hotly debated, especially as visual disorders are often accompanied by a phonological deficit.

Whether a magnocellular dysfunction is the underlying cause of these proximal visual impairments is far from clear. Several studies have provided evidence that dyslexics have elevated detection thresholds or abnormal visual evoked potentials for stimuli in the spatial and temporal ranges of the magnocellular system [24,49–53], although it has been disputed whether some of the stimuli used uniquely tap the magnocellular system [54,55]. However, a growing number of studies report findings that are inconsistent with a visual deficit that is specific to the magnocellular system [20••,22••,31••,56•,57–59]. These studies have often found that visual deficits, when present, cover the whole range of spatial and temporal frequencies. Questions have also been raised as to whether group differences could be explained by attention or memory rather than sensory deficits [57,60]. Moreover, visual deficits seem to be restricted to a subset of dyslexics; in seven recent studies displaying individual

data, 37/128 (29%) dyslexics had elevated visual thresholds in the target conditions [22••,24,31••,34,49,51,53]. Finally, no demonstration has been provided that magnocellular dysfunction, when present, engenders visual problems that are more proximal to reading, such as visual instability, crowding or stress. In fact, in the case of visual stress, there is evidence that the symptoms are unrelated to magnocellular dysfunction [61].

To summarise, a minority of dyslexics seem to have visual problems. Visual stress seems to be dissociated from the phonological deficit, and is therefore a possible independent cause of reading disability. However, the underlying biological cause of these visual disorders and their precise impact on reading still needs to be elucidated. The hypothesis of a magnocellular origin does not seem to be well supported.

Motor control in dyslexia

Motor difficulties are also frequent in the dyslexic population [62–64]. However, as for auditory and visual deficits, some studies have failed to find any link between dyslexia and motor difficulties [25•,65]. Furthermore, motor impairments are restricted to a subset (estimated at between 30 and 50%) of the dyslexic population [31••,62]. One possible hypothesis is that motor impairments emerge from a general temporal processing or timing deficit [64]. There is, however, contradictory evidence [20••]. Another possibility is that motor impairments arise from a cerebellar dysfunction [9•], which is supported by the parallel findings of poor dyslexic performance in task automaticity, implicit learning, time estimation and, most recently, eyeblink conditioning [66]. However, independent studies have not always confirmed these findings, notably those regarding implicit learning [67], automaticity [31••] and time estimation [31••,62]. Finally, there is little evidence for a causal link between motor difficulties and phonological processing, and/or reading [62].

A general sensorimotor syndrome?

The recurrent theme of this discussion so far is that sensory and/or motor disorders do occur more often in the dyslexic than in the non-dyslexic population, but with a limited prevalence, variable manifestations, and limited consequences on reading skill. However, an intrinsic limitation of the research reviewed here is that most studies focus on one domain (auditory, visual or motor), and often use just one or two tasks to assess it. This approach leaves open radically different possibilities (and intermediate solutions). It could be that about one-third of dyslexics are affected by a multi-modal sensorimotor syndrome, with the rest of the population entirely spared. Alternatively, one-third of dyslexics could have an auditory deficit, another third a visual deficit, and the rest a motor disorder, so that every dyslexic would have a sensory or motor disorder that might explain his/her reading disability.

A few recent studies have begun to tackle this issue. They have investigated, within the same subjects, auditory and visual processing [22••,24,68]; auditory, visual and tactile processing within and across modalities [69,70]; and auditory, visual and motor functions [20••, 25•,31••]. Overall, these studies show that there is partial, but not total, overlap between the deficits in the different domains; and that, even when a considerable array of tasks is used in each modality, some dyslexics are entirely spared by sensorimotor deficits and seem to have a pure phonological dyslexia.

One viable hypothesis is that the sensorimotor dysfunctions discussed in this review form a general sensorimotor syndrome, which has variable manifestations across different individuals, and is an optional, rather than a defining, feature of developmental dyslexia. In fact, there is good evidence that sensorimotor dysfunction is an optional feature of several developmental disorders, including specific language impairment (SLI), autism, dyspraxia, and Williams syndrome [71–75]. The optional character of the sensorimotor syndrome also makes sense in the light of behavioral genetic studies showing that phonological deficits are highly heritable, whereas auditory and visual disorders are not [56•,76,77].

Phonological processing in dyslexia

In contrast to sensorimotor disorders, a deficit in phonological processing remains the most consistent finding in all studies of dyslexia, as confirmed again by our recent study that showed that 100% of the dyslexic sample were affected [31••]. It is sometimes argued that the phonological theory is a tautology rather than an explanation, that phonology and reading are two sides of the same coin, in the sense that phoneme awareness is enhanced by reading skill as well as the other way around. This point might be valid if the phonological deficit could be reduced to a problem with phoneme awareness; however, this is not the case.

Indeed, beyond phonological awareness, dyslexics have at least two other major phonological problems, in rapid naming (of pictures, colours, digits or letters) and verbal short-term memory, neither of which can be said to rely on reading. A major debate in the recent literature is whether these are independent phonological deficits or whether they are different manifestations of a single underlying deficit. Evidence has been provided that indicates that phonological awareness and rapid naming deficits are relatively independent and additive [78,79]; however, the debate is far from closed. More generally, it must be pointed out that phonology does not reduce to awareness, naming and memory; consequently many aspects of dyslexics' phonology remain to be investigated [80].

Another important element that helps to judge the direction of causality is provided by longitudinal studies. For

instance, although school-age and adult dyslexics are sometimes shown to have abnormal patterns of visual fixation and attention [10•,48,81], it has never been clear whether this was a cause or a consequence of their reading problems. Indeed, recent research shows that visual training regimes such as regular video game playing can dramatically alter one's psychophysical abilities (Green, Bavelier: Video game playing: rot your brain or expand your mind? presented at the Cognitive Neuroscience Society Annual Meeting; San Francisco, CA: April 2002), this might also happen when learning to read. Evidence that these visual problems exist even before schooling and predict future reading difficulties would be needed to indicate causality. In contrast, in the case of phonology, it has been amply demonstrated that pre-school phonological skills predict future reading skills, and that they are already poor in would-be dyslexics [82–84].

Conclusions

Although the phonological deficit is still in need of a complete cognitive and neurological characterisation, the case for its causal role in the aetiology of the reading and writing disability of the great majority of dyslexic children is overwhelming. The most recent research reveals that this phonological deficit cannot be accounted for by a lower-level auditory processing deficit, let alone a deficit specific to 'rapid' or 'temporal' processing. More generally, all the sensorimotor problems investigated in dyslexia have both limited prevalence and limited effects on reading skill, although it remains possible that certain visual deficits, such as visual stress, may sometimes sufficiently disrupt reading ability so as to lead to a diagnosis of dyslexia. According to our current state of knowledge, developmental dyslexia seems best characterised as a specific phonological deficit, optionally accompanied by a sensorimotor syndrome. A complete theory of dyslexia will have to explain both the neurological origin of the specific phonological deficit, and the reasons why a sensorimotor syndrome occurs more often in the dyslexic than in the general population.

Acknowledgements

I thank Uta Frith and Sarah White for their comments on this paper.

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