

Developmental dyslexia: specific phonological deficit or general sensorimotor dysfunction?

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Summary

Dyslexia research is now facing an intriguing paradox: it is becoming increasingly clear that a significant proportion of dyslexics present sensory and motor deficits; however, as this “sensorimotor syndrome” is being studied in greater detail, it is also becoming increasingly clear that sensory and motor deficits will play only a limited role in a general causal explanation of specific reading disability.

Teaser

Sensory and motor disorders are present in a significant proportion of developmental dyslexics, but play only a limited role in the aetiology of reading disability.

Running head

Sensorimotor dysfunction in dyslexia

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 can seem 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 specific to representations and processing of speech sounds: this is the phonological theory [1-3] (Figure 1). At the brain level, this cognitive deficit would arise from a congenital dysfunction of certain cortical areas involved in phonology and reading [4-6]. On the other side, researchers agree with the idea of a phonological deficit, but see it as secondary to a more basic auditory impairment, and part of a general sensorimotor deficit. Separate theories have been developed based on 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: 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 [10] (Figure 2). In this theory, dyslexia is therefore seen as a general sensorimotor syndrome.

The phonological theory has been largely predominant for about 20 years. Gradually, more and more studies have emerged providing evidence for auditory, visual and motor impairments in dyslexics, providing support for the sensorimotor theories. This trend was reflected in the last two reviews of dyslexia in this journal [13,14]. But the last two years of research suggest that the tide may be turning again [15].

Auditory processing in dyslexia

Many studies have further confirmed the presence of auditory deficits in the dyslexia population. Moreover, deficits are demonstrated across a wide range of auditory tasks, from Tallal's [7] classic temporal order judgement and repetition tests [16-20], to frequency and intensity discrimination [21-23], gap detection [20,24], illusory movement detection [25], frequency and amplitude modulation detection [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: 1) what proportion of dyslexics is affected? 2) can the deficit be characterised in terms of "rapid auditory processing"? 3) does it explain the phonological deficit?

From the very 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, this was possibly 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]. The recent years have seen great improvements 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, one finds a total of 67/174 (39%) dyslexics with 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, since magno-cells are particularly sensitive to high temporal frequencies [10]. However, a closer look reveals major inconsistencies between data and theory: some deficits are found in tasks that don't tap rapid auditory processing, like frequency discrimination [21] [22], or frequency modulation detection at 2 Hz [26]. On the other hand, expected rapid processing deficits are often not observed; in fact, when inter-stimulus intervals have been manipulated in a systematic manner, dyslexics were not found to be poorer at short than at long intervals (sometimes they were even better) [18,20-22]. Finally, three separate studies have investigated dyslexics' auditory processing on a large array of psychophysical tests administered within subject: they have concluded 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 varies across individuals. A coherent characterisation of dyslexics' auditory performance remains elusive.

The next question is: 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 hypothesised that impaired perception of brief sounds and transitions would be particularly detrimental to speech perception, hence would undermine the development of the child's phonological representations [35,36]. Counter-evidence against this hypothesis was soon put forward [37]. Recent studies have now amply confirmed that there is no reliable relationship between performance on rapid auditory processing tasks and speech categorisation and discrimination [29-31]. 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 the most auditorily impaired dyslexics 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 dysphasic and dyslexic children's language and reading skills [38-40]. Unfortunately, these studies have not protected themselves against placebo and Hawthorne effects by running double-blind randomised controlled trials. A few independent studies that have attempted to assess the effects of the controversial Fast Forward program [39] have not found it more efficient than more traditional intervention programs, and have challenged the role played by the part of the training focusing on temporal processing [41-43].

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

Visual processing in dyslexia

The debate on visual deficits in dyslexia is articulated around three similar questions as for the auditory deficit: 1) do visual disorders cause reading difficulties? 2) do those visual disorders have a magnocellular origin? 3) what proportion of dyslexics is 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 which provokes visual distortions and sometimes leads to impaired reading fluency, which can be improved by using coloured overlays or glasses [45,46]. Other visual problems that are often mentioned include binocular fixation instability and poor vergence control [10], increased visual crowding [47], as well as 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 since visual disorders are often accompanied by a phonological deficit.

Whether a magnocellular dysfunction is the underlying cause of those proximal visual impairments is far from clear. A number of studies do provide 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 inconsistent with a visual deficit specific to the magnocellular system [20,22,31,56-59], often finding 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: looking at 7 recent-studies displaying individual data, one finds 37/128 (29%) dyslexics with 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, like 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. At least 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 need 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 dyslexia population [62-64]. But just like for audition and vision, there have been negative findings [25,65], and evidence that motor impairments are restricted to a subset (estimated between 30 and 50%) of the dyslexia 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], supported by parallel findings of poor dyslexic performance in task automaticity, implicit learning, time estimation, and most recently eye-blink conditioning [66]. However, independent studies have not always confirmed these findings, notably 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 normal population, but with a limited prevalence, variable manifestations, and limited consequences on reading skill. However, an intrinsic limitation of the research reviewed is that most studies focus on one domain (auditory, visual or motor), very often using just one or two tasks to assess it. This approach leaves open radically different possibilities (and intermediate solutions): that about one third of dyslexics are affected by a multi-modal sensorimotor syndrome, with the rest of the population entirely spared; or, alternatively, that one third of dyslexics have an auditory deficit, another third a visual deficit, and the rest a motor disorder, so that every single dyslexic would have a sensory or motor disorder that might explain his/her reading disability.

A few recent studies have only 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 modality [69,70], and auditory, visual and motor functions [20,25,31]. Overall, they concur in showing 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 therefore that the sensorimotor dysfunctions discussed in this article form a general sensorimotor syndrome, which has variable manifestations across different individuals, and is an optional, rather than defining, feature of developmental dyslexia. In fact, there is good evidence that it is an optional feature of a number of developmental disorders, including SLI, autism, dyspraxia, and Williams syndrome [71-75]. The optional character of the sensorimotor syndrome also makes sense in the light of genetic studies showing that the phonological deficit is 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 reporting 100% of the dyslexic sample affected [31]. It is sometimes argued that the phonological theory is a tautology rather than an explanation, phonology and reading being 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: rapid naming (of pictures, colors, digits, 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 phonological awareness and rapid naming deficits are relatively independent and additive [78,79]; however, the debate is far from closed. More generally, it is fair to say that phonology does not reduce to awareness, naming and memory; many aspects of dyslexics' phonology remain to be investigated [80].

Another important element 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 retardation. Evidence that these visual problems exist even before schooling and predict future reading difficulties would be needed. 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].

Conclusion

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 extremely strong. Moreover, the most recent research reveals that this phonological deficit cannot be accounted for by a lower-level auditory processing deficit, let alone one specific to "rapid" or "temporal" processing. More generally, all the sensorimotor problems investigated in dyslexia have been shown to have both a limited prevalence and limited effects on reading skill, although it remains possible that certain visual deficits like visual stress may sometimes sufficiently disrupt reading so as to lead to a diagnosis of dyslexia. In the 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 why a sensorimotor syndrome occurs more often in the dyslexic than in the general population.

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This is a unique longitudinal evaluation of the auditory temporal processing hypothesis. The authors have examined the relationship between auditory (Tallal's repetition test), phonological and reading skills from kindergarten to Grade 2. They have found that dyslexic children tended to have impaired auditory processing at long rather than short inter-stimulus intervals, and only compared to the chronological-age control group; and that early auditory deficits did not predict later phonological and reading impairments.

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- A clever study comparing syllable discrimination within and across phonemic boundaries, and finding that dyslexics have, on average, better discriminative abilities within-boundaries, which would suggest that their perception is less categorical and more based on purely acoustic cues. The use of sine-wave speech allows the authors to tap speech and non-speech modes with the same stimuli: they find that enhanced within-boundary discrimination is stronger in speech mode, precluding an explanation in terms of temporal processing (this replicates [37] and is similar to findings by [30] and [31]).
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- This study describes possibly the largest battery of phonological, auditory, visual and cerebellar/motor tests administered to dyslexics and controls. It reports a very limited incidence of visual and motor deficits. Auditory deficits are present in 60% of the dyslexic sample, but with very variable manifestations across individuals (not particularly rapid processing) and at most an aggravating influence on the phonological deficit. Every single dyslexic in this sample shows a phonological deficit, with a third of them who seem entirely spared by any concurrent sensory or motor deficit.
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These two papers introduce and summarise a "Special forum on Fast ForWord" including five small-scale independent assessments of the controversial remediation program [39]. The first paper recalls all the important issues concerning the proper evaluation of remediation programs in general and that of Fast ForWord in particular (NB: they also apply to [40]). The second paper concludes that Fast ForWord does not seem more effective than other more conventional remediation programs.

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Keywords

Dyslexia; reading; phonology; magnocellular; audition; vision; motor control; temporal processing.

Figures

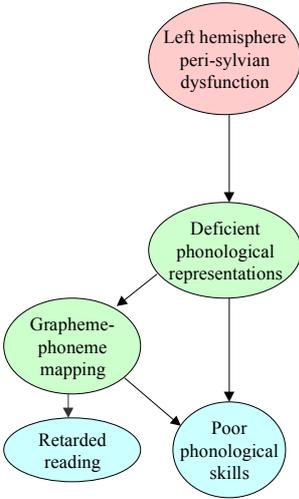


Fig. 1

Fig. 1. The phonological theory of dyslexia. Bubbles represent impairments at the neurological (red), cognitive (green) and behavioural (blue) levels; arrows represent causal connections.

Fig. 2

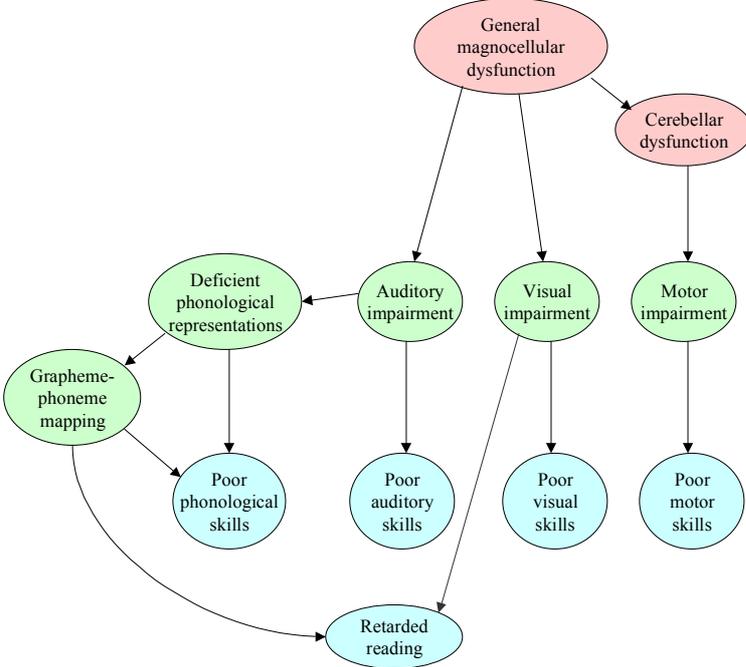


Fig. 2. The general magnocellular theory of dyslexia.