

Dyslexia: a deficit in visuo-spatial attention, not in phonological processing

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Developmental dyslexia affects up to 10 per cent of the population and it is important to understand its causes. It is widely assumed that phonological deficits, that is, deficits in how words are sounded out, cause the reading difficulties in dyslexia. However, there is emerging evidence that phonological problems and the reading impairment both arise from poor visual (i.e., orthographic) coding. We argue that attentional mechanisms controlled by the dorsal visual stream help in serial scanning of letters and any deficits in this process will cause a cascade of effects, including impairments in visual processing of graphemes, their translation into phonemes and the development of phonemic awareness. This view of dyslexia localizes the core deficit within the visual system and paves the way for new strategies for early diagnosis and treatment.

Misreading dyslexia

Developmental dyslexia, a specific difficulty in reading despite adequate learning opportunities, affects from 5 to 10 per cent of the population [1], but there are many manifestations of reading failure (Box 1). The etiology of dyslexia itself has been hotly debated for a long time. Theories have ranged from the reading disorder being a high-level learning disability, to a visual perceptual defect. Although it is generally recognized that a majority of poor readers have severe problems in phonological awareness [2,3], it is still an open issue whether the *causal* deficit in dyslexia is necessarily phonological. The debate is particularly timely in light of recent reevaluations – especially in the USA, Australia and UK – of how children learn to read.

In this paper, we present evidence for an alternative possibility, namely that a fundamental defect in the visual pathways, with or without a corresponding defect in the auditory system, can potentially cause a cascade of effects that can ultimately manifest as a reading problem, including phonological impairments. Understanding the mechanisms underlying dyslexia allows better informed policy making with regard to teaching and remedial intervention.

Phonological theory of dyslexia – controversies

Poor phonological processing is a core component of reading failure. Poor phonological skills are consistently

related to poor reading. Pre-reading phonological skills predict later reading failure, and phonological interventions have been demonstrated to be successful [2]. However, there are a number of reasons why poor phonological coding might not be the whole story, or even be an etiological factor in dyslexia. Some cases of dyslexia are clearly not phonological, for example where the reading errors are for irregular words, not non-words, and impairments in reading non-words are not always matched by deficits in phonological awareness [4,5]. There are reports of children [5,6] and adults with brain damage [7,8], who have difficulties in non-word reading but nevertheless exhibit good phonological awareness. Such evidence should, at the very least, lead us to question the causal link between a phonological deficit and dyslexia. Phonological representations have themselves been shown to be normal in dyslexics, but a task-specific deficit might occur in the access to such representations [9]. Moreover phonological sensitivity might occur partially as a consequence of reading instruction [10], and children who are explicitly taught the alphabetic code before reading instruction are better at phonological awareness tasks than children who are not taught letters and sounds prior to formal reading instruction [11]. Such findings should prompt us to explore additional cognitive/neurophysiological possibilities to provide a more coherent account for causal factors in dyslexia. We propose that the poor phonological sensitivity that is characteristic of many forms of dyslexia could be a failure of the sensory/cognitive system to fine-tune phonological representations through reciprocal cortical feedback in the way that occurs in normal reading.

Misordering of letters and reversal of letters in a word are common complaints from dyslexic readers, and sensitivity to spatial sequencing of the constituent components of text-like object arrays predicts reading in adults [12] and children [13]. Such difficulties in ascertaining the sequence of letters in words cannot be easily explained by phonological deficits. Studies of neurophysiological bases of pattern and object recognition indicate that such sequencing of letters is a non-trivial problem for the brain. Neurones in the inferotemporal neocortical areas that presumably mediate pattern recognition typically have large receptive fields [14]. Such position invariance helps to recognize an object irrespective of its location in the visual field, but this property, in the case of identifying letters in reading, inevitably leads to loss of information about the

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Box 1. Reading errors in dyslexia

We do not really understand what it is that a dyslexic reader sees. We know that some poor readers experience distorted text:

distorted text, can result in poor orthographic representations

Ambiguity regarding the invariance of some letters can make decoding difficult. We can simulate the decoding difficulties of poor readers where d's, p's, q's, and g's are interchangeable:

w hatwo ou l k t o p a
ulpy o p t o f y?

Even just reorganizing the internal letters of words can slow down decoding and make it difficult to read:

We all konw aobut the Cambrdirge Unviresity efefcet, we can raed it, but it is mcuh solwver and we mkae ltos mroe scacades and rgreesressoins.

Castles and Coltheart [5] made the distinction between 'Surface' and 'Phonological' dyslexia. In Phonological dyslexia, an impairment in reading pronounceable non-words (e.g. 'plimp'), indicates a difficulty in implementing *grapheme-to-phoneme correspondence* rules, or 'sounding-out' a word. Non-words have no representation in our mental dictionary, or *lexicon*. Thus in order to read such words, we must sound them out. The same challenge is faced when presented with a real word for the first time – as occurs when learning to read. Over repeated exposure, we might develop a visual representation of the word and we can access directly its *lexical* representation. Surface dyslexia then, is characterized by difficulties using the lexical route, with a corresponding problem in developing direct visual representations of words. Difficulties manifest in reading irregular words such as 'meringue' and regularization errors are typical, for example 'meringoo', since the surface dyslexics tend to over-rely on the phonological route. In fact most dyslexic readers have some degree of both Phonological and Surface dyslexia. Thus *some* dyslexic readers have exclusive difficulties in developing a visual representation of words and have 'pure' Surface dyslexia, but *most* dyslexic readers also experience some form of surface dyslexia. In addition, other poor readers suffer from a disorder called Visual Discomfort, in which sufferers describe very visual problems such as the words 'shimmering', or 'floating', and 'letters moving over each other'. The relationships between visual discomfort and the more cognitive aspects of reading remain unclear.

sequence of letters within a word. However, such information is clearly preserved to an excellent degree in normal reading. How is this achieved?

Visuo-spatial attention and dyslexia

There are two possible solutions to the above conundrum: (i) information of the relative location of each letter is somehow tagged to the process of recognizing that particular letter or (ii) letters are recognized sequentially, with only one or a few letters being processed at a time by the object recognition system and this temporal sequence preserves the spatial sequence of the letters. Although there is no direct evidence supporting the former possibility, the latter is consistent with many neurophysiological and psychophysical studies as will be described below.

Reading is a recent cultural trait in human history, unlikely to have been a direct product of evolution. Learning to read is thus an opportunistic training of neural mechanisms underlying a range of cognitive, perceptual and motor skills that had evolved for other purposes. One of the most relevant, neuro-cognitive functions used for reading might be what has been termed in the visual search literature as 'a spotlight of attention' [15]. In our usual cluttered world, targets of interest rarely possess unique features that help them to pop out from among distracting elements in a scene. Thus a serial search is usually undertaken that supposedly sweeps a spotlight of attention across a scene in a random fashion. The process helps to recognize one item at a time and also to 'bind' the different attributes of each object such as its form, color, depth, motion and size. We have suggested [16,17] that in reading, the same top-down search mechanisms are used to sweep the spotlight of attention serially over the letters of a word during the periods of fixation (see Figure 1 and animation in Supplementary Materials). The speed of serial visual search processes varies between 15 and 44 millisecond per item [18] and it has been noted that this is in the same ballpark as the average speed of reading

each letter in a text [16]. The time required by children to learn to read effectively could be due to the need for training the visual search mechanism, which is usually randomly and not systematically deployed across the visual field [18], to proceed in a sequential manner from left to right at a fine enough spatial scale across the letters of each word. Contrary to earlier beliefs, there is also now evidence that

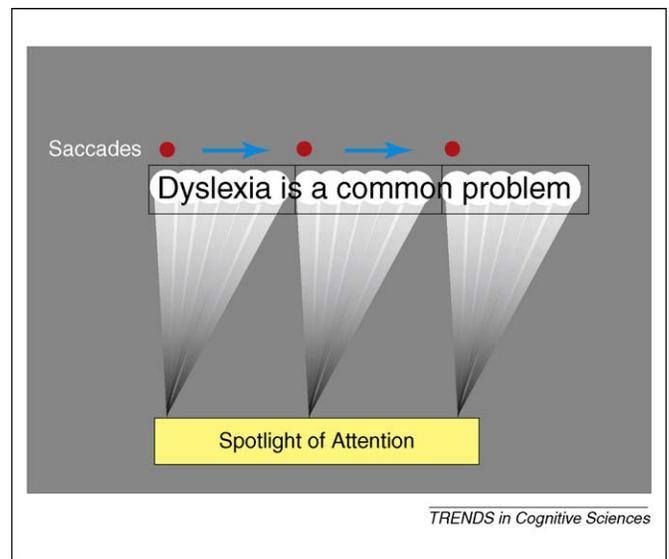


Figure 1. Role of visuo-spatial attention in reading. During reading, both normal and dyslexic readers make a number of saccades of about 25 milliseconds each, with intervening fixations each lasting about 250 milliseconds, although the number of saccades and the fixation durations are highly dependent upon the reading material and the skill of the reader, with poor readers making far more regressions, shorter saccades and longer fixations. During each fixation, approximately 7 or 8 letters are read [65], but they have to be processed in the appropriate sequence. We propose that in reading the brain uses essentially the same circuitry and attentional mechanisms that are used for serial visual search, but scans the spotlight of attention sequentially along the letters of the text during those periods of fixation. The letters are then processed in that temporal order to yield the spatial order of letters in a word. (See also the animation in supplementary data of how attention might shift during each fixation, shown at approximately one fourth the real speed).

words are not usually read as wholes, but as letters or small groups of them in a sequence [19].

Recent neurophysiological and brain imaging studies provide support for the existence of a neural system that could mediate a gating function that could have been co-opted for reading. Such modulation is most likely driven from areas of the dorsal stream, in particular from the posterior parietal cortex, which has been implicated in spatial attention [20]. The visual information coming into the striate cortex via the three major parallel pathways – magnocellular, parvocellular and koniocellular – is further channeled via two major streams, dorsal and ventral, projecting into the parietal and temporal cortices respectively [16,21]. The dorsal stream is believed to be concerned with spatial localization, movement, depth and visually guided action such as reaching and saccades, whereas the ventral stream is thought to be concerned with object recognition and perception [22]. The dorsal stream receives a fast input, ostensibly via the magnocellular pathways, which can potentially provide feedback to the primary visual cortex to gate the inputs entering the ventral stream areas (Figure 2). Consistent with this, serial visual search is more efficient with stimuli that involve the magnocellular channel, which provides the dominant visual input to the dorsal stream [23,24]. These gating inputs are presumably the means by which serial visual search functions: the dorsal stream using its cruder spatial resolution and information of locations of objects to sequentially select specific items in the visual field to be processed in the ventral stream.

If our scheme is a description of the crucial processing steps in reading that lead to grapheme identification and to subsequent matching of graphemes with phonemes, it follows that reading can be affected by a deficit at any step along the neural pathway, such as:

- (a) Poorer sampling density of the magnocellular system in the retina [25].
- (b) Specific deficit anywhere along the visual pathway to the dorsal stream [26,27].
- (c) Concomitant damage of other parallel pathways (eg. koniocellular) that could otherwise compensate for any magnocellular damage [28].
- (d) Damage in the dorsal cortical stream – to area V5/MT or the posterior parietal cortex [28–30].
- (e) Damage to feedback pathways from the dorsal stream areas to visual area V1 or the ventral stream.
- (f) Lesion at the site where the attentional modulation occurs.

A relationship to dyslexia has been demonstrated in some of the above cases (a, b and d), but in others (c, e and f) the link remains hypothetical. Nevertheless, a common outcome from all of the above is a poorer allocation of spatial attention. Indeed, visual search is impaired in dyslexic children [17,31], and many studies have identified a range of problems with visuo-spatial attention in children with dyslexia [31–35]. Consistent with the notion that reading utilizes the same mechanisms that are essential for selecting and stringing together small local elements in the visual scene, it has been demonstrated that sensitivity to the spatial sequence of word-like symbol

strings predicts reading proficiency in children [12] and adults [13].

The idea we have proposed [16,17] of a serial allocation of attention across the text in reading is also an essential part of the SERIOL model [36]. However the latter differs from ours in that it postulates a gradient of attention only during learning to read, but not in skilled reading, where the gradient is supposed to be automatically activated. Further descriptions of SERIOL and other models that seek to explain how letter order is encoded are beyond the scope of this paper.

Magnocellular deficits in dyslexia – controversies

Because intuitively one would expect the visual sensitivity in reading to depend upon the fine pattern recognition skills of the ventral stream, it is not surprising that schemes of a visual deficit in dyslexia restricted to the magnocellular pathway [26,27,37] have been controversial [38,39]. However, our scheme puts any such defect in perspective. The defect need not be with the magnocellular cells themselves, but it could be anywhere along the dorsal stream. Furthermore, any defect in the magnocellular pathway need only reduce the density of magnocellular cells in a few critical regions of the visual field, say for a degree or two to one side of the fovea, to cause a reading deficit. This can cause serious difficulties in the sweep of the focus of attention along the length of each word. Such a defect is not likely to be picked up by classical tests of magnocellular function. By the same token, unless a magnocellular deficit affects sampling at the critical visual field locations, widespread M (magnocellular) cell losses can co-exist with normal reading abilities. The reality about dyslexia is that it is not a unitary disease and there are probably a variety of manifestations that relate to the actual site of impairment. A recent study suggests that deficiencies at different levels of the pathway from retina to parietal cortex could relate to poorer performance in different aspects of reading [40]. The bottom-line of our scheme has been that dyslexia is a visual processing deficit in the proper recognition of the sequence of letters by an attentional mechanism, a suggestion that has been supported by a host of other studies [31–35,41,42]. A recent study that teased apart the correlation with reading ability of motion perception (in Ternus task) and attentional mechanisms (in a visual search task), found that a significant visual deficit in the dyslexic group was found only when the task required visual search, but not when it involved motion perception alone [43]. This again underscores the point that visual search and reading both exert considerable demands on the dorsal stream, but possibly in a very restricted region of the visual field [44].

Phonological deficits in dyslexia – cause or effect?

Despite providing a physiological framework for reading and its impairments, the scheme proposed here also has to confront and explain the severe phonological deficits seen in many dyslexics. It is possible that the development of phonological awareness itself might be at least partially dependent upon a normal input from the visual system into brain regions subserving grapheme–phoneme correspondence. Indeed there is substantial evidence indicating that

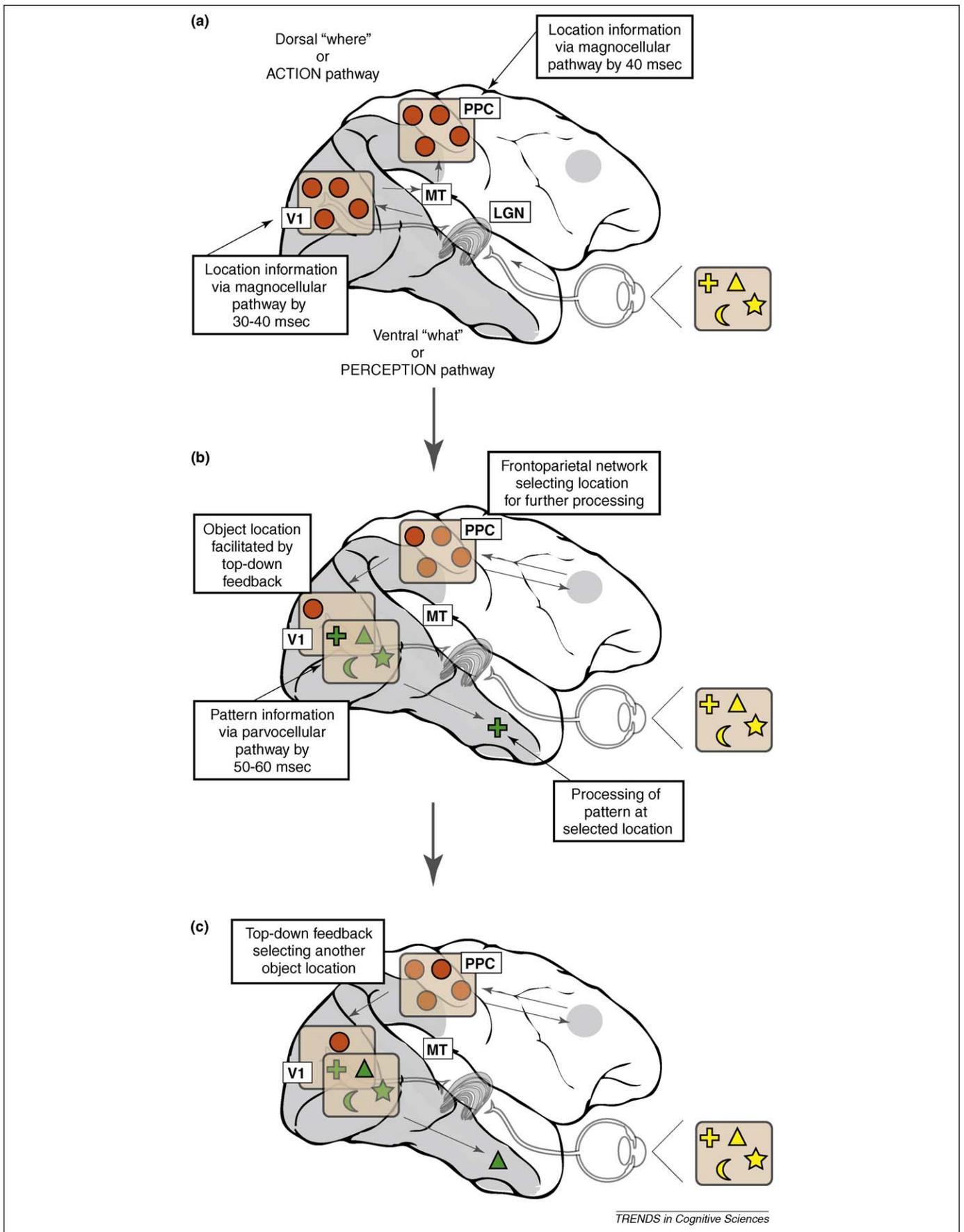


Figure 2. Neural circuitry that is exploited for reading is the system that normally enables serial visual search. (a) Visual pathways of the macaque, showing signals proceeding from the eyes to the lateral geniculate nucleus (LGN) and on to primary visual cortex (V1), with the faster signals of the magnocellular channel reaching the dorsal stream areas, namely middle temporal area (MT) and the posterior parietal cortex (PPC) as early as 40 milliseconds [66]. (b) The location information and crude pattern information in the input is used by the fronto-parietal network to select the object location to be facilitated among the signals reaching V1 via the slower

orthographic training itself seems to influence and/or enhance phonological awareness [45–47]. Thus demonstrations, that phonological awareness predicts future reading skills, could be due to orthographic training facilitating the development of *both* phonological awareness and reading skills. This might occur in one or both of two ways: (i) The segmentation of words into component graphemes might provide the necessary substrate for improving phonemic awareness through facilitating the process of grapheme–phoneme correspondence [48]. (ii) Multimodal integration of visual and auditory inputs during early development might be necessary to fine-tune the acoustic system and its ability to process changing sound inputs [16,35,49]. At the crux of this argument is an assumption that the development of a ‘healthy’ neural network – such as in skilled reading – is dependent on connections that build through all components of the network, and viable network nodes that allow strengthening and fine-tuning of all components of the network. That pre-readers at risk for dyslexia demonstrated reduced magnocellular sensitivity before they began to read [40], supports this notion of reciprocal feedback from the dorsal stream and the need for all components of the reading network to be fully functioning, because subtle impairments in one part of the network can have profound consequences for the development of the whole network.

It is conceivable that with our proposed inadequate parsing of a stream of text into graphemes in dyslexic individuals, areas of the brain that subservise grapheme–phoneme correspondence might show reduced activity with compensatory increases in other areas. Thus altered neural activity in a region in the reading impaired need not mean that these areas are the sites of the critical defect. Brain imaging studies on dyslexia can be seen in this light. Posterior regions, particularly the angular gyrus, supra-marginal gyrus, posterior part of the superior temporal gyrus and portions of middle occipital and middle temporal gyri show reduced activity in dyslexics, whereas the left inferior frontal gyrus shows increased activity [1]. In our view, the reduced activity in the posterior brain regions that are likely to be involved in grapheme–phoneme correspondence could be due simply to a poorer input into these regions. This is supported by evidence that early processing stages of the reading network are delayed or absent in dyslexics [50].

The visual processing deficit could, however, be part of a general temporal processing deficit, which would explain the high incidence of deficits in rapid auditory processing among dyslexic children [51]. Recently, learning disabilities including severe reading impairments, have been associated also with abnormal brainstem timing, indicating a deficit at a very early level of the sensory pathways [52]. However, dyslexics’ auditory deficits might be related more to their difficulties in focusing spatial auditory attention rather than impairments in low-level temporal processing *per se* [53], a finding also supported by studies that could not directly attribute the reading difficulty to

temporal processing problems [54]. Thus there might be many reasons for the impaired phonemic awareness in the dyslexic population, whereas reading difficulties themselves could have their origins primarily in abnormal visual attentional mechanisms. Poorer phonemic awareness, due to a variety of reasons including abnormal visual processing, could make the reading difficulty worse without being the major cause of it. This might explain the improvement that has been reported in some cases of phonological remediation [55]. This study’s claim that the computer-based exercises that target auditory temporal processing, as in the commercially marketed Fast ForWord, improve reading skills, has been highly controversial. A number of independent studies that have incorporated extensive controls have been able to show improvements in auditory processing from Fast ForWord and similar training regimes, but no comparable improvements in reading skills [56,57]. Thus, although a general temporal processing deficit might cause slower processing of auditory signals, the primary reason for the reading difficulty in dyslexic children might arise within the visual system and be exacerbated by any concurrent auditory deficit.

Finally, the recent finding in 7–12-year-old children that contrast responsivity in area MT+ (but not in V1) is significantly correlated with phonological awareness [58] indicates that magno-dominated dorsal stream activity may be crucial for reading. In the light of our hypothesis, it is interesting that this study showed that there was little correlation between MT+ responsivity and rapid naming, age and IQ and among the various reading related measures tested, but the strongest correlation was with phonological awareness. This underscores our suggestion that development of grapheme–phoneme correspondence and phonological awareness benefit from intact dorsal stream activity in the visual system.

Causality

As Castles and Coltheart [5] point out in their comprehensive paper on the role of phonological coding in reading, the burden of test of causality falls upon researchers being able to demonstrate in longitudinal designs that deficits both precede and predict reading development, and that appropriate training facilitates reading acquisition. Such studies in the context of a dorsal stream deficit in dyslexia are in their infancy, but these studies are encouraging. Children who are less sensitive to coherent motion in preschool subsequently go on to have poorer literacy skills in grade 1 [59]. Contrast sensitivity in kindergarten predicts reading ability two years later [60]. Temporal order judgment in preschool children predicts their single word reading skill in Grade 1 [61]. Pre-reading children at risk for dyslexia – that is, who have a first-degree relative with dyslexia – are significantly less sensitive to coherent motion and visual frequency doubling compared to a large unselected sample of same-age children [40].

parvocellular channel. The object in the selected location is then processed in the ventral, ‘what’ pathway and recognized. Neurons in the ventral stream are known to have longer latencies [67]. (c) Orchestration of serial search by the fronto-parietal network chooses one location after another until the target is found. Consistent with such neural substrate for a feedback from the dorsal stream, physiological studies have shown both focal modulation of striate cortical activity by attention [68,69] and a neural mechanism for a fronto-parietal network controlling spatial attention in early visual areas [20,70].

Box 2. Future directions

- In order to provide a comprehensive argument that poor visual coding is a core deficit in dyslexia, we need more extensive studies looking at whether visual training is effective in remediating dyslexia.
- Reading is a multi-sensory, multi-cognitive task. Further research should be targeted at determining which aspects of dorsal coding are intrinsic to which aspects of the reading process.
- The literature to date suggests that not all dyslexics demonstrate a dorsal deficit. However, the studies conducted might not adequately target the very subtle visual deficits that some dyslexics might experience. Thus further research is required to identify such specific deficits.

A small number of training studies have also been conducted. Fischer and Hertnegg [62] trained children for three weeks on a saccadic control task. The dyslexic children's saccadic control normalized, to be consistent with the non-dyslexic control group. However, there was no measure to indicate if the visual improvements translated into improved reading. Solan *et al.* [63] trained poor readers on visuo-perceptual tasks. They demonstrated that children improved on reading comprehension and word attack measures after the intervention. However, the children were also "provided the opportunity to develop improved cognitive strategies" (p. 644), making it difficult to evaluate the impact of the visual training on reading independently of the increased exposure to words presented in a novel way. Similarly, Lorusso *et al.* [64] used a technique in which the children were required to make a rapid attentional shift to a stimulus in either the right or left visual field. They demonstrated increases in reading accuracy and speed compared to a control group that was exposed to more traditional remediation methods. However, again the stimuli in the task were words that the children had to identify that "became increasingly difficult in terms of word length and complexity of spelling" (p. 201). Thus, although such results seem promising, there is a dire need for controlled studies that investigate the impact of visual training on dorsally mediated tasks in reading development and remediation. Moreover, studies need to be conducted to determine for example, whether dorsal training facilitates reading acquisition in normally developing readers, and the resilience of such dorsal visual training on reading development over time (Box 2).

Consistent with the need for further research is the notion of reciprocal feedback, that is, the degree to which reading *per se* facilitates the development of visual coding in the same way that it seems to do in phonological coding. We are in the process of investigating whether individuals who have never learned to read also demonstrate less sensitive dorsal functioning.

Concluding remarks

The critical deficit in developmental dyslexia might be one that affects the focal visual attentional mechanisms essential for efficient reading. The poor phonological awareness that is seen in most dyslexics might not be the cause of the reading difficulty, but could be the result of the poor orthographic inputs feeding into the regions mediating

grapheme-phoneme correspondence and due to a general temporal processing deficit affecting all modalities.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at [doi:10.1016/j.tics.2009.12.003](https://doi.org/10.1016/j.tics.2009.12.003).

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