OPINION

Sensory theories of developmental dyslexia: three challenges for research

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Abstract | Recent years have seen the publication of a range of new theories suggesting that the basis of dyslexia might be sensory dysfunction. In this Opinion article, the evidence for and against several prominent sensory theories of dyslexia is closely scrutinized. Contrary to the causal claims being made, my analysis suggests that many proposed sensory deficits might result from the effects of reduced reading experience on the dyslexic brain. I therefore suggest that longitudinal studies of sensory processing, beginning in infancy, are required to successfully identify the neural basis of developmental dyslexia. Such studies could have a powerful impact on remediation.

Children with developmental dyslexia fail to acquire efficient reading and spelling skills despite adequate tuition and an absence of overt sensory and/or neural deficits. Dyslexia is found in all languages studied so far, although its manifestation differs with orthography[1]. Nevertheless, in all orthographies, dyslexia disadvantages both school learning and later employment. A cost–benefit analysis in the United Kingdom suggested that having dyslexia incurred costs of over UK£80,000 on lifetime earnings[2]. Therefore, identifying the underlying cause (or causes) of dyslexia would enhance the life chances of affected children.

Proposals that visual and/or auditory dysfunction underlie dyslexia are driving much current research (see Further information). However, these theories are challenged by three key facts. First, learning to read trains sensory and attentional processes, making it difficult to determine whether sensory deficits are the cause of dyslexia or an effect of the reduced reading experience of individuals with dyslexia. Second, sensory processes underlie all childhood learning; therefore, evidence of putative sensory deficits that affect only reading is not persuasive. Similarly, a sensory deficit that also occurs in other developmental disorders in which reading is unaffected lacks explanatory power. Third, the protracted developmental time course of reading acquisition and its relatively late onset (following tuition from the age of five or later) means that the cognitive effects of sensory dysfunctions should be visible before the onset of reading. Indeed, some of the genes associated with dyslexia are involved in early cortical neuronal migration and are likely to impair sensory processing from infancy onwards[3,4]. For example, an important longitudinal study of Norwegian preschoolers at family risk for dyslexia found atypical development of sensory structures but not of the temporo-parietal areas that are usually identified as atypical in neuroimaging studies of older individuals with dyslexia, implying that another factor (such as impoverished reading experience) underlies the latter changes[5]. Indeed, if sensory deficits are a cause of dyslexia, they should be detectable even at birth.

In this article, I address these three challenges by scrutinizing prominent sensory theories and applying three tests. First, I assess the extent to which developmental research designs relevant to establishing causality have been applied[6]. These are outlined in BOX 1, and enable the sensory consequences of reduced reading experience to be identified. In general, once the studies that led to the proposal of each theory have been presented, further studies testing these theories that did not use these research designs are omitted from this article (with the exception of chronological age (CA)-matched studies showing that dyslexic performance is unimpaired). Second, I consider the evidence for systematic and hypothesis-driven effects of the proposed sensory deficits on related cognitive skills. Last, when available, I discuss data from longitudinal and infant studies to assess evidence for the early fingerprints of sensory dysfunction. Developmental disorders of learning such as dyslexia represent the extreme bottom end of the normal distribution of a culturally acquired skill (reading). Preliterate infants and children and illiterate (unschooled) adults are groups that are independent of this distribution, and so tests of sensory theories in these populations are of particular importance for identifying causality. It is also important to note that the behavioural manifestations of a sensory impairment can change over the course of development.

Neurotypical reading development

Learning to read is sometimes erroneously considered to be a visual skill, but it is actually a linguistic process[7]. A brain that can read gains linguistic information from a visual code that represents speech. Hence, speech processing skills are integral to reading. Phonological recoding skills (which allow children to convert visual symbols to sound) have a crucial early role in accessing meaning from print[8]. These skills develop extremely rapidly in some languages (within the first 3 months of instruction for consistent alphabetic orthographies such as Finnish and Italian)[1,8,9], but they develop much more slowly in inconsistent alphabetic orthographies (such as English, in which whole-word recognition strategies are also useful). Individual differences in 'phonological awareness' — the ability to reflect on the constituent sound elements in words — predicts how well and how rapidly phonological recoding skills are learned in all languages studied so far[1,8] (BOX 2). By contrast, individual differences in visual processing (for example, in visuospatial skills) are only occasionally correlated with individual differences in reading acquisition for typically developing children[10–12].

Box 1 | **Research designs that can disentangle cause from effect in dyslexia**

The reduction in reading experience that is inherent in being dyslexic can itself cause differences in sensory processing between participants with dyslexia and controls. Adopting the following research designs can to some extent control for the effects of reading experience on the brain. If similar outcomes are found using combinations of these designs, causality is more likely to be present.

Reading level-matched design. In these studies, children with dyslexia (10 year olds, for example) are matched to younger children (such as 7 year olds) who have attained the same level of reading. If children with dyslexia perform worse than the younger reading level-matched children, this suggests a causal role for the factor being investigated, as the children with dyslexia have higher chronological and mental ages and better metacognitive skills. Subsequent longitudinal and training studies are required to establish causality. A result in which both groups perform equally is causally ambiguous. The higher metacognitive abilities of the children with dyslexia might be masking a deficit.

Research with illiterate adults. Illiterate adults have never been taught to read and hence have never developed a specialized letter processing system. Nevertheless, they have grown up in cultures in which letters and print are ubiquitous. If illiterate adults show no deficit in a sensory task involving letters and words in which children with dyslexia perform worse than age-matched controls, this is a good indication that the sensory deficit in the children with dyslexia is a product of reduced reading experience.

Research with pre-readers. Infants and pre-reading children provide a critical test for causal sensory theories of dyslexia. The candidate mechanism should already show impairments in pre-readers who go on to be diagnosed with dyslexia, and should systematically affect reading acquisition in these children once instruction commences.

Longitudinal studies. The best studies, but also the rarest, follow the same children over the whole learning trajectory to establish temporal cause versus effect.

Training studies. The strongest test of causation in development is an intervention study. If sensory process A causes cognitive deficit B, then providing training in A should ameliorate B. A control group should receive matched training omitting the key sensory variable.

Cross-language studies. If a sensory deficit is a primary cause of dyslexia, it should be found across languages. The manifestation of the deficit might vary with language, but this variability should be predictable from normative development. For example, auditory deficits might affect speed rather than accuracy in phoneme awareness tasks in consistent alphabetic orthographies.

Testing effects on other cognitive systems. Any putative sensory cause of dyslexia should have predictable and hypothesis-driven effects on other aspects of cognitive development. For example, an auditory processing deficiency should also affect music cognition.

Cross-language comparisons estimate that between five and six years of schooling are required for most children to become fluent, silent and efficient readers[13]. After around 10 years of age, cross-language variations in literacy depend on social and economic factors. By contrast, developmental dyslexia occurs at around the same rate (7%) across languages[2], and seems to be unrelated to social or economic factors. Difficulties in the phonological recoding of print to sound lie at the heart of dyslexia in all languages studied[1,9]. A dominant view is that these difficulties arise from impaired or atypical phonological representations of spoken word forms in the neural mental lexicon[14,15]. These atypical representations impair the cognitive achievement of phonological awareness (BOX 2). Because the acquisition of phonology is multimodal from infancy, with auditory learning supported by visual information from the lips and face, and motor information from learning to talk[16], in principle both visual and auditory sensory dysfunction could cause the cross-language 'phonological deficit' that characterizes developmental dyslexia.

Given the core developmental role of phonological recoding, however, a child who is reading less text will accumulate far less experience of the component skills of reading. For example, visual scanning, oculomotor control and associated visuospatial attention skills will all be practised less, as will phonological recoding itself. Such a child would quickly begin to show apparently atypical sensory processing. These interpretive issues are magnified when adults with dyslexia are studied instead of children. By adulthood the dyslexic brain has had 15 years or more of impoverished reading experience. In the cognitive domain, it is well established that the 'rich get richer' during the development of reading (for example, children who read more develop better vocabularies, which in turn enhances their phonological recoding skills)[17]. These effects also apply to sensory processing, compounding the need for sensory dysfunction theories to rigorously test causality. Furthermore, the effects of reduced reading experience may be expected to vary across languages. For example, a child reading a transparent script such as Italian, in which letter-by-letter recoding to sound is a successful early strategy, would experience far more spatial serial processing of letters than a child reading an opaque script such as English. Therefore, a sensory dysfunction that causes dyslexia should be found across languages, and its effects should vary in systematic ways according to variations in orthography and phonology.

Magnocellular theory

When visual information leaves the occipital lobe, it follows two main pathways. The ventral pathway is involved in object identification and recognition, whereas the dorsal pathway primarily processes spatial position. The dorsal pathway develops more slowly[18] and encompasses the subcortical magnocellular system, which detects motion, low-frequency spatial information and high-frequency (fast) temporal information: information that is critical for representing and parsing the visual field. The magnocellular theory of dyslexia states that deficits in magnocellular visual processing are a primary cause of the disorder[19].

The magnocellular theory developed from psychophysical and post-mortem data suggesting the abnormal function and appearance of magnocells in the lateral geniculate nucleus in individuals with dyslexia[20,21]. Importantly, these early data relied on CA-matched studies, in which readers with dyslexia were matched to good readers of the same age. Because reading experience differs dramatically in CA-matched designs, the results of these studies are completely ambiguous with respect to causality. The magnocellular deficit was reported to extend to dorsal cortical systems in a further series of CA-matched studies that revealed dyslexic deficits in perceiving coherent motion in random dot arrays, in binocular control and (in fewer studies) in other 'magnocellular' tasks such as the frequency-doubling illusion[22–24]. These studies led to the proposal that difficulties in vergence control (that is, directing both eyes to the same point in space during the sequential tracking of print) led to 'reversed' letter effects when reading (reading 'saw' as 'was', for example). According to this proposal, difficulties in guiding eye movements caused unstable or jumbled visual

Box 2 | **Phonological awareness and the oscillatory hierarchy**

Linguists identify various levels of phonology (see the table). The infant brain is sensitive to many of these levels, but reflective awareness of phonology develops more slowly. Measured by the ability to identify and manipulate phonological units, the development of phonological awareness follows a hierarchical sequence in all languages studied so far, and shows a causal relationship with literacy[1]. Reflective awareness of phonemes is not the natural end point of development, but depends on direct tuition and alphabetic learning (including learning to spell). Illiterate adults[101] and Chinese adults who have learned to read logographically[107] (matching characters to meaning) show poor phoneme awareness. Although infants can make categorical phonemic distinctions, grouping acoustically distinct sounds (called allophones) together and treating them as the same phoneme, the sounds reflected by the alphabet are an abstraction from the acoustic signal. For example, English spelling convention uses the letter 'p' to represent the second phoneme in words such as 'spin' and 'spoon', and the consonant 't' to represent the first phoneme in words such as 'track' and 'tray', even though the acoustic sounds are closer to 'b' and 'ch' (REF. 108). Indeed, beginning spellers misrepresent these sounds, writing the acoustically accurate 'sbn' or 'chrak'. Alphabetic learning has lasting effects on the brain; adult oral language processing is affected by spelling knowledge[109], whereas pre-reading children do not show orthographic effects during oral language judgements[110].

Recent theories of speech processing based on cortical oscillations identify an oscillatory hierarchy that approximately parallels the phonological hierarchy summarized here. The oscillatory hierarchy underpins the neural encoding of speech[111], and might provide an acoustic corollary of phonological units. Newer linguistic theories, such as those that propose 'rich phonology', argue that representations for words are stored in continuous time as high-dimensional spectrotemporal auditory patterns[112]. According to such accounts, phonological awareness is an emergent property of acoustic structure[79]. By contrast, traditional linguistic theories assumed that neural phonological representations involved sequential collections of phonemes "akin to a pronouncing dictionary" (REF. 113). Newer perspectives suggest that the preliterate brain may code language quite differently, prioritizing speech rhythm[77,79]. Clearly, the field is wide open for a principled application of the oscillatory hierarchy to the phonological 'deficit' in dyslexia, across languages. Phonological awareness of the highest levels in the hierarchy shown here (phrasal and syllable stress, which are rarely marked in orthographies) have been particularly understudied.

Phonological level	Oscillatory frequency (EEG band)	Example(s)	Age at which reflective awareness develops
Intonational phrase	~1 Hz and lower	Who's a pretty boy then?	Not yet ascertained
Stressed syllable	~2 Hz (delta)	PE-ter PI-per PICKED a PECK of PICK-led PEPP-ers	Not yet ascertained
Syllable	~5 Hz (θ)	• an-i-mal • wig-wam	2–3 years
Onset-rime	~cued by rising θ-slope	• c-at • str-eam • cl-amp	3–4 years
Phoneme	~35 Hz (γ)	c-l-a-m-p	With alphabetic tuition

representations of words and hence reading difficulties. Although all children go through a developmental stage of letter reversal when learning to read[25], imaging research in children and adults with dyslexia revealed atypical (reduced) neural activity in the brain area related to motion processing (visual area 5 (V5) in the extrastriate visual cortex; also known as middle temporal (MT) area)[26]. This was taken as further evidence for the theory[19], even though CA-matched designs were used, and thus cause and effect cannot be disentangled.

There are few reading level (RL)-matched or longitudinal studies of magnocellular processing, and few cross-language or intervention studies. Only one RL-matched study has reported a positive result, based on the frequency-doubling illusion[27]. This Italian study found that children with dyslexia had higher thresholds in the illusion than both CA- and RL-matched controls[27]. By contrast, a CA-matched German study measured vergence control and sequential tracking directly by asking children to identify identical letters in meaningless letter strings[28]. Children with dyslexia were as good as the CA-matched controls and showed equivalent eye-movement patterns. Furthermore, their ability to perceive coherent motion was not related to their performance. A Dutch longitudinal study of pre-reading children at familial risk for dyslexia, however, reported a significant relationship between coherent motion detection and reading one year later in control children[29]. By contrast, a study of English-speaking preschool children (who did not have dyslexia) found no relationship between later reading and performance on the coherent motion task[24]. Instead, individual differences in normative performance with the frequency-doubling illusion were related to later reading skills.

More systematic application of the research designs described in BOX 1 to more languages might clarify this currently mixed picture. It is also important that studies include, as a control, parvocellular tasks that are of equal difficulty to the magnocellular tasks; some data show that when task difficulty is equated, participants with dyslexia are impaired in both types of task, weakening claims that magnocellular dysfunction has a causal effect[30].

The results of an important study that investigated the effects of training phonological recoding skills pose further difficulties for the magnocellular hypothesis[31]. This study used neural imaging to measure activity in V5 before and after training of children with dyslexia, and made comparisons with CA- and RL-matched controls (FIG. 1). Training consisted of tutoring programmes that taught the component skills of either reading (such as phonological skills) or mathematics. Before training, children with dyslexia showed reduced V5 activation compared with CA-matched children, but equivalent activation compared with RL-matched children. Following phonological training, which improved reading skills in the group with dyslexia, V5 activity in this group increased. Importantly, mathematics training did not improve reading skills or V5 activity, suggesting that the relationship between magnocellular function and reading is mediated by reading experience[31]. Sensory processes such as oculomotor control, visual attention and spatial position encoding are all trained when print is recoded to sound, and all of these processes are subserved by the magnocellular visual system.

As yet, no studies have examined systematic effects on other cognitive systems that depend on magnocellular function. One obvious prediction is that spatial orienting in infancy should be affected[32]. Additional longitudinal studies are required, examining magnocellular function in infants and pre-readers at family risk for dyslexia. Studies of magnocellular function in illiterate adults are also absent, as are studies utilizing the

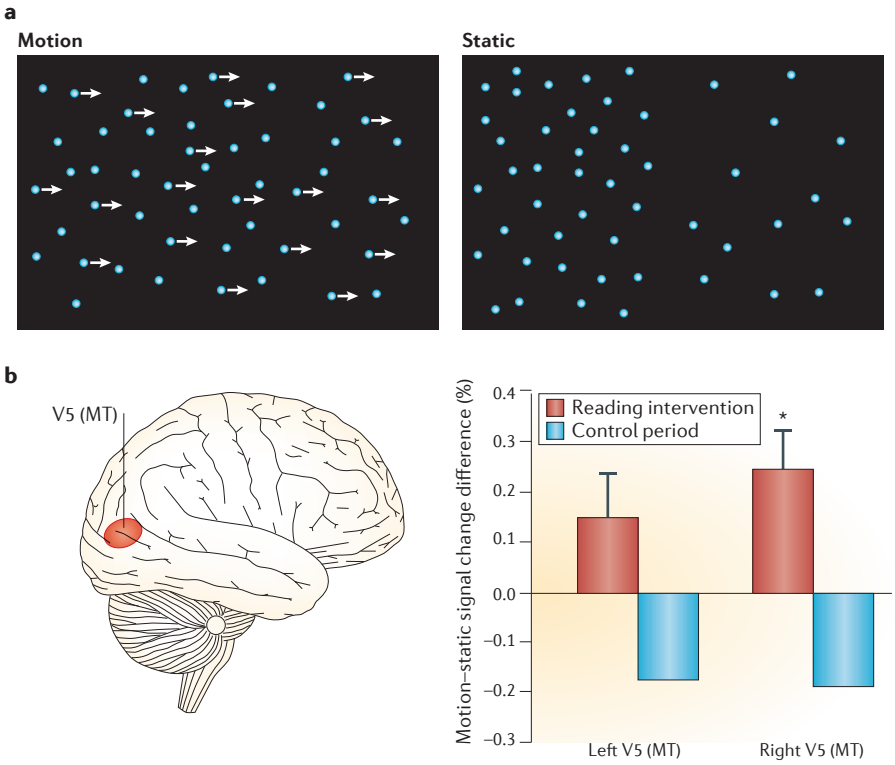

Figure 1 | **Coherent motion detection and visual area 5 activation in dyslexia.** Because motion is detected by the magnocellular system, coherent motion detection has been the key task used in studies of magnocellular deficits. Depressed neural activity in visual area 5 (V5; also known as the middle temporal area (MT)) during task performance has been used as the biological marker of such deficits. **a** | In the coherent motion task, the child has to detect the direction of motion in a dense array of moving dots (left panel). For scanning studies, the usual control display shows static dots (right panel). **b** | In a study providing phonological training[31], it was demonstrated that activity in right V5 (left panel) during a coherent motion task showed a significant increase after the intervention period (right panel), that was not seen in the control periods. A significant change in activity (indicated by an asterisk) followed a phonological intervention, so this study suggests that the differential performance of children with dyslexia in coherent motion tasks is mediated through reading. Part **a** is courtesy of Guinevere Eden, Georgetown University, Washington DC, USA. Part **b** was published in *Neuron*, **79**, Olulade, A. O., Napoliello, E. M. and Eden, G. F., Abnormal visual motion processing is not a cause of dyslexia, 180–190, © Elsevier (2013).

research designs outlined in BOX 1 with readers of non-alphabetic scripts. Because the grain size (the unit size at which spelling maps to phonology) of the visual code is usually larger in non-alphabetic scripts, processes such as fine oculomotor control might receive less training via reading experience in these scripts. Hence, it might be possible to demonstrate parametric effects of orthography on V5 activation (that is, dyslexic readers of languages such as Chinese might show smaller V5 effects). Along these lines, a study in Hebrew found no magnocellular deficits in dyslexia in a series of perceptual tasks with adults in a CA-matched study[33].

Despite its prominence, therefore, the magnocellular theory seems to identify a sensory dysfunction that is primarily related to reduced reading experience rather than dyslexia per se. In addition, immaturity of the dorsal visual system seems to characterize a number of developmental disorders, including dyslexia, autism, Williams syndrome and dyscalculia[34]. Dorsal deficits are hence not specific to dyslexia, further weakening the causal claims of the magnocellular theory.

Impairments in visual attention

The visual environment presents far more information than can be processed effectively. Visual attention enables the selection for processing of information most relevant to ongoing behaviour and the suppression of information that is less relevant (inhibition). Two prominent theories suggest that impairments in visual attention might underlie dyslexia.

Impaired visual attention span. The span of visual attention is the amount of visual information that can be maintained across brief disruptions to sensory input (for example, during blinking or saccades). Visual span is vital for coherent experience. One theory suggests that visual span capacity is reduced in developmental dyslexia and that this primary impairment in the ability to process multiple-item arrays limits reading development[35–37].

Visual attention span is typically measured as the number of individual elements that can be processed simultaneously in the 'attentional window' — the region in visual space to which selective attention is being directed. A classic span task presents an array of five elements (such as five letters or digits) very briefly, and then records reaction time when participants name either all of the elements in the array (full report) or single elements at different cued positions (partial report). Usually, reaction times show an M-shape (faster report for the elements in positions one, three and five), and accuracy shows a W-shape (greater accuracy at these positions). Reduced speed and accuracy at positions two and four is argued by some to reflect crowding effects — visual interference from the elements flanking the target. Crowding effects can be reduced by surrounding the target with congruent stimuli that share its global contours (lines or curves, for example), producing congruence effects.

Crowding effects seem to be larger in Italian children with dyslexia than in controls in CA-matched designs[38,39], and CA-matched visual span studies with French-speaking children show a reduced visual span in children with dyslexia[35,36]. Furthermore, one French RL-matched study found that severely impaired older children with dyslexia (aged 9–14 years with an average reading age of 7 years) showed poorer phonological recoding skills and visual spans than RL-matched controls (aged 8 years)[37]. As outlined above, such positive results in CA-matched designs could reflect reading experience. In addition, the classic visual span task uses letters, which are known to be processed less efficiently by children with dyslexia[40–42], and the oral report method requires rapid access to phonology, which is also impaired in dyslexia. Hence, any visual attention task based on letters or requiring oral reporting is inherently ambiguous with respect to causality[40].

When the research designs summarized in BOX 1 are applied to visual span theory, the confounding role of reading experience becomes apparent. For example, a recent Portuguese study compared illiterate adults

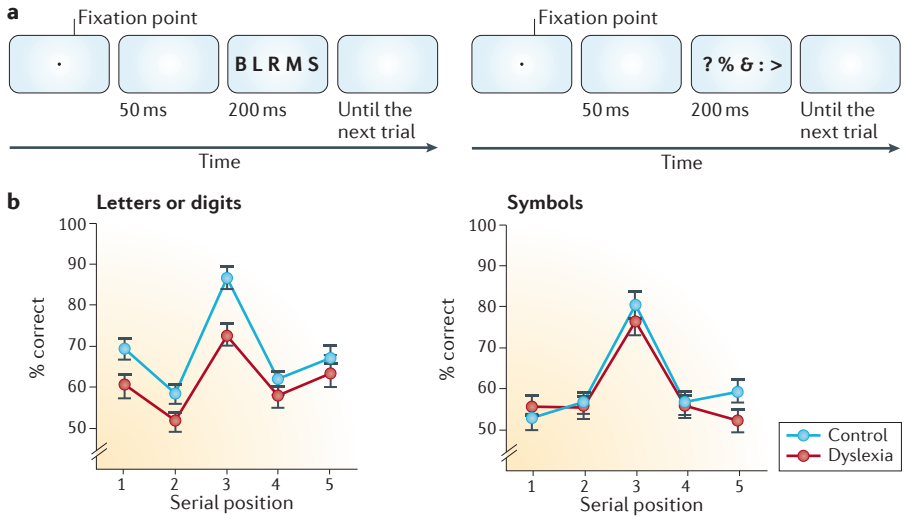

Figure 2 | **Performance in the visual span task in dyslexia.** **a** | The visual span task usually tests visual short-term memory capacity using an array of five letters or digits (left panel), but tests can also use colours (not shown) and symbols (right panel). The figures illustrate screen images from a single trial during the task. The child either reports the whole array or the letter/symbol at a particular location. **b** | The performance of children and adults with dyslexia on this task in chronological age-matched studies is reliably impaired (left panel; graphs shown mean percentages with error bars indicating the standard error). However, when symbols or coloured dots are used instead of letters/digits, no reduction in visual span capacity is found (right panel)[40], and children with dyslexia perform at equivalent levels to age-matched children. This makes it unlikely that the ability to process multiple visual elements in parallel is a primary impairment causing developmental dyslexia. Left panel of part **a** is adapted from Valdois, S., Lassus-Sangosse, D. and Lobier, M., Impaired letter string processing in developmental dyslexia: What visual-to-phonology code disorder? *Dyslexia*, **18**, 77–93, John Wiley & Sons, Ltd. © 2012 John Wiley & Sons, Ltd. Part **b** is reprinted with permission from REF. 40, John Wiley & Sons, Ltd.

with children with dyslexia and typically reading (RL- and CA-matched) controls in a visual attention task measuring congruence effects[43]. Both letter stimuli and pseudo-letter stimuli were presented. All groups showed congruence effects for pseudo-letter stimuli; however, no congruence effect was found for the illiterate adults when letter stimuli were used. A significant congruence effect was found for the children with dyslexia using letter stimuli. This result suggests that reading experience governs the emergence of congruence effects for letters. Indeed, the strongest predictor of the size of the congruence effect for letter stimuli in children with dyslexia was phonological recoding ability[43].

As described above, it is important to determine whether there are systematic effects of a proposed sensory deficit on stimuli that are not letters or words. The classic five-element visual span task was used in two studies[40,44] that compared visual span for arrays of letters and digits with visual span for non-alphanumeric stimuli (FIG. 2). In a CA-matched design, children with dyslexia showed reduced visual spans for letters and digits. However, their performance was identical to that of CA-matched controls for unfamiliar symbols[40] and coloured dots[44]. Hence, despite their reading difficulty and reduced reading experience, French children with dyslexia showed preserved visual spans for materials that had not been recoded to sound with the same frequency as letters and digits. Again, this supports a non-causal interpretation of visual span deficits. Currently lacking are training studies or longitudinal studies with at-risk infants or pre-readers (BOX 1).

It is important to note that even if reduced visual attention span, increased crowding effects and letter congruence effects are a product of reduced reading experience, the reading ability of individuals with dyslexia might be improved if these visual factors are minimized. Increased spacing between letters, hypothesized to reduce crowding, significantly improved sentence reading accuracy for 10-year-old French and Italian children with dyslexia[45]. However, an Italian RL-matched control group did not show accuracy benefits from increased spacing (no French RL-matched group was included). This suggests that increased spacing, a typical feature of books for younger readers, has a selective benefit in dyslexia. Replication of this finding in other languages is required; more complex orthographies, such as English, that require orthographic processing at larger grain sizes might not show equivalent between-letter spacing effects.

Impaired visuospatial attention. A second visual attention theory argues that a 'spatial cueing' deficit (an impaired ability to orient spatial attention) causes dyslexia[46–48]. An efficient attentional system can orient rapidly in response to exogenous cues[49]. Exogenous cues are independent of the information being attended to (such as letter colour when reading) but have an automatic sensory 'cueing' effect, facilitating stimulus detection and response time by orienting attention to the most informative aspects of the visual field without moving the eyes. According to visuospatial attention theory, attentional shifting is 'sluggish' in individuals with dyslexia[50]. It is suggested that the dyslexic brain cannot move smoothly from letter to letter while suppressing flanking letters when recoding print to sound.

Even infants show visual cueing effects[49]. When visual array sizes exceed visual span capacity, infants as young as 5 months old show processing benefits from exogenous cues. However, attentional cueing is not an all-or-nothing process even in infancy, and experience of cues with different perceptual characteristics (motion versus a stationary square, for example) is required for attentional facilitation. Hence, it is important to establish that reading experience per se is not facilitating typically developing children's use of spatial cues in the paradigms used to establish the dyslexic spatial cueing deficit.

The classic spatial cueing paradigm compares the effects of 'valid' (informative) and 'invalid' (uninformative) cues. These cues are usually presented with different time delays between cue and target presentation. A 'cueing effect' results in enhanced orienting (measured via faster reaction times or better accuracy) to the target. A series of studies involving Italian children with dyslexia has shown impaired spatial orienting of attention in dyslexia[47,48,51,52] (FIG. 3). Importantly, however, only the children with dyslexia who have phonological recoding deficits showed a spatial cueing deficit in these studies[47,48].

One strength of the Italian studies (FIG. 3) is the inclusion of RL-matched controls and the use of longitudinal designs (BOX 1). For example, a key study involving 22 10-year-old children with dyslexia showed no spatial cueing effect at 100 ms delay in 13 children with dyslexia with poor phonological recoding,

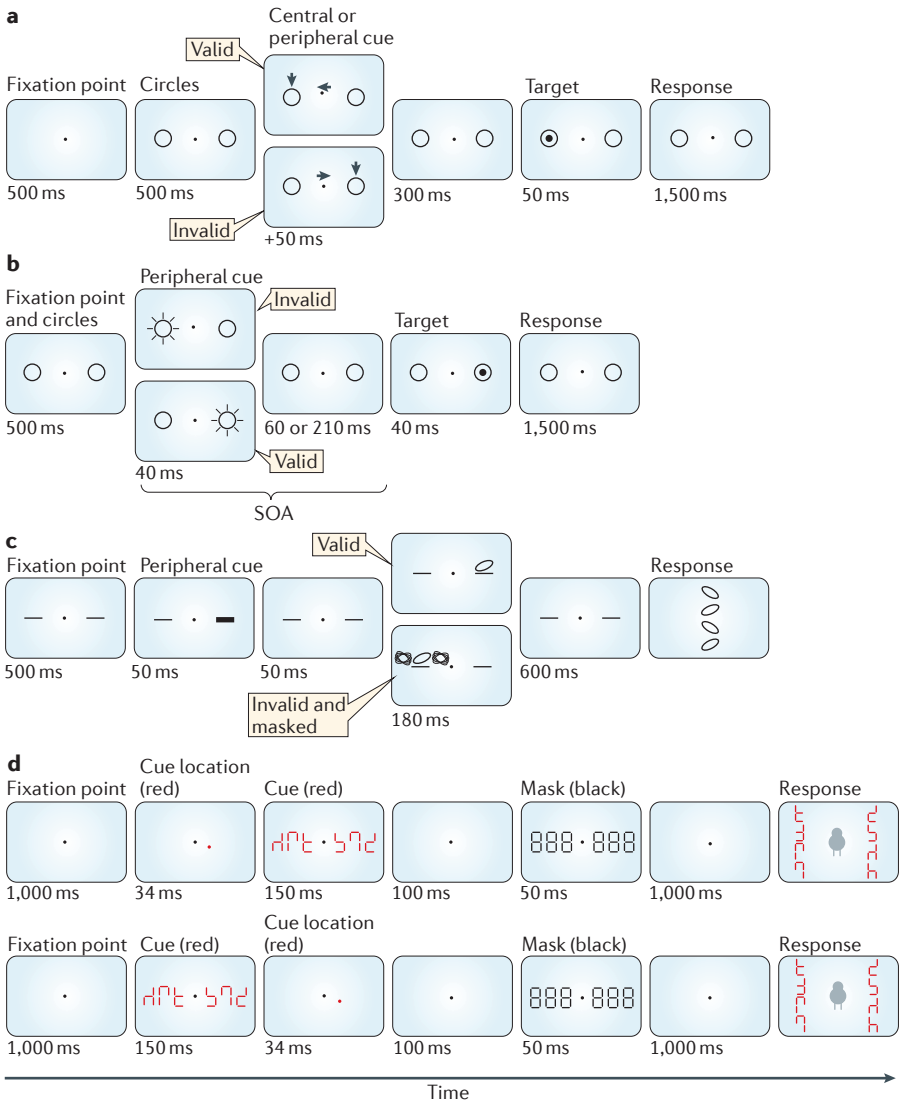

Figure 3 | **Visuospatial cueing tasks.** A range of Posner cueing tasks have been used in studies of the visuospatial attention deficit in dyslexia, four examples of which are shown here. The figures illustrate potential screen images from a single trial during each task. Typically, a target's future location is cued briefly, and the dependent measure is whether this spatial cue facilitates subsequent detection of the target. Some studies require the participant to respond to a target presented to the left or right visual field, and compare the reaction time when a valid cue is presented ahead of the target from the reaction time when an invalid cue is used (parts **a** and **b**)[47,48]. By varying the length of the interval between cue and target (350 ms in part **a** and 100 ms or 250 ms in part **b**), these studies claim to demonstrate sluggish spatial attention shifting in individuals with dyslexia (that is, the longer interval is required for successful cue utilization or the cue is not utilized successfully even with a long interval). Other studies (parts **c** and **d**) require the cue to be selected from a multiple-element array of targets (ovals in part **c** and false font in part **d**[51,52]) using different temporal intervals between cue and target, and using masking in some experimental conditions. The dependent measure is the accuracy of target detection when valid cues are supplied. The variability of the paradigm, dependent measure and cue-target asynchronies make it difficult to extract a consistent developmental picture of the exact nature of dyslexic impairment. Part **a** is adapted with permission from The relationship between visuo-spatial attention and nonword reading in developmental dyslexia. Facoetti, A. *et al.*, *Cognitive Neuropsychology*, 2006, Taylor & Francis, reprinted by permission of the publisher (Taylor & Francis Ltd, http://www.tandf.co.uk/journals). Part **b** is adapted from Facoetti, A. *et al.* Multisensory spatial attention deficits are predictive of phonological decoding skills in developmental dyslexia. *Journal of Cognitive Neuroscience*, **22**, 1011–1025, 2010 MIT Press Journals. Reprinted by permission of MIT Press Journals, © 2009 Massachusetts Institute of Technology. Part **c** was published in *Current Biology*, **22**, Franceschini, S. *et al.* A causal link between visual spatial attention and reading acquisition, 814–819, © Elsevier (2012). Part **d** was published in *Current Biology*, **23**, Franceschini, S. *et al.* Action video games make dyslexic children read better. 462–466, © Elsevier (2013).

who instead showed a (sluggish) cueing effect at 250 ms[48]. Younger RL-matched children in the study could utilize the faster (100 ms delay) cue. In a longitudinal study, 82 Italian pre-readers were studied[51]. Of these, 14 children were later classified as poor readers and were found to have had reduced pre-reading attentional orienting. However, the poor readers had also had poorer pre-reading phonological awareness compared to the 68 unimpaired readers, and verbal IQ was not controlled between groups, complicating interpretation. Thus, although a number of the research designs described in BOX 1 have been used to explore the role of sluggish orienting of visuospatial attention in dyslexia, the causal claim currently depends on a mixture of effects on accuracy and reaction times, and there is not clear evidence for sluggish attention in every study.

A recent training study complicates the picture further. A group of 20 10-year-old Italian children with dyslexia were given experience with either active or non-active video games[52] over 9 days of training. The active gaming produced significant gains in visuospatial attention and reading speed (although not reading accuracy). However, the active gamers also improved their speed of phonological recoding to sound (in a pseudo-word reading measure). Hence, the study did not establish that the video gaming improved spatial attention and therefore improved word reading speed. The reading speed improvements could equally have been caused by the increased speed of phonological recoding found for the active gamers.

The fact that in Italian studies, only children with dyslexia who have phonological recoding problems have been shown to exhibit a spatial cueing deficit might again point to reduced reading experience as the driver of this attentional deficit. Italian children with dyslexia who can read pseudo-words efficiently do not show spatial cueing problems (they are also unimpaired in the magnocellular frequency-doubling paradigm[27]). This group of children are seldom described in any detail, but their lack of visual attention impairments undermines the causal argument for this theory. These children show impaired real-word and text reading with unimpaired pseudo-word reading, and comprise around half of the dyslexic sample in the Italian studies[47,48]. The attentional deficit in Italian children with dyslexia is also limited to impaired inhibition (suppression) in the right visual field in some studies[46,47]. This again suggests a role for reduced experience of recoding print to sound. The reduced practice in serial

Glossary

Cortical oscillations
The synchronous firing of neurons in networks of various sizes in different areas of the cortex, producing rhythmic patterns of activity. These oscillatory rhythms have endogenous temporal rates, and can phase-reset their activity to synchronize firing with incoming sensory information, thereby contributing to the processing of the input.

Formant transitions
Rapid changes in frequency (concentrations of acoustic energy within a narrow frequency band in the speech signal) as a speaker transitions from a consonant to a vowel, providing important cues to phonetic identity.

Frequency-doubling illusion
An illusion that depends on the spatial and temporal frequency of a flickering sinusoid grating (a pattern of lighter and darker bars). When a grating with a spatial frequency of 0.1–0.4 cycles per degree flickers faster than 15 Hz, the viewer sees a grating with much narrower lines (that is, the physical spatial frequency appears to double).

Logographically
When the meaning of a symbol is directly recognized without requiring recoding to sound. English logographs include £, % and >.

Magnocellular system
One of two major pathways in the visual system. The magnocellular system contains cells with larger cell bodies than the other (parvocellular) system. Magnocells respond optimally to motion and to visual stimuli that reverse contrast (areas of light versus dark) at lower spatial frequencies and at higher temporal frequencies. Responses are transient and the system is colour-blind.

Mental lexicon
The brain's mental dictionary, containing information about the meaning, pronunciation and grammatical status of words in the spoken language.

Onset-rime division
When a spoken syllable is divided at the vowel or syllable nucleus, the consonant phoneme(s) preceding the vowel are the linguistic onset and the vowel and any subsequent consonant phoneme(s) are the linguistic rime, as in 's-ee', 's-eep', 'sl-eep' and 'sl-ept'.

Orthography
The correct writing system of a language, used here to refer to the chosen symbol–sound correspondence system (such as the Western alphabet, Cyrillic alphabet, Chinese characters or Devanagari).

Phonemes
The smallest units of sound that change a word's meaning.

Phonology
The inventory of the sound system of a language, comprising knowledge of the sounds themselves and the specific patterns or regularities by which sounds in words can be organized.

Posner cueing tasks
A neuropsychology paradigm for measuring spatial attention by cueing a target's future location and measuring whether this cue facilitates target detection. A Posner task usually includes a contrast between valid and invalid cues (only valid cues indicate the future correct location) and endogenous versus exogenous cues (endogenous cues are central to the visual field, whereas exogenous cues are outside the focus of attention or in the periphery).

Saccades
Rapid jerk-like movements of the eyeball that redirect the fovea to a new location in the visual field without a head movement or the conscious awareness of the observer.

Specific language impairment
A developmental disorder of language acquisition that delays the mastery of skills in children who have no hearing loss or other developmental delays.

left-to-right focusing of spatial attention that accompanies reduced experience of recoding print to sound should selectively affect the right attentional inhibition system[47]. One important test would be to study an orthography such as Hebrew, in which orthographic processing is from right to left. If reduced practice in phonological recoding underlies spatial cueing deficits in dyslexia, Hebrew readers with dyslexia should show impaired inhibition in the left visual field.

Italian is one of the most consistent alphabetic orthographies, and recoding individual letters to sound is the hallmark of early Italian reading. This linguistic factor could also explain why such strong spatial cueing deficits are found in Italian children with dyslexia[48]. German is also a relatively transparent language, but it is less transparent than Italian. German children with dyslexia showed no spatial attention deficit compared to CA-matched controls and no right visual field effect in a spatial attention task requiring precedence detection[53].

Currently, there are no studies of infants or of children at family risk for dyslexia that show spatial cueing deficits before learning to read. Similarly, there are no studies exploring other likely consequences of a spatial cueing deficit, such as reduced orienting of spatial attention in the natural visual environment. Specification of the temporal parameters governing 'sluggishness' across different paradigms and/or age groups is also needed to give in-principle explanations for the range of cue–target delays used to date. Finally, languages other than Italian also need to be tested.

Currently, the simplest explanation of the data is that reduced experience of phonological recoding, which for Italian children does involve moving spatial attention from letter to letter, underlies the spatial cueing deficit in dyslexia. Such fine-grained sequential shifts of attention are practised far less when reading scripts such as Chinese, or even when reading inconsistent alphabetic scripts such as English. It is possible that more systematic cross-language investigations will reveal parametric effects of orthography, with the visuospatial attention deficit actually being driven by reduced reading experience.

Finally, it should be noted that magnocellular and visual attention theories are now merging, with claims that impaired (magnocellular-dependent) spatiotemporal parsing of the visual text (a process linked to the parietal cortex) is a sufficient cause for dyslexia[54]. However, this 'meta-theory' currently lacks any systematic application of the research designs in BOX 1.

Impaired auditory processing

Because speech is an acoustic signal, auditory dysfunction offers a parsimonious developmental cause of the phonological deficits that characterize dyslexia. However, the speech signal is complex, and our understanding of the neural processing of speech is incomplete. Nevertheless, sensitivity to rhythmic information in speech is present in the womb, neonates can distinguish languages that belong to different rhythm classes[55] and sensitivity to phonetic information is present soon after birth[16]. Furthermore, the cortical oscillatory mechanisms (BOX 2) underpinning speech encoding seem to function at a range of temporal rates in the womb and at birth[56]. The perceptual organization of speech information (assigning acoustic elements to the groupings comprising linguistic units; BOX 2) takes longer, and also benefits from top-down learning[57]. Impairments in the ability to perceptually organize the acoustic structure of the speech stream should have consequences for phonological processing.

Rapid auditory processing deficit. The first auditory theory of sensory dysfunction proposed developmental difficulties in processing auditory information that arrived rapidly and sequentially[58]. Rapid auditory processing (RAP) theory focused on spectral structure (the variations in frequency that occur as a speaker moves from a consonant to a vowel, for example) because the spectral changes related to linguistic units are typically rapid (within 40 ms for formant transitions[57]). Work with children with specific language impairment[59,60] demonstrated that their ability to process rapidly arriving (within a time window of ~40 ms[58]) auditory information was impaired compared with that of CA-matched control children. In a subsequent CA-matched study, RAP problems were demonstrated in 8 out of 20 children with dyslexia that were tested[61], leading to the claim that difficulties in processing rapidly

changing information in speech (such as formant transitions) caused the phonological deficit in developmental dyslexia[58] (FIG. 4A). However, the reliance on CA-matched data meant that this proposal generated considerable debate, with critics failing to find a RAP deficit in dyslexia[62,63], showing that comparable difficulties were not apparent with speech stimuli[64], or showing that slowing down temporal information in speech did not improve the phonological performance of children with dyslexia[65].

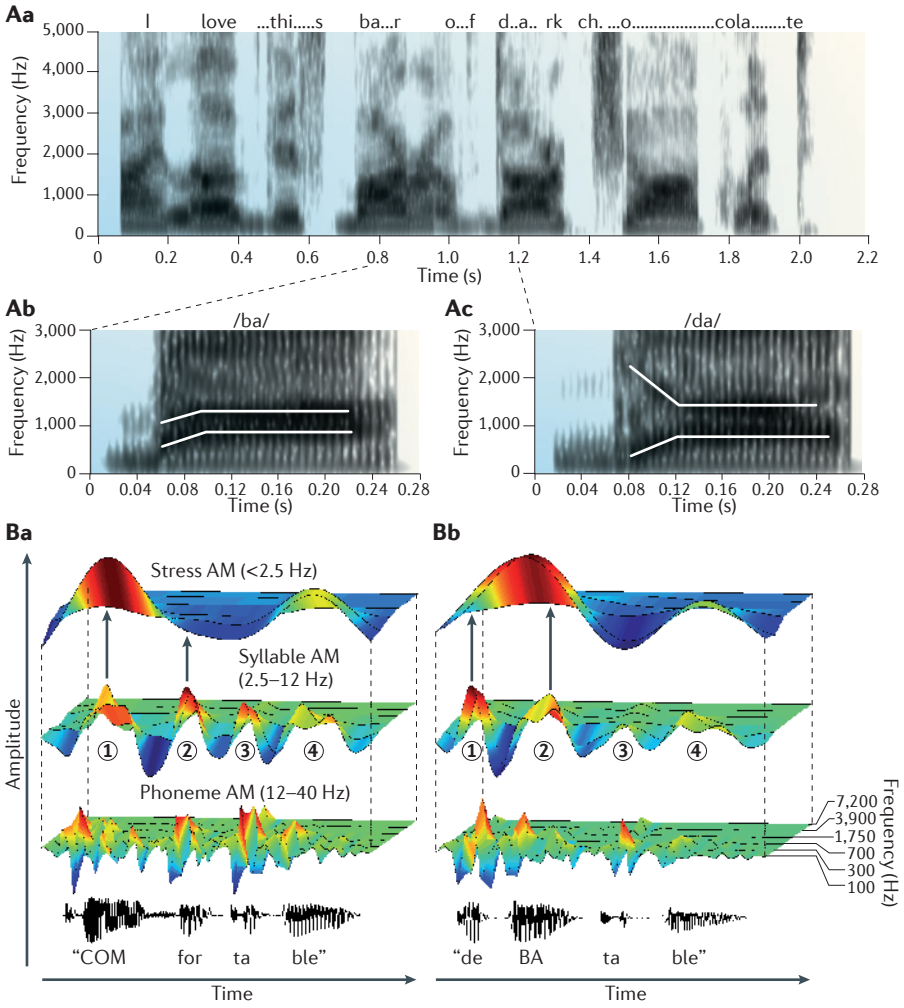

Figure 4 | **Examples of linguistic information assessed by auditory theories.** The figure shows a schematic depiction of the linguistic information that it is claimed is affected by non-speech auditory processing deficits in the detection of rapid frequency changes[58] and the detection of amplitude modulations below 10 Hz[77]. **Aa** | An image of the speech spectrogram shows how the frequency of the speech signal changes over time. **Ab,Ac** | A higher magnification view of the changes in frequency as the syllables 'ba' and 'da' (in the words 'bar' and 'dark') are spoken. The rapid rises and falls in frequency visible in the first 40 ms of the stimuli, which are indicated by white lines, distinguish the different syllables[58]. **Ba,Bb** | Changes in amplitude modulation (denoted 'AM' in the figure) at three different linguistically relevant temporal rates (stressed syllable, syllable and phonetic rate) are shown for the four-syllable words 'comfortable' (primary stress on first syllable) and 'debatable' (primary stress on second syllable) spoken in real time[114]. The raw acoustic signal is shown at the bottom in black. The whole modulation spectrum of the envelope is shown (*z* axis). The figure shows clearly that information in the lower spectral frequencies (foreground of each rate, less than ~700 Hz) carries the most information about syllabic patterning. The stress band shows the highest amplitude modulation for the stressed syllable in each case, whereas the syllable band shows an amplitude peak for each individual syllable (numbered 1–4 in the figures). Phase alignment of the amplitude peaks at the stress and syllable rates contributes to prosodic prominence[79,80,114] (indicated by arrows). Panel **A** from REF. 58, Nature Publishing Group. Panel **B** reprinted from Goswami, U. and Leong, V., Speech rhythm and temporal structure: Converging perspectives? in *Laboratory Phonology*, **4**, 1–257; Berlin, Germany, De Gruyter 2013; pp. 75, fig. 3.

Subsequent RAP studies have continued to use ambiguous CA-matched designs; however, training studies and longitudinal studies are also available. A computer application intended to train RAP has shown apparent within-participant benefits[66,67] (although no control groups of children who play similar acoustic software games that train non-rapid auditory processing have been studied). These software games also explicitly train phonological awareness, hence the gains that have been demonstrated are inherently ambiguous. In a longitudinal study, American pre-readers at familial risk for dyslexia showed differences in brain activity when compared with typically developing CA controls (specifically, hypoactivation in the left frontal cortex) when listening to non-speech stimuli with rapid frequency transitions[68]. Because similar hypoactivation characterizes older children and adults with dyslexia in RAP tasks, the demonstrated atypical activation was argued to be supportive of RAP theory[68].

Important longitudinal work with English-learning infants might be promising in terms of demonstrating causality; nevertheless, results to date implicate a role for RAP deficits in specific language impairment and not dyslexia[69]. Furthermore, longitudinal studies of RAP in preschoolers in other languages show negative results. For example, Dutch preschoolers at familial risk of dyslexia failed to show any RAP difficulties, and performance in RAP tasks did not predict later phonological awareness[70]. The claim that RAP deficits cause dyslexia is also undermined by a recent CA-matched study that found that children with dyslexia were significantly better than CA-matched controls in discriminating rapid rises in frequency that changed a synthesized 'ba' syllable into the syllable 'wa' (REF. 71). The group with dyslexia were able to discriminate between 'ba' and 'wa' syllables distinguished by a 15 ms rise in frequency, whereas CA-matched controls required frequency rises of 30 ms or more. If replicated, such a result would seem to rule out formant transitions as a source of the phonological deficit in dyslexia.

Meanwhile, because phoneme awareness is largely a product of learning an alphabetic script (BOX 2), experience of phonological recoding is vital for learning phonemic structure from alphabetic information. Difficulties in perceiving rapid information in speech should hinder a child's ability to learn phonemic structure, and again the severity of impairments should vary with orthographic consistency. Accordingly, studies of French adults with dyslexia showing atypical neural processing of amplitude modulations in the

γ-range[72] are not evidence that this atypical processing causes dyslexia. The atypical processing could equally be a result of years of reduced phonological recoding experience. Similarly, if Dutch adults with dyslexia show comparable neural processing (similar multi-voxel activity) to age-matched controls in a syllable discrimination task, this does not prove that dyslexia arises from an impairment in accessing intact phonological representations[73]. Dutch is another transparent orthography, so even reduced experience of phonological recoding to sound would have developmental effects on the quality of phonological representations.

Finally, there are currently no studies exploring whether musical cognition is impaired as a result of putative RAP difficulties. Logically, it should be affected. However, developmental studies of categorical perception using synthetic speech stimuli have reported significantly more sensitive performance by children with dyslexia[74,75]. In categorical perception tasks, children with dyslexia show better discrimination within a phonemic category than both CA- and RL-matched controls[75]. These data have been interpreted as showing that allophonic perception, which is normally discarded in infancy[16], is preserved in dyslexia[74]. Although this position is the theoretical opposite of RAP theory, it is important to note that significant dyslexic advantages in allophonic perception would be consistent with demonstrations of atypical neural activation in RAP studies[67,68]. However, the atypical activation would reflect allophonic processing. Currently, therefore, the evidence for the RAP theory of developmental dyslexia does not meet the conditions for establishing causality.

Amplitude modulation (rise-time) deficit. A more recent theory concerning auditory temporal processing focuses on relatively slow information and concerns intensity (amplitude) rather than frequency (pitch). Rise-time theory proposes that there are dyslexic impairments in discriminating amplitude envelope rise times at slower temporal rates, which affect the detection of speech rhythm and prosody[76,77]. The amplitude envelope of speech is its slow-varying energy contour, which contains a range of amplitude modulation patterns at different temporal rates. These rates have different 'rise times' (the time required to reach the modulation peak). Recent modelling of the patterns of amplitude modulation at different frequencies and timescales in child-directed speech suggests that these slower amplitude modulation patterns support the developmental emergence of phonological awareness at the larger grain sizes that are available to preliterate children[78–80] (BOX 2; FIG. 4). The three dominant rates of amplitude modulation in child-directed speech[78] are shown in FIG. 4B.

Rise time is related to the perceptual organization of speech rhythm and syllable structure, and school-age children do not yet show adult-like use of the amplitude modulation structure of speech[57]. However, a series of studies measuring rise-time sensitivity in children with dyslexia across languages[81–85] reported impaired discrimination of rise time compared with CA-matched controls, with children with dyslexia performing similarly to younger RL-matched children. Only Greek children with dyslexia showed no differences compared to either CA- or RL-matched groups, which was an ambiguous result[86]. Significant relationships between phonological awareness and rise-time sensitivity were found in all studies reporting positive results. These early studies all explored rise-time relationships with sub-syllabic phonological awareness (onset-rime and phoneme awareness).

Deficits in novel tasks that theoretically should be affected by impaired rise-time discrimination were subsequently demonstrated for English children with dyslexia. These children showed previously unsuspected prosodic deficits, with younger children with dyslexia (9 year olds) showing significantly poorer prosodic awareness than RL-matched controls (7 year olds)[87,88]. Rhythmic processing of non-speech sounds was also affected, with children with dyslexia showing impaired tapping to a metronome beat at 2 Hz, and individual differences in tapping accuracy that were related to phonological awareness and reading[89]. Most strikingly, children with dyslexia were also significantly poorer than younger RL-matched children at judging rhythm in music[90]. In fact, the musical rhythm task was a stronger predictor of reading than phonological awareness in this study[91]. Furthermore, in a 7-year longitudinal study following around 40 children with dyslexia along with CA- and RL-matched controls from the age of 8 years, a significant dyslexic impairment in rise-time sensitivity compared to RL-matched children emerged at the age of 12 years[90] (by that test point, RL controls were aged 10 years). This makes it unlikely that the rise-time impairment in dyslexia is a sensory product of reduced reading experience. Superior metacognitive skills might have masked this sensory difference at earlier test points.

In a different longitudinal study, rise-time sensitivity in English-speaking pre-readers (4 year olds) was a significant predictor of rhyme awareness at the age of five[92]. The longitudinal study of at-risk Dutch pre-readers mentioned above found that sensitivity to slow frequency modulations predicted later reading ability and phonological awareness[29].

Many of the conditions for establishing causality have therefore been met for rise-time theory, although further longitudinal studies are required. To date, there have been two major longitudinal studies, one Finnish and one Dutch, in which babies at family risk for dyslexia were followed from birth. Neither study included rise-time measures; nevertheless, both identified a range of neonate and infant auditory weaknesses using electroencephalography (EEG) that predicted later phonological awareness and reading ability[93–95]. A small-scale family-risk longitudinal study in English children revealed timing difficulties in syllable production for the at-risk children at the ages of two and three[96].

The results of training studies also support rise-time theory. Training studies with both dyslexic and poor readers have shown that behavioural interventions designed to enhance the perception of rhythm in language (using music, drumming, marching and poetry) improved phonological awareness, reading and spelling[97,98]. Motor entrainment (assessed using a tapping-to-music task) was also measured and improvements in entrainment were significantly related to reading improvements[98]. The role of entrainment to the beat may be important conceptually in explaining neurocognitive links between rise time, rhythm, phonological awareness and reading ability.

These potential neurocognitive links were spelled out explicitly in temporal sampling theory, which was developed to take account of the auditory cortical oscillatory hierarchy shown in BOX 2 (REF. 77). Temporal sampling theory proposed that the ability of children with dyslexia to perceive the patterns of amplitude modulation at slow timescales in speech might be impaired by poor rise-time detection (FIG. 4B). Rise times function as auditory 'edges', resetting ongoing neuronal oscillations so that oscillatory peaks are aligned with amplitude modulation peaks. Atypical oscillatory alignment would thus affect the perceptual organization of amplitude modulations, meaning that stressed syllables, syllables and the onset-rime division would be poorly encoded. Such amplitude modulation difficulties would affect phonological skills across languages. If the higher levels of the oscillatory

hierarchy entrain poorly to amplitude modulation information, this would also affect the downstream identification of smaller units such as phonemes when reading is taught (BOX 2).

Neural tracking of rise times and the amplitude envelope can be measured directly using EEG. Hypothesis-driven EEG studies revealed that children with dyslexia indeed exhibited different event-related potentials to amplitude envelopes with longer (90 ms) rather than shorter (15 ms) rise times compared to CA-matched controls[99]. The same children were significantly impaired in neuronal entrainment to a rhythmic syllable stream presented at a rate of 2 Hz[100]. When hearing or seeing a speaker rhythmically repeating "ba, ba, ba", the children with dyslexia showed phase alignment at less informative temporal points in the incoming signal, which degraded speech representational quality[100]. The difference in preferred delta phase between the groups was 0.16 radians at 2 Hz, which is the equivalent of 12.8 ms, an interval within the phonetically important 40 ms window identified earlier by RAP theory[58]. Therefore, the consistent timing difference could have cascading consequences for the optimal encoding of phonetic information by these children. Individual differences in preferred phase were indeed related to performance in a phoneme deletion task[100]. Importantly, different phase alignment between groups was not found in a visual speech control condition[100]. Nevertheless, RL-matched EEG studies are currently missing.

Also missing is a demonstration that neural encoding of low-frequency envelopes is impaired when children are listening to sentences. Studies of illiterate adults would also be interesting. Illiterate adults can detect rhymes but not phonemes[101], which would suggest that their rise-time sensitivity is unimpaired. Further cross-language studies are also required because rise-time theory would predict prosodic and rhythmic difficulties in dyslexia across languages. If difficulties with syllable stress were found in Italian children with dyslexia who do not show phonological recoding difficulties (as well as in those who do), this would be particularly persuasive evidence for a primary auditory deficit. Difficulties with syllable stress and prosodic awareness would also be predicted for Greek children with dyslexia.

Future directions

This necessarily selective analysis of studies of sensory dysfunction in children with dyslexia suggests that differences in reading experience (phonological recoding) drive the visual sensory processing difficulties that have been identified in individuals with dyslexia. It is too early to dismiss the visual theories, however, because the available studies suffer from under-use of the research designs described in BOX 1. There is also little data on the typical development of vergence control and visual attention, and no data on vergence control and visual attention in illiterate populations. Furthermore, ameliorating visual difficulties by increasing letter spacing or using shorter lines of text seems to have a positive impact on reading by children and adults with dyslexia[45,102].

Based on current data, temporal sampling theory[77] comes closest to meeting the conditions for establishing causality. Rise-time theory can also explain why deficient phonemic processing and atypical rapid-rate acoustic processing may occur in dyslexia. This would be a likely consequence of downstream perturbation of the acoustic modulation hierarchy in speech (BOX 2). Low-frequency amplitude modulation is detected by the fetus. If extraction of primitive phonological structure (the nested hierarchy of prosodic stress, syllables and onset-rime units; BOX 2) is mediated by neuronal oscillatory entrainment at different speech timescales, and if entrainment to slower amplitude modulation rates is impaired in developmental dyslexia, then the mental lexicon would develop a different 'auditory organization' (REF. 103) from birth. Phonological representations would develop differently in dyslexia; for example, impaired speech rhythm detection might be compensated for by extra sensitivity to phonetic cues[77]. Developmentally, this atypical trajectory would preserve spoken language processing while impairing written language processing (for example, too many 'phonemes' would map to one letter)[74,75].

It is also possible that a primary deficit in a third factor, such as pan-sensory oscillatory entrainment, could explain both the auditory and visual sensory difficulties documented here[104–106]. Because amplitude envelope rise time relates to signal intensity, it may be that oscillatory entrainment to luminance or spatial rather than temporal information is impaired in the dyslexic visual system. Systematic studies are required across languages to explore auditory, visual and motor processes. It is important to note also that atypical sensory processing from infancy would lead to developmental compensation by other sensory processes and/or systems, and this too could be tested in hypothesis-driven ways[104]. A useful way forward experimentally would be for all of the key tasks identified in this Opinion article to be included in longitudinal studies, beginning in infancy, in as many languages as possible. Identifying the correct cause (or causes) of dyslexia would benefit the education of millions of children, enabling early environmental enrichment. In the future, accurately targeted enrichment may even allow this academically limiting disorder of learning to be eradicated.

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Competing interests statement
The author declares competing interests: see Web version for details.

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Oscillatory "temporal sampling" and developmental dyslexia: towards an over-arching theoretical framework: http://journal.frontiersin.org/ResearchTopic/1064#overview

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