1 Theories of Dyslexia

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1.1 Introduction

Dyslexia is a disorder of development. Classically, a child has shown apparently typical language acquisition and cognitive development until faced with the task of learning to read. Suddenly the child struggles: ‘In spite of laborious and persistent training, he can only with difficulty spell out words of one syllable’ (Hinshelwood 1896, p. 1378). Why this apparently specific problem with reading and writing? One hundred years later, a child with dyslexia aged 9 years wrote ‘I have blond her, Blue eys and an infeckshos smill. Pealpie tell mum haw gorgus I am and is ent she looky to have me. But under the surface I live in a tumoyl. Words look like swigles and riting storys is a disaster area because of spellings’ (I have blond hair, blue eyes and an infectious smile. People tell Mum how gorgeous I am and isn’t she lucky to have me. But under the surface I live in a turmoil. Words look like squiggles and writing stories is a disaster area because of spellings) (author’s private notes).

Theories of developmental dyslexia attempt to provide a systematic causal framework for understanding this specific learning difficulty. Most theories aim to identify the critical factor/s or ‘core deficits’ underlying the child’s struggle to learn, in order for remediation to be focused and effective. Yet there are a large number of theories of dyslexia, some mutually exclusive, and there is more heat than light. Many theories are over-reliant on data from a single language or a small set of languages, and no theory is universally accepted by researchers in the field. In this chapter, I focus on some of the most dominant theoretical frameworks, seeking points of unification. Importantly, I will adopt a developmental perspective, which means that theories and experiments relying on adult data will not be considered.

A focus on development is absolutely critical to identifying core factor/s for effective remediation. Currently, ‘multiple deficit’ theories of dyslexia are gaining in popularity (Pennington 2006; McGrath et al. 2020). A key developmental question is whether some of the multiple deficits, typically identified in studies of older children with dyslexia, are in fact a downstream developmental consequence of a single atypical factor present from birth that has had multiple systemic effects. Stringent research designs are required to identify such factors, as any deficits found once reading instruction has commenced may be a consequence of the severely reduced reading experience that is inevitable for dyslexic individuals. Both
intervention studies and reading-level match studies can help to identify causal factors (Goswami 2015a), as can the study of typical learners.

A focus on development also requires consideration of sensory and neural data regarding how the typically functioning brain creates a speech processing system. Reading is essentially comprehending speech when it is written down. Accordingly, sensory and neural factors that cause individual differences in speech processing may play a role in the emergence of dyslexia. During the acquisition of spoken language, infants and children learn the sounds and combinations of sounds that are permissible in their language/s. Their brains develop phonological representations of the sound structures of individual words. As reading is learnt, these phonological structures are linked to visual codes. We know from infant research that phonological representations are developed via auditory, visual, and motor learning: phonological representations prior to reading are already multimodal (Kuhl 2004). Research testing theories of dyslexia must thus begin with infant studies. Taking a snapshot of a single sensory, neural, or cognitive parameter at one age point is insufficient, even with reading-level-match designs. Longitudinal studies beginning in infancy are the key to understanding causation.

To complicate the picture further, studies at multiple levels of developmental description in multiple languages are required. Researchers need to combine the assessment of individual differences in neural learning, sensory processing, cognitive processing, and children’s behaviour in the same children, following these children over time using narrow age banding. Studies with pre-readers, including intervention studies, are particularly valuable. Theories that can identify factors for effective intervention prior to learning to read may offer the promise of eliminating dyslexia (Goswami 2020). Unfortunately, few current theories of dyslexia have been tested by studies that include all these important criteria. Nevertheless, these kinds of data will be my focus here.

1.2 Typical Development of Reading

If reading is defined as the cognitive process of understanding a visual code for spoken language, then, logically, individual differences in acquiring reading could be related developmentally to either spoken language processing or visual code processing, or both. Different cultures have invented a range of visual codes for representing spoken language, and skilled readers appear to access meaning directly from these visual codes. Nevertheless, phonological activation (activation of brain areas associated with linguistic sound-structure processing) is mandatory during skilled reading. This suggests that, developmentally, efficiency in learning a visual code cannot be separated from spoken language skills (see Figure 1.1). The visual code is not a neutral visual stimulus. It is a culturally specific code that is taught and learnt using symbol–sound correspondences. This learning typically begins a few years into the development of a spoken language system. The neural spoken language system is then changed forever by this symbol–sound learning. There is developmental remapping of phonology with the acquisition of print (Frith 1998).
For example, if young children are asked to choose pictures whose names begin with the same sound as ‘truck’, child readers will choose items like ‘turkey’ while preliterate children will choose items like ‘chair’ (Read 1986). Read argued that this occurs because the ‘t’ sound in ‘truck’ is affricated and hence is phonologically closer to ‘ch’, a phonetic distinction still heard by the preliterate brain. Learning via print that the symbol used to represent the ‘ch’ sound in these words is ‘t’ changes children’s phonological judgements. Indeed, the young pre-readers studied by Read would misspell ‘truck’ as ‘chrac’ and ‘ashtray’ as ‘aschray’, errors that disappear as children learn conventional English spelling patterns.

As might be expected, brain imaging studies across languages show that symbol learning is linked to sound from the very beginning of acquiring reading (Blau et al. 2010; Froyen et al. 2009; Maurer et al. 2005; Maurer et al. 2011; Yang et al. 2020). The brain areas associated with phonological processing also show extensive developmental changes during the first two years of learning to read (Łuniewska et al. 2019). Further, spoken languages differ in the units of sound represented by the chosen visual coding system. For example, Japanese Kana represent individual syllables, Chinese Kanji represent morphemes, and the alphabet represents phonemes. Further while European languages such as Italian, Greek, and Spanish use alphabetic codes with highly consistent grapheme–phoneme correspondences, languages such as English, Danish, and French use markedly less consistent grapheme–phoneme correspondences (the ‘consistency’ problem; see Ziegler and Goswami 2005). When one letter can make multiple sounds, this slows learning for all children.

The development of ‘phonological awareness’ (the child’s ability to consciously detect and manipulate the component sounds in words) follows a similar developmental sequence across languages. Phonological awareness also predicts reading...
acquisition in all languages studied to date. Phonological awareness has been called *metalinguistic awareness*, as its measurement depends on the child becoming *consciously aware* of knowledge that is already organized perceptually in her mental lexicon. The perceptual organization of speech information by a child (assigning acoustic, motor, or visual elements to the groupings comprising words in a particular language) contributes to individual differences in phonological representations. Awareness of syllables and onset-rimes (division of any syllable at the vowel, as in H-OP or ST-OP) appears to develop before learning to read across languages, whereas direct tuition is typically required to develop phoneme awareness. A child’s access to stressed syllables, syllables, onset-rimes, and phonemes reflects their perceptual organization of the different ‘psycholinguistic grain sizes’ that comprise words (Ziegler and Goswami 2005). Interestingly, phoneme awareness develops at a faster rate for children who are learning to read orthographies with consistent grapheme–phoneme correspondences (Ziegler and Goswami 2005). This cognitive evidence that development itself changes the mechanisms and operation of the perceptual systems that support reading means that it is imperative to use rigorous longitudinal designs to test theories of dyslexia (see Figure 1.2).

The neural/sensory processes that underpin the development of phonological representations during spoken language acquisition are now better understood. Infants are sensitive to the acoustic boundaries that separate phonetic categories in all human languages, as are other mammals, birds, and insects (Kuhl 2004). This perceptual sensitivity is not equivalent to having conscious access to phonemes.

**Figure 1.2** Schematic depiction of key issues to consider when testing the evidence base for different theories of developmental dyslexia.
The infant brain also computes conditional probabilities between phonetic elements, syllables, and other speech sounds, thereby identifying possible ‘words’ (statistical learning; Saffran 2001). Some recent neurally driven work has revealed another important set of acoustic statistics that relate directly to phonological awareness at different psycholinguistic grain sizes. Statistical learning of interrelated changes in signal intensity (amplitude modulation) at different temporal rates in the speech amplitude envelope provides perceptual information relevant to extracting phonological units such as syllables and stressed syllables (Leong and Goswami 2015). Computational modelling of these amplitude modulation changes reveals that the different rates of amplitude modulation in speech are temporally dependent on each other (phase dependency). The amplitude modulations are also arranged in a hierarchy, with the slowest modulations governing the temporal timing of faster modulations. Computational modelling of both infant-directed speech and rhythmic child-directed speech (English nursery rhymes) shows that this amplitude modulation phase hierarchy by itself provides sufficient perceptual information for the brain to extract phonological units of different sizes with more than 90% efficiency (Leong et al. 2014, 2017). Accordingly, the acoustic statistical amplitude modulation structure of both infant-directed speech and rhythmic child-directed speech enables ‘acoustic-emergent phonology’ (Leong and Goswami 2015).

Neurally, we know that neuroelectric oscillations responsive to speech inputs (rhythmic changes in electrical brain potentials in large cell networks) are also hierarchically organized (Gross et al. 2013), and that the temporal rates of oscillation of these brain rhythms match the amplitude modulation rates in speech (Giraud and Poeppel 2012). ‘Rise times’ in amplitude (the rates of change between sound onset and sound peak in a given amplitude modulation) provide sensory landmarks that automatically trigger brain rhythms and speech rhythms into temporal alignment, via phase-resetting ongoing neural activity (Doelling et al. 2014). Accordingly, a nascent phonological system can be extracted from the speech signal via the automatic alignment of neuroelectric oscillations to the amplitude modulation information in speech, beginning in infancy (Goswami 2019a, 2019b). This automatic learning can, in principle, enable perceptual organization of the speech stream into syllable stress patterns, syllables, and onset-rime units. Importantly, for languages with consonant–vowel syllable structures, this automatic alignment for onset-rime units also yields information about single phonemes.

This brief survey of typical development suggests that comprehensive theories of dyslexia need to examine putative neural or sensory causes beginning from birth. I first review theories that attempt to explain the origins of the cognitive difficulties with phonology (‘phonological deficit’) that characterize dyslexia across languages. I then consider theories based on processing visual codes for spoken language, and finally I consider theories that utilize general properties of learning such as statistical learning and neural noise exclusion. In each case, I consider whether the theory is supported by longitudinal data, examine intervention and reading-level-match studies, consider whether the candidate deficit has been tested in other developmental disorders, and consider whether other cognitive difficulties predicted by the
candidate deficit are present in children with dyslexia (see Figure 1.2). I also consider whether the available evidence draws on a range of languages.

1.3 Theories Explaining Individual Differences in Phonological Development

Auditory theories of dyslexia propose that sensory processing differences, present from birth, lead affected children to develop atypical phonological representations of spoken language. These impaired phonological representations then affect the efficient learning of a visual code for spoken language. As noted, visual codes for speech are not arbitrary visual stimuli; they are linked to phonology from the onset of learning. Accordingly, if the visual symbols being taught are difficult to match systematically to the perceptual organization of speech-based information in the child’s mental lexicon, then learning to read will be slow and inefficient whichever symbol system is being acquired. I review two key theories proposing a sensory basis for the phonological deficit: one based on rapidly arriving pitch cues in speech and one based on slowly varying amplitude modulation-related information (Tallal 1980; Goswami 2011). As auditory learning begins in the womb (the amniotic fluid transmits the low-frequency amplitude modulation information in maternal speech), studies of infants and pre-readers are particularly important tests of causation.

1.3.1 Rapid Auditory Processing Theory

Rapid auditory processing (RAP) theory was first proposed by Tallal and Piercy (1973) to explain developmental language disorder and was later extended to dyslexia. The variations in frequency (pitch) that occur as a speaker moves from producing one phoneme to another are typically rapid (within 40 ms for formant transitions). The core theoretical proposal of RAP was that the ability to process this rapidly arriving and sequential acoustic information was impaired in developmental language disorder. Using tones of different frequencies, Tallal and Piercy showed that twelve children with developmental language disorder (aged 6.5–9 years) had difficulties in processing rapidly arriving information compared to chronological age-matched controls. In a subsequent study with dyslexic children aged 8–12 years, RAP problems were demonstrated in eight of twenty children compared to chronological age-matched controls (Tallal 1980). Accordingly, it was argued that difficulties in processing rapidly changing information in speech (such as the formant transitions that help differentiate phonemes) caused phoneme awareness deficits and thereby developmental dyslexia (Tallal 2004).

Given that conscious awareness of phonemes largely emerges as a consequence of learning to read, these acoustic difficulties should affect phoneme awareness in dyslexia across languages. However, this is not the case. For example, dyslexic 10-year-olds learning consistent alphabetic writing systems such as German become as accurate as chronological age-matched controls in recognizing and manipulating phonemes (Landerl and Wimmer 2000). RAP deficits are also found in a minority of Chinese
dyslexic children (e.g., Chung et al. 2008), even though the Chinese writing system is not phoneme based. This may suggest that phonemic awareness is not the causal link between RAP deficits and dyslexia. Recall also that the neural and cognitive representation of phonology changes with reading experience. It is thus logically possible that sensory differences in RAP found with older children are a consequence of poorer reading experience, across languages. Reading-level-match studies are currently not available, but when chronological age-match studies comprise pre-readers or infants who are at family risk for dyslexia, then RAP difficulties are not reported. For example, a longitudinal study of Dutch preschoolers at familial risk for dyslexia failed to show any RAP difficulties, and performance in RAP tasks did not predict later phonological awareness (Boets et al. 2011). Currently missing from the literature are longitudinal studies linking RAP in infancy to phonological measures in different languages and studies of RAP in developmental disorders other than developmental language disorder. Other cognitive impairments that might be predicted on the basis of a RAP deficit, such as perceiving rapid pitch changes in music, have only been studied in dyslexic adults (with null results; see Zuk et al. 2017).

Regarding neural data, US-American pre-readers at family risk for dyslexia showed differences compared to typically developing chronological age-matched controls in brain activity when listening to non-speech stimuli containing either rapid or slow frequency transitions (Raschle et al. 2014). This is potentially important, yet RAP training studies show null results regarding remediation of reading (see Strong et al. 2011 for a meta-analysis). Neural studies measuring the auditory steady-state response in dyslexic children to fast amplitude-modulated noise (rates of 20 Hz+) are also thought relevant to RAP and phoneme awareness (Poelmans et al. 2011; Vanvooren et al. 2014; De Vos et al. 2017, 2020). The auditory steady-state response measures the amplitude following response of neural oscillations, and it is claimed that the auditory steady-state response to faster amplitude modulation rates illuminates the integrity of phonemic processing (Vanvooren et al. 2014). The only longitudinal data set (in Dutch) suggests that neural synchronization to ‘phoneme rate’ (20 Hz) amplitude-modulated noise increases markedly with the onset of learning to read (De Vos et al. 2017). This suggests that differences in older children for rapid rates of amplitude modulation may be driven by reading experience. Differences for dyslexic teenagers and adults regarding ‘phoneme rate’ auditory steady-state responses are mixed (see Lizarazu et al. 2021 for review). Accordingly, to date, there is no strong evidence that RAP represents a core causal factor in dyslexia.

1.3.2 Temporal Sampling Theory

Temporal sampling theory proposes atypical sensory/neural processing of the amplitude envelope of the speech signal by children with dyslexia, particularly at amplitude modulation rates <10 Hz that carry syllabic information (Goswami 2011, 2015, 2018, 2020). The amplitude envelope is the slow-varying energy contour of speech that determines the perception of speech rhythm. The amplitude envelope contains a range of amplitude modulation patterns hierarchically nested at
different temporal rates. As noted, the phase relations between these different amplitude modulation rates provide systematic statistical cues to phonological units such as stressed versus unstressed syllables, syllables, and onset-rimes (acoustic-emergent phonology; Leong and Goswami 2015). Notably, these acoustic cues operate at time windows much longer than RAP’s 40 ms (50 Hz) window. Yet these slow intensity changes are also relevant to extracting phonemic information. For example, speech-modelling studies show that the critical information in phoneme deletion tasks is related to changes in the phase relations of delta-rate and theta-rate amplitude modulations (Flanagan and Goswami 2018), and brain imaging studies show that these slow amplitude modulations carry phonetic information (Di Liberto et al. 2018). Extracting acoustic-emergent phonology via statistical learning requires successful phase alignment of amplitude modulation information in speech with brain electrical rhythms (oscillations). In turn, this requires efficient discrimination of amplitude rise times. Temporal sampling theory was originally based on studies showing that children with dyslexia in a range of languages were impaired in rise-time discrimination compared to chronological age-matched controls (English, Spanish, French, Finnish, Chinese, Hungarian, and Dutch; Goswami 2015a). Rise-time discrimination was also related to phonological awareness at different grain sizes in these different languages (Goswami et al. 2011).

Longitudinal studies of rise-time discrimination by infants at family risk for dyslexia are now emerging. For example, an Australian study has found impaired rise-time discrimination at 10 months in at-risk infants, who by their second year showed impaired phonological learning in a novel-word-learning task, along with delayed achievement of phonological constancy (Kalashnikova et al. 2018, 2019, 2020). Significant longitudinal associations with language were present: for example, rise-time discrimination in infancy predicted vocabulary size at age 3 years (Kalashnikova et al. 2019). By age 4, non-speech rhythm deficits were present in the at-risk group, but visual–verbal paired-associate learning deficits were not (Kalashnikova et al. 2020). The rhythm deficits were identified using a musical rhythm task that has also been used with Italian dyslexics (Flaugnacco et al. 2015). These infant data are consistent with longitudinal studies of at-risk infants in other languages, which have identified a range of neonate and infant neural auditory weaknesses based on syllabic stimuli that predict later phonological awareness and reading ability (Guttorm et al. 2010; Leppänen et al. 2010; van Zuijen et al. 2013).

Longitudinal studies of preschool children report rise-time deficits in those at family risk for dyslexia (Law et al. 2017). Furthermore, rise time measured in preschoolers aged 3–5 years predicts phonological awareness, letter knowledge, and reading at ages 6 and 7 years (Corriveau et al. 2007; Vanvooren et al. 2014). In these studies, the rise-time measure used accounted for 10–16% of unique variance in later phonological awareness, and up to 10% of the variance in later letter knowledge. Longitudinal studies with school-aged children reveal significant impairments compared to younger reading-level-matched children for rise-time discrimination, musical rhythm perception, and linguistic rhythm perception (Goswami, Huss et al. 2013; Goswami, Mead et al. 2013). The musical rhythm measure can account for up to 42% of unique variance in reading skills (Huss et al. 2013).
However, while rise-time discrimination was a significant predictor of phonological development from 8 to 11 years in Goswami et al.’s longitudinal study, phonological development was not a predictor of rise-time development (Goswami et al. 2021), suggesting that the direction of causality is from auditory processing to language skills (Kalashnikova et al. 2019). Rise-time discrimination is also impaired in developmental language disorder, another developmental disorder wherein both speech processing and rhythm perception are affected (Corriveau et al. 2007; Cumming et al. 2015). Comparisons with other developmental disorders where reading is typically unimpaired, such as autism, have yet to be carried out.

Neurally, the accuracy of speech–brain oscillatory alignment in the delta band (~2 Hz, relevant to prosodic organization) is significantly poorer for children with dyslexia compared to both reading-level- and chronological age-matched controls, even when speech recognition accuracy is equated across groups (Power et al. 2016). When rhythmic processing of syllables is equated between dyslexic and chronological age-matched control children (via an acoustic thresholding manipulation), the dyslexic brain shows a phase difference in delta-band speech–brain alignment compared to controls (Power et al. 2013). Accordingly, the brains of children with dyslexia appear to be encoding a significantly less accurate representation of low-frequency envelope information in speech. This finding has been replicated in Spanish (Molinaro et al. 2016). These neural data are important as they provide a direct measure of the quality of children’s phonological representations when linguistic behaviour is equated, thereby isolating potential causal loci of impairment.

Intervention studies based on improving rhythmic cognition (typically via music-, motor-, or oral-poetry-based rhythmic tasks) can improve phonological awareness and reading for both typically developing and dyslexic children (e.g., Bhide et al. 2013; Flaugnacco et al. 2015). Furthermore, rhythmic synchronization abilities, such as tapping, clapping, or drumming in time with a beat, predict phonology and reading development in a range of languages (e.g., Ríos-López et al. 2019; Woodruff Carr et al. 2014). These relations for typically developing children would be expected by temporal sampling theory. New insights into the mechanistic role of neuronal oscillations for encoding the speech signal, and of amplitude modulation rise times as automatic triggers for speech–brain oscillatory alignment, thus support a specific developmental pathway from auditory sensory processing to phonology and reading that is impaired in dyslexia (Goswami 2020). Accordingly, temporal sampling theory is supported by longitudinal, neural, infant, and preschool data in many languages (Figure 1.2). The research base suggests that children at risk for dyslexia are encoding poorer-quality representations of the speech signal from birth, in part via impaired automatic (statistical) learning of the amplitude modulation phase hierarchy that facilitates ‘acoustic-emergent’ phonology (Leong and Goswami 2015). Accordingly, the development of phonological awareness is impaired, which impairs reading acquisition. temporal sampling theory is thus accruing converging developmental data at multiple levels of description (neural, sensory, cognitive, and behavioural). Currently missing from the literature are longitudinal multiple-level data testing temporal sampling theory from infancy in languages other than English, and reading-level-match studies across languages.
1.4 Theories Relating to Atypical Visual Behaviour During Reading

Children with dyslexia do not scan print as efficiently as chronological age-matched controls, showing more fixations and less rhythmic saccades. Sometimes, they complain that print seems to move around on the page. Observations such as these have led to a range of visual theories of dyslexia, of which I consider three prominent examples here.

1.4.1 Impaired Visual Attention Span Theory

During reading, in most languages children are scanning small symbols arranged in rows from left to right. The impaired visual attention span theory proposes that such multi-letter parallel processing is atypical in dyslexia, because the number of individual elements available for simultaneous processing in the ‘attentional window’ is reduced, limiting reading development (Valdois et al. 2004). A visual attention span deficit has been found to dissociate from a phonological deficit in some French and Portuguese children, which is interpreted to show that impaired visual attention span is an independent cause of dyslexia (Bosse et al. 2007; Germanò et al. 2014). However, the classic visual attention span task used in these studies presents an array of five letters very briefly, and records reaction time for naming either all the letters in the array (full report) or single letters at different cued positions (partial report). The reliance on letters as visual attention span stimuli unfortunately introduces an immediate confound, as letters are a culturally specific code that is taught and learnt using symbol–sound correspondences, and children with dyslexia process letters less efficiently. This may explain why visual attention span tasks account for 24–36% of variance in the reading scores of children with dyslexia (Bosse et al. 2007). Studies using unfamiliar symbols or coloured dots as stimuli do not report visual attention span deficits in dyslexia (Ziegler et al. 2010; Valdois et al. 2012). Nevertheless, visual attention span deficits in dyslexia using the classic task have also been found in Chinese, a non-alphabetic language (Chen et al. 2019). However, Chen et al. used visual attention span tasks based on Chinese characters, Chinese radicals, and digits, and only found a visual attention span deficit on a reading-level match for the Chinese characters, again reminding us that any visual code for representing speech is not a neutral visual stimulus. Zhao et al. (2018) studied Chinese children with dyslexia from grades 2 to 6, using a visual one-back task rather than the classic visual attention span task. Here they only found a visual attention span deficit for older dyslexics (5th and 6th grade). Typically developing Chinese children who were also studied showed no relation between visual attention span and reading development. These effects complicate theoretical interpretation. If visual attention span is a primary causal factor in dyslexia, then it should be present irrespective of reading experience and it should also relate to reading in typically developing children across languages.

Visual attention span has been shown to be related to individual differences in typical reading development in French (Bosse and Valdois 2009). Again, visual attention span contributed significant unique variance to progress in reading,
particularly in grade 1 (15%), but again the study relied on letter-based tasks. No studies testing pre-readers are available to determine cause and effect. Further, a reduced attentional window could be expected to cause developmental problems in other areas of cognition that require simultaneous processing of distinct visual elements, for example categorization, but this has not been studied to my knowledge. The sensory and neural factors that may cause impaired visual attention span are unknown, but seem unlikely to be specific to reading. Currently absent from the literature are studies measuring visual attention span in infants or pre-readers that show a reduced span in at-risk children. Ideally, such studies should use non-letter-based visual attention span tasks. Further, visual attention span has not been measured in other developmental disorders to demonstrate specificity to dyslexia.

An interesting visual attention span training study was recently conducted in French (Zoubrinetzky et al. 2019). Children with dyslexia aged 10 years received an adaptive training programme based on visual search, visual discrimination, and visual categorization tasks, developed specifically to train visual attention span (MAEVA: see Valdois et al. 2014, Zoubrinetzky et al. 2019). The children also received a targeted phoneme discrimination training programme (RapDys: see Collet et al. 2012), based on the observation that school-aged children with dyslexia continue to perceive allophones (other possible spoken sounds that could be linked to a letter, but that are typically grouped together as one letter when teaching traditional grapheme–phoneme correspondences: see Serniclaes et al. 2004). The RapDys training was expected to improve phonemic awareness only and MAEVA was expected to improve visual attention span only. A cross-over design was used, in which children first experienced six weeks of training with RapDys or with MAEVA, or vice versa, and then experienced the second intervention (hence acting as their own controls). Unexpectedly, both training regimes improved both visual attention span and phonemic awareness, although minor differences could be discerned (e.g., only RapDys improved non-word reading). The finding that training phonemic discrimination improves the visual attention span is difficult to align with the theoretical claim of visual attention span as a causal deficit that is independent of phonological impairments.

A recent visual attention span training study has also been reported for Chinese (Zhao et al. 2019). The study used non-symbolic materials, and compared the effects of visual attention span training for dyslexic children aged 10 years who did show visual attention span deficits in contrast to those who did not. Training improved character reading speed for both groups, and sentence reading speed for both groups when compared to chronological age-matched controls. The lack of specific training effects for the visual attention span deficit group only is suggestive of training affecting a third unknown factor that is causing the changes in reading speed (tertium quid; see Figure 1.2). Accordingly, to date it seems most likely that a reduced visual attention span is a consequence rather than a cause of dyslexia (Goswami 2015b).

1.4.2 Sluggish Attentional Shifting Theory

A second visual attention theory (sluggish attentional shifting theory) argues that the orientation of spatial attention is ‘sluggish’ in individuals with dyslexia (Facoetti
et al. 2010). The dyslexic brain is thought to be unable to move visuospatial attention smoothly from letter to letter when recoding print to sound. This impaired ability to orient spatial attention (while simultaneously suppressing flanking letters) then causes dyslexia. As reading experience itself trains spatial orienting, it is important to establish that reduced reading experience per se is not causing the sluggish attentional shifting deficit. This is particularly important as most sluggish attentional shifting data comes from Italian, a consistent alphabetic orthography where letter-by-letter recoding to sound is the typical reading strategy. Further, only Italian dyslexic children with phonological recoding deficits (poor non-word reading) show a spatial cueing deficit in these studies – a deficit which can account for as much as 32% of unique variance in non-word reading (Faccoetti et al. 2010). However, Italian dyslexic children who can read non-words efficiently do not show sluggish attentional shifting. Data such as these suggest that sluggish attentional shifting may arise from reduced practice in recoding print to sound.

Data from pre-reading children and at-risk infants are therefore critical. A longitudinal study of eighty-two Italian pre-readers found that fourteen of the children later classified as poor readers had both reduced pre-reading attentional orienting and poorer pre-reading phonological awareness (Gori et al. 2014). No sluggish attentional shifting studies of infants at family risk for dyslexia are yet available to my knowledge. This is a critical test as impaired attentional shifting should be present from infancy. Sluggish attentional shifting should also affect many other areas of cognition (e.g., visuospatial working memory), though this has yet to be explored. Indeed, studies of children with developmental dyscalculia are converging on visuospatial working memory deficits as a primary impairment (see Chapter 2 Menon and Chang 2022, and Chapter 19, Galuschka and Schulte-Körne 2022), yet a diagnosis of dyscalculia typically depends on reading development being unaffected (Szűcs and Goswami 2013). Utilizing sluggish attentional shifting tasks with children with dyscalculia could thus be important for demonstrating causal specificity.

Regarding intervention data, training visuospatial attention does seem to benefit reading in school-aged children with dyslexia. A series of intervention studies involving action video gaming have documented significant gains in visual–spatial attention, phonological skills, and reading (speed, not accuracy), in both Italian and English dyslexic children, although not in Polish children (Franceschini et al. 2015; Franceschini et al. 2017; Łuniewska et al. 2018). This is interesting and partly supports SAS theory. However, it is intriguing that phonology improves in these studies, despite the lack of any phonological intervention. Reading speed should improve if sluggish attentional shifting is speeded up by action video gaming, but a causal pathway is required to explain why phonology improves. For example, a recent action video gaming study showed gains in non-word repetition, a classic phonological task (Franceschini and Bertoni 2019). Such changes again suggest that training is affecting a third, unknown factor that is causing these changes (tertium quid). Phonological interventions do not (to my knowledge) improve visual–spatial attention; they specifically improve phonological skills and phonological decoding. SAS theory does not explain why remediating a primary deficit in shifting spatial attention should affect phonological development.
Mechanistic neural data could possibly help with this conundrum, but such data are still sparse for SAS theory. Functionally, atypical parietal activation is often reported, yet the parietal cortex is also a locus of atypical function in developmental dyscalculia, a disorder diagnosed on the basis of impairments in mathematics accompanied by intact reading. A training study in German using pre- and post-intervention brain imaging compared the efficacy of three interventions for 9-year-olds with dyslexia: a phonological intervention, an intervention based on orienting visual attention to word fragments, and an intervention training sight vocabulary (Heim et al. 2015). The children received fMRI scanning during single-word reading before and after the interventions. Differences in post-training neural activation were found for both the phonological and attention groups, who both improved in reading. The attention group showed stronger activation in the left Heschl’s gyrus following intervention—a surprising result. SAS theory would not expect visual attention training to affect activation in the primary auditory cortex. This neural finding appears consistent with the global improvements in reading and phonology shown in the action video gaming interventions, again suggesting that there may be a yet-to-be identified third factor that is causing the documented changes in spatial attention, reading speed, and phonology. Intervention designs coupled with longitudinal studies beginning in infancy offer one means of improving our understanding of potential causal relationships.

1.4.3 Magnocellular Theory

Dysfunction of the visual magnocellular system has been suggested as a neural cause of dyslexia for decades, and is still a popular theory (Cornelissen et al. 1995; Stein and Walsh 1997; Vidyasagar and Pammer 2010; Stein 2019). The magnocellular visual system plays a key role in eye movements, motion processing, and vergence control (when both eyes converge on the same location). As stable visual fixation is required for processing symbols such as letters, atypical functioning of the magnocellular system could impair reading, for example causing letters to appear to jump around. Numerous studies of visual motion processing find that older children with dyslexia show poorer performance; however, most of these studies rely on chronological age-match designs and so causation is ambiguous (Stein 2019 for recent review). Two reading-level-match studies are available for 11-year-old Italian children with dyslexia: one reporting a deficit in perceiving coherent dot motion (Gori et al. 2015), and one reporting a deficit in the frequency-doubling illusion, a magnocellular-reliant visual illusion based on vertical dark lines (Gori et al. 2014). Gori et al. (2015) also provided longitudinal data, testing seventy-two pre-readers aged 5 years with the same coherent dot motion task. Two years later, the twelve children in the sample who were poor readers were found to have displayed significantly poorer coherent dot motion sensitivity as pre-readers (Gori et al. 2015). Pre-reading coherent dot motion showed a significant predictive relationship with later Italian text reading for all the children, accounting for 10% of unique variance; a similar predictive relationship has been reported in a Dutch study (Boets et al. 2011). Regarding English, a study of pre-readers at family risk for dyslexia used the frequency-doubling illusion as well as coherent dot
motion to assess magnocellular function at age 5 years (Kevan and Pammer 2008). The at-risk preschoolers showed poorer performance in both tasks, and preschool thresholds for frequency doubling but not coherent dot motion in the whole sample of fifty-eight children predicted reading accuracy and non-word reading a year later (Kevan and Pammer 2009), accounting for between 5% and 12% of unique variance. These data are supportive of pre-reading differences in magnocellular function, some of which then affect later reading.

Intriguingly, chronological age-matched action video gaming intervention studies with Italian dyslexic children improve coherent dot motion performance (Gori et al. 2015). As found for SAS theory, phonological processing and reading speed also improve in these coherent dot motion/action video gaming studies. A recent study with 7-year-old English dyslexic children trained motion processing directly (using moving stripe patterns), and reported improvements in motion direction detection, reading speed, and phonological awareness, as well as in visual and auditory attention (Lawton 2016). In all these training studies, improved magnocellular function affected phonology, an outcome that cannot be explained by the foundations of magnocellular theory (i.e., by improved vergence control, guidance of saccades, or visual fixation). Again, such data suggest that there may be a yet-to-be-identified third factor that is causing these across-the-board improvements.

To my knowledge, only one training study reporting magnocellular outcomes has included a reading-level match. Olulade et al. (2013) reported significant changes in dyslexic visual motion processing, with the expected enhancement of neural activation in visual cortex (V5), but following a phonological intervention. When compared to reading-level controls, the children with dyslexia showed similar levels of V5 activation. These findings suggest that visual motion processing is related to reading, but that the causal relation may be from reading to vision and not vice versa. Atypical visual motion processing in dyslexia in chronological age-match studies may be a consequence of reduced reading experience. A combination of intervention and longitudinal studies, beginning in infancy, are required to clarify the causal connections between phonology, reading, and magnocellular function in children. It is also worth noting that immaturity of the dorsal system (encompassing the subcortical magnocellular system) appears to characterize a number of developmental disorders, including autism, Williams syndrome, and dyscalculia (Atkinson et al. 1997). Children with autism can be hyperlexic, hence much better than expected for their age at recoding print to sound, and children with dyscalculia have intact reading. Accordingly, dorsal-stream deficits are not specific to dyslexia, and children with atypical dorsal-stream function can still develop good reading skills. Clearly, there is a relationship between visual motion processing and reading, but whether it is causal regarding dyslexia remains to be assured.

1.5 Global Theories Based on General Factors

A range of theories based on general neural factors have been proposed to explain developmental dyslexia. The key weakness of all such theories is that they
have difficulty in explaining why impairments in the proposed general neural factors only affect children’s learning regarding reading, and do not affect learning in other cognitive domains such as categorization, conceptual development, number skills, and long-term memory. For completeness, I briefly consider three such theories. It is notable that none of these theories has yet been tested with infant or longitudinal studies, nor in other developmental disorders, nor via intervention studies.

1.5.1 Cerebellar/Automatization Theory

Atypical activation in the cerebellum is often reported in neuroimaging studies of dyslexia. A general theory based on the idea that deficits in the cerebellar network present prior to learning to read could cause dyslexia was proposed by Nicolson and Fawcett (Nicolson and Fawcett 1990; Nicolson and Fawcett 2018; Fawcett et al. 1996; Nicolson et al. 2001). The theory has undergone a number of updates, but the core idea is that cerebellar deficits cause developmental difficulties in automatizing learnt skills, affecting any form of procedural learning (habit formation), but in particular affecting phonological processing (via articulation) and reading (via automatizing grapheme–phoneme correspondences). Most recently, Nicolson and Fawcett (2018) have linked the cerebellar theory to neural noise theories (reviewed below). Their new cerebellar theory, ‘delayed neural commitment’, proposes increased noise in the neural circuits associated with hearing, speech, and possibly other processes, and argues that the development of all ‘automatic’ skills and all forms of implicit learning would be impaired if processing noise were experienced during learning. In particular, it is proposed that this neural noise would impede the development of reading skills via the cerebellum. The neural networks that develop to support articulatory and phonological processes are argued to depend on error-based learning processes that are ‘scaffolded only by the cerebellum’ (p. 112, my italics).

Nicolson and Fawcett (2018) are to be commended for trying to develop a theoretical framework that can encompass a range of both sensory and cognitive theories. However, their reliance on neural noise means that the theory is too general and too under-specified to take the dyslexia field further. Many of the assumptions made in their framework about both cortical neural networks and the operation of noise in cortical networks appear outdated. For example, Nicolson and Fawcett assume that neural commitment is fixed early and cannot be undone by later learning, whereas in vivo animal studies show that even the cerebellar connections underpinning habits are plastic and can be modulated (Vaaga et al. 2020). Indeed, the neural coding of a range of learnt skills appears to ‘drift’ over time, with no apparent detriment to performance (e.g., for animal navigation; Rule et al. 2020). Similarly, all biological mechanisms are noisy, and in the neural engineering field modelling of noisy synaptic mechanisms suggests that there is an optimal network size for any given task (Raman et al. 2019). For networks below the optimal size, it is actually advantageous for learning to add noise (i.e., to add apparently redundant neurons and connections: ‘hyperconnectivity’). Mechanistically, hyperconnectivity reduces the impact of imperfect learning rules. Given the complexity of the brain and of neural
learning mechanisms, it is probably too early to propose delayed neural commitment as a theoretical explanation for a specific learning difficulty such as developmental dyslexia.

1.5.2 Neural Noise Theory

All sensory systems show impaired functioning in the face of noisy input. For example, trying to understand speech presented in background noise is more difficult than trying to understand speech in a quiet environment. Varieties of noise exclusion deficit theories in dyslexia have been around for more than a decade (Sperling et al. 2005; Hancock et al. 2017). The most recent theoretical instantiation is based on evidence that some genes implicated in dyslexia lead to neural hyper-excitability and increased neural noise in guinea pigs, rats, and other mammals (Hancock et al. 2017). Accordingly, Hancock and colleagues propose that excessive neural noise in cortical areas related to reading may cause dyslexia by disrupting phonological awareness and audiovisual integration. For example, they argue that such hyper-excitability could affect auditory temporal sampling. Although this idea could provide an alternative physiological basis for the temporal sampling (TS) theory, there are currently no data explaining why excessive neural noise should only affect certain areas of the cortex, and indeed the authors acknowledge the substantial evidence gap between animal models and genetic effects in the human brain. Nevertheless, this theoretical effort to unpack neural mechanisms related to impaired temporal sampling, for example via neurochemical messengers such as glutamate and GABA, is highly innovative (Pugh et al. 2014). Hancock et al. (2017) note that their model would be ‘directly falsifiable by showing that individuals with dyslexia do not, in general, have noisy, hyper-excitable cortex’ (p. 444). The difficulty here is that a generally noisy cortex would also be expected to hamper cognitive processes other than reading. A noisy cortex should affect all areas of cognitive development, yet dyslexia is by definition a specific learning difficulty (although co-occurrence with language and maths difficulties is common; see Chapter 4, Banfi, Landerl, and Moll 2022). On the other hand, as it is already known that the neural system for reading is atypical in dyslexia, the animal work showing that when networks are below the optimal size it is advantageous for learning to add noise could be relevant to neural noise theory (Raman et al. 2019). The difficulty is devising a stringent way to test this idea in humans. Further, it is worth noting that there are neural noise hypotheses regarding a number of other developmental disorders, for example autism and schizophrenia (Rubenstein and Merzenich 2003).

1.5.3 Implicit Learning Theory

A theoretical variant of the procedural learning deficit postulated by the cerebellar theory is that dyslexia is caused by a domain-general deficit in implicit learning. Implicit learning is the ability to automatically extract regularities in environmental input in order to predict future events. The core idea regarding reading is that an impaired ability to learn statistical-sequential patterns would affect learning of
grapheme–phoneme correspondences (Arciuli and Simpson 2012). Statistical learning deficits in dyslexia have most typically been explored using either serial reaction time tasks or artificial grammar learning tasks. Serial reaction time tasks are usually motor learning tasks. For example, children may learn to press a button in response to a particular stimulus while experiencing a sequence of stimuli with underlying statistical structure. Children who can learn the implicit sequence get faster at pressing the button. Artificial grammar learning tasks involve strings of letters or other stimuli that reflect statistical regularities, ‘rules’ that initially are not imparted to the participant. Following a learning phase, new strings are shown, and participants are asked whether they follow the rule. Correct classification shows implicit learning.

The data across languages for both artificial grammar learning tasks and serial reaction time tasks are very mixed, with no clear evidence for a statistical learning deficit in dyslexia (see Schmalz et al. 2017 for review). As with other general factor theories, a key weakness is that the theory fails to explain why domain-general impairments in implicit learning only affect children’s reading and do not affect other cognitive domains reliant on implicit learning, such as categorization and conceptual development. Indeed, neuroscience studies make it clear that implicit learning is not a domain-general mechanism. Neural systems do learn the patterns or regularities in environmental input via statistical learning, but the critical statistical patterns in different domains of sensory input are poorly understood and may not be those assumed by theorists (Goswami 2020).

### 1.6 Towards a Unifying Theory of Dyslexia?

While no single theory reviewed herein is broadly accepted by the field, the current analysis suggests that there are multisensory differences between children with and without dyslexia, across languages, with relatively strong evidence for atypical sensory processing of acoustic aspects of the speech signal and some evidence for atypical processing of visual motion. More of the research strategies shown in Figure 1.2 have been applied to testing acoustic processing theories than visual motion processing theories, however, as the latter theories still lack infant and pre-reader data. Nevertheless, both acoustic differences in rise-time processing and visual differences in peripheral processing are still present in highly remediated adult dyslexics, suggestive of enduring sensory differences (Pasquini et al. 2007; Schneps et al. 2012). These sensory differences are consistent with data from longitudinal neuroimaging studies of children at risk for dyslexia in Norwegian and German. The Norwegian study reported structural brain differences compared to controls in both primary auditory (Heschl’s gyrus) and primary visual cortices (V2) prior to the onset of literacy tuition (age 6 years). Structural differences in primary auditory cortex were the only differences found consistently at later measurement points, including after dyslexia had been diagnosed at age 12 (Clark et al. 2014). A German longitudinal neuroimaging study reached similar conclusions (Kuhl et al. 2020). The left primary auditory cortex showed structural differences in at-risk children who later
turned out to have dyslexia at all measurement points in the study, including prior to schooling. Functional connectivity between auditory cortex and higher-order speech processing areas also showed differences prior to literacy learning. Combining these structural and connectivity differences predicted later dyslexia with very high accuracy, and more effectively than phonological measures.

Nevertheless, the identification of preliterate differences in primary visual cortex in the Norwegian longitudinal study is thought provoking. In a study exploring the neural circuits underpinning rhythmic pulse pattern recognition in field crickets (insects that rely on transmitting and receiving acoustic rhythmic signals, the signals that are a focus of the temporal sampling theory), the authors noted that the overall network design of the auditory feature detector circuit for pulse patterns was very similar to that of the elementary motion detector circuit found in the visual pathway of these insects (Schöneich et al. 2015). Schoeneich et al. argued that the similarity pointed to a fundamental circuitry layout underlying the temporal processing of sequential events that was shared among different sensory modalities across different nervous systems, also citing studies of flies, crickets, and humans. This cross-species circuitry could suggest that a neural mechanism related to temporal sampling in both auditory and visual domains may be impaired in children with dyslexia.

Recently, a visual analogue to the auditory temporal sampling theory has been proposed by Archer et al. (2020), who suggest that atypical theta sampling in the visual domain could underpin magnocellular differences between individuals with dyslexia and controls, and could also affect oculomotor control. Their proposal encompasses both ‘bottom-up’ oscillatory mechanisms, which they suggest may exhibit atypical phase-locking to theta frequency fixations during reading (i.e., linked saccades, which typically occur every 300 ms or 3 Hz), and ‘top-down’ oscillatory control of faster visual signals in order to control oculomotor movements that capture images of alphabetic letters. While currently tied to the act of reading itself, these ideas enable visual temporal sampling mechanisms to be explored in infancy. In particular, visual temporal sampling mechanisms related to bottom-up processing should be measurable in infancy.

The account proposed by Archer et al. (2020) is an anatomically and functionally sophisticated version of ideas also proposed by Goswami et al. (2014), who made an early attempt to unify theorizing in developmental dyslexia. Their proposal was based on the function of neuronal oscillations in sensory systems. Goswami et al. suggested that oscillatory temporal sampling mechanisms could have systematic effects in more than one sensory system, which should be detectable from infancy. In principle, deeper understanding of these systematic effects across development could offer explanations for the multiple deficits that are typically seen in older children with dyslexia. Again, the similarity in the fundamental circuitry layout underlying the temporal processing of rhythmic acoustic events and visual motion processing in crickets noted by Schöneich et al. (2015) is thought provoking. In the future, such circuits could potentially offer a neural target for investigation.

However, given the complexity of neural learning, testing a unified multimodal temporal sampling theory of this nature makes it imperative to begin in infancy and to conduct longitudinal studies (see Figure 1.2). Studying developmental trajectories
is critical for understanding the complex interplay of auditory and visual sensory/neutral and cognitive processes during the development of reading, particularly as orthographic systems are not neutral visual stimuli. To add to the challenge, studies are required in multiple languages, in order to disentangle core causal features from developmental aspects that arise from the nature of the phonology and/or orthography of a particular language. For example, adult dyslexic readers of Farsi (which is read right to left) show a spatial attention deficit that is a mirror image of Italian dyslexic deficits (Kermani et al. 2018), suggesting that these spatial attention deficits are a consequence of dyslexia rather than a cause. By adulthood, readers in most languages will have read millions of words and practiced reading daily. Those with dyslexia will have read and practiced far less, because of the effort involved in fluent reading. Disentangling the effects of reading experience on the brain across the many different sensory and cognitive components shown in Figure 1.1 is experimentally challenging. Nevertheless, it is mandatory if societies are to devise efficient educational methods for remediating developmental dyslexia.

1.7 Conclusion

I have emphasized repeatedly in this chapter that a focus on development is absolutely critical to identifying core factors when testing theories about developmental dyslexia and devising effective remediation. An experimental road map for our field was set out in Figures 1.1 and 1.2, summarizing some of the key developmental issues to consider when testing a unified theory of dyslexia. Computational modelling of different theories is also important (e.g., Ziegler et al. 2020; see also Chapter 6, Malanchini and Gidziela 2022). Nevertheless, remediation methods that work in stringently designed studies should always be offered to children with dyslexia, even if there is disagreement over their theoretical basis. For example, using larger print, using shorter sentences in texts, not crowding letters spatially, providing early and explicit instruction in phonological awareness at levels other than grapheme–phoneme correspondences, and using modern multimedia solutions (such as screen reader software for reading aloud words, and speech recognition software for writing and spelling) should all be encouraged (Schneider et al. 2000; Schneps et al. 2013; Zorzi et al. 2012).

An interesting outstanding research question is whether targeted remediation given prior to learning to read could ameliorate dyslexia altogether (Goswami 2020). For example, building on the auditory temporal sampling theory, amplifying the speech signal so that 2 Hz amplitude modulations are exaggerated combined with engineering larger rise-time cues to facilitate sensory triggering of automatic phase entrainment should normalize phonological development across languages. It has already been shown that rise-time enhancement improves the performance of children and adults with dyslexia when listening to speech in noise (Van Hirtum et al. 2019; Van Hirtum et al. 2021). If infants and toddlers always heard speech with such modifications, perhaps through a discrete hearing aid, would a phonological mental lexicon develop that was comparable to not-at-risk children? If so, this would level
the sensory playing field before the child entered school and began learning to read. Presenting the deaf brain with a modified speech signal has been very successful in the case of cochlear implants. If used from infancy, these implants support the development of spoken language skills. Which factors to remediate, and how to go about such remediation prior to the onset of learning about print, are critical questions for theories of developmental dyslexia.

Suggestions for Further Reading


