Why theories about developmental dyslexia require developmental designs

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This article examines the importance of developmental designs in dyslexia research using a neuroconstructivist framework. According to neuroconstructivism, the lowest level of impairment should be identified as early as possible, and developmental effects on higher-level cognition examined longitudinally. A number of recent studies proposing candidate low-level impairments have not used such developmental designs. The role of normal variation in postulated causal factors on development is ignored, inadequate control groups are used, and the nature and timing of environmental inputs are not measured, even though reading is taught systematically and both reading acquisition and dyslexia vary with orthography. It is suggested here that only a phonological deficit arising from low-level auditory processing problems meets the criteria for a neuroconstructivist approach.

It is widely agreed that developmental dyslexia is caused by a ‘phonological core deficit’[1]. However, the lower-level deficits creating this cognitive deficit are hotly debated. The most logical low-level candidate mechanism, a deficit in basic auditory processing, has been rejected [2]. It has been argued that magnocellular [3], cerebellar [4], attention-shifting (parietal) [5] or general sensory-motor (temporal) [6] deficits could cause reading and spelling problems. Studies of developmental disabilities require developmental research designs, such as Karmiloff-Smith’s ‘neuroconstructivist’ research strategy [7], if meaningful causal inferences are to be drawn from data. When developmental criteria are applied to existing data, recently-proposed lower-level deficits prove unsatisfactory. It is suggested here that it is time to return to a serious investigation of basic auditory processing deficits in dyslexia, moving the research focus from the segmental (rapid temporal processing) to the supra-segmental temporal level.

Normative development of reading and spelling across orthographies

Many aspects of language develop according to normative timetables, but reading requires specialized tuition. This means that an environmental factor (age of school entry) determines initial reading development. English children begin formal schooling at age 4 or 5, Finnish and Norwegian children at age 7, Dutch and German children at age 6. There are also individual differences in the rate of normative acquisition determined by IQ, social class and gender: progress in reading is related to IQ [8], children from lower social classes typically acquire reading more slowly [9], and in virtually all languages boys lag girls in reading acquisition [10]. Millions of children in the world never learn to read because of environmental factors: poverty or inadequate schooling means that they are never taught. Being illiterate is not the same as being dyslexic, however. To be classified as dyslexic, children must display problems in acquiring written language despite receiving adequate training, having normal intelligence and lacking obvious sensory or neurological damage.

Studies in developmental psychology have also shown that across gender and social class, the rate and success of reading development varies with the nature of the orthography being learned ([11], see Table 1). Children learning to read consistent alphabetic orthographies like Italian, Finnish and German show the fastest rates of acquisition, attaining 90% accuracy in recoding letter strings to sound within the first months of reading instruction. Children learning to read English require on average 3 to 4 years of instruction to reach similar levels of successful recoding [12]. The main developmental task for children learning consistent orthographies is to improve their reading comprehension and

<table>
<thead>
<tr>
<th>Language</th>
<th>Familiar real words (% correct)</th>
<th>Monosyllabic non-words</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greek</td>
<td>98</td>
<td>97</td>
</tr>
<tr>
<td>Finnish</td>
<td>98</td>
<td>98</td>
</tr>
<tr>
<td>German</td>
<td>98</td>
<td>98</td>
</tr>
<tr>
<td>Austrian German</td>
<td>97</td>
<td>97</td>
</tr>
<tr>
<td>Italian</td>
<td>95</td>
<td>92</td>
</tr>
<tr>
<td>Spanish</td>
<td>95</td>
<td>93</td>
</tr>
<tr>
<td>Swedish</td>
<td>95</td>
<td>91</td>
</tr>
<tr>
<td>Dutch</td>
<td>95</td>
<td>90</td>
</tr>
<tr>
<td>Icelandic</td>
<td>94</td>
<td>91</td>
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<tr>
<td>Norwegian</td>
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<td>93</td>
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<tr>
<td>French</td>
<td>79</td>
<td>88</td>
</tr>
<tr>
<td>Portuguese</td>
<td>73</td>
<td>76</td>
</tr>
<tr>
<td>Danish</td>
<td>71</td>
<td>63</td>
</tr>
<tr>
<td>Scottish English</td>
<td>34</td>
<td>41</td>
</tr>
</tbody>
</table>

Table 1. Data for reading of familiar words and non-word reading by children learning different languages during their first year of instruction (from Seymour et al. [11])
their spelling. English-speaking children must first improve their basic recoding skills. By the age of around 10 years, differences in reading development due to orthographic consistency have largely disappeared. Now the factors that co-vary with poorer reading acquisition are gender and culture. Countries like Spain and Italy, with greater variation in living standards, show lower overall attainment by age 15 than countries like the UK [10].

**Developmental dyslexia across orthographies**
The manifestation of developmental dyslexia also varies with orthography. Dyslexic children learning to read orthographically consistent languages only show impaired recoding when they are tested at the very beginning of the acquisition process. They achieve high levels of accuracy in recoding relatively quickly [13,14]. The process of recoding remains effortful and slow, thus the diagnosis of dyslexia depends on reading speed. However, languages that are consistent for reading are seldom consistent for spelling. Poor spelling is the other international hallmark of dyslexia. Dyslexic children learning languages like Finnish can recode any letter string, but are functionally dyslexic because it takes them so long to recode each word that reading comprehension is impaired. Dyslexic children learning languages like English rarely achieve consistently accurate recoding, even after years of remediation. In inconsistent orthographies, dyslexics have both speed and accuracy problems.

**Investigating the causes of developmental disorders**
It is clearly crucial to understand normative development when generating causal theories of developmental disorders. Indeed, Karmiloff-Smith [7] argued that disorders must be studied in early infancy and longitudinally, to explore how alternative developmental pathways might lead to different phenotypical outcomes. Studying the end state in older children and adults ignores the dynamics of development. Second, it is important to separate behavioural outcomes from underlying cognitive processes. Similar behavioural outcomes could stem from different cognitive processes, and so control groups should be matched on cognitive processes and not behavioural scores. Third, environmental input will be crucial in shaping development, and will be dynamic rather than static. This means that the impact and timing of different environmental inputs needs to be understood.

Karmiloff-Smith’s analysis suggests three criteria that can be applied to different explanations of developmental disorders to assess their rigour:

1. What is the role of normal variation in the assumed causal factors on development?
2. Have experimental tasks been devised that differentiate behaviour from cognitive processes, and are appropriate control groups being used?
3. How do differences in the nature and timing of environmental inputs affect the development of the assumed causal factors?

In addition, the following methodological considerations are crucial for interpreting dyslexia data.

**Matching for verbal and non-verbal IQ**
Careful matching of experimental and control groups for IQ is very important. Comprehensive measures of IQ need to be used, and differences in verbal as well as nonverbal IQ must be minimised. Tests such as the Ravens Progressive Matrices are not a good choice for studies of dyslexia when they are the only matching measure used. This is because nonverbal tests are by definition related only weakly if at all to verbal skills. Dyslexics matched to controls on a test like the Ravens can show large differences in verbal IQ, and accordingly large differences in any experimental task with even the mildest verbal loading [15]. This does not mean that these experimental tasks have pinpointed the underlying causes of dyslexia.

**Selecting control groups**
Selection of appropriate control groups is part of criterion 2. Experimental studies of dyslexia require both chronological age (CA: children matched to the dyslexic children for IQ and age) and reading level (RL: children matched to the dyslexic children for IQ and reading level) controls [16]. The (younger) RL control group enables causal hypotheses to be generated (note that RL matches are impossible in studies exclusively of adults). Dyslexic children will typically have had 2–3 years more tuition in reading than RL controls and will also have higher mental ages. If deficits are found in particular cognitive tasks compared with both CA and RL controls, dyslexic children’s development is significantly slower than it should be given their developmental level and given the level of reading attained. This suggests a causal link with dyslexia (a training study is then required to test the causal hypothesis [17]). The third desirable control group is one of ‘garden variety’ poor readers, children matched to the dyslexics for age and reading level but of lower IQ This group will be similar to the dyslexics in terms of their behaviour on some cognitive tasks, but provide a means of investigating whether different cognitive processes have led to similar behavioural outcomes.

Note that it is sometimes assumed that studies exploring low-level causal deficits only require CA controls, because neurological maturation is being measured [18]. This argument is flawed. Child-driven variation in environmental effects (e.g. avoiding reading) can have differential effects on neurological maturation [7], making younger RL controls informative. Similarly, low-level deficits may no longer be detected in remediated adults, but may have been crucial earlier in development. For example, it cannot be concluded that auditory disorders do not influence the development of phonology because only subsets of adults show such disorders [2].

**Interpreting studies of adult (remediated) dyslexics**
When only adult ‘developmental dyslexics’ are studied, it must be recognized that there is a strong risk that ‘garden variety’ poor readers have been selected rather than dyslexics. Typically, such studies select two samples of undergraduates, those who report a history of reading disorders and those who do not. Processing abilities in the two groups are compared, and deficits are found in the ‘dyslexic’ group. Without concurrent empirical verification
of self-reported dyslexic status and any form of IQ matching, strong conclusions cannot be drawn from such data. Absence of IQ matching is particularly important in studies based on psychoacoustic tasks, where attentional skills are paramount (for example [19,20]). Similar caution applies if the two groups are matched for IQ using a non-verbal measure only, or if differences in IQ are not controlled for (for example [21,22]). Several newer low-level causes proposed for dyslexia rely on such designs, including certain auditory or parietal deficits, for example, auditory stream segregation, pure tone frequency discrimination, sluggish attentional shifting/parietal deficits [19,21,5]. It is crucial to assess theories derived from adults using developmental designs and developing children. For example, Wimmer et al. [23] report evidence from children suggesting that the increased parietal activity reported by Hari and Renvall [5] does not reflect a basic, biologically founded attentional deficit in dyslexia. Rather, it is a reflection of dyslexic individuals’ deliberate strategies for reducing visual attention to a small number of letters when trying to recode non-words.

Applying a neuroconstructivist research framework

I will now assess some explanations of developmental dyslexia using the criteria derived from Karmiloff-Smith’s neuroconstructivist research strategy.

The phonological core deficit

Dyslexic children across all languages so far studied show impairments in tasks that rely on the efficient functioning of the phonological system. Classically, they display deficits in three core areas:

(i) phonological awareness (the ability to identify or manipulate sounds within words)
(ii) phonological memory (the short-term retention of speech-based information)
(iii) rapid production of familiar phonological labels in response to symbols (such as digit names, colour names, object names; this is often called ‘rapid automatised naming’ or RAN)

In terms of our first criterion concerning normative development, the connection is clear and strong. Experimental studies across languages have revealed a robust link between children’s phonological skills and their progress in reading and spelling. In fact, variation in ‘phonological awareness’ is the most accurate predictor of later reading and spelling acquisition that we have, even for languages like Chinese that do not use the alphabetic principle [24]. Importantly, training phonological awareness improves reading acquisition across languages. Children who learn to read early or who appear to teach themselves to read have excellent phonological skills [25]. Children with moderate hearing impairment show poorer phonological skills and impairments in reading, but not the marked impairments characteristic of dyslexia [26] (profoundly deaf children are however very poor readers).

Importantly also, phonological skills develop. The classic developmental profile is described in Box 1. Performance at each linguistic level can be poor for several reasons. For example, children with lower IQ’s perform more poorly than children with higher IQ’s. This raises our second criterion, the differentiation of behaviour from cognitive processes via appropriate control groups. Crucially, in garden variety poor readers phonological deficits arise from a different developmental cause. Impoverished vocabulary leads to poor group performance in phonological awareness tests. For words that they actually know, individual low-IQ poor readers can usually manipulate phonology quite efficiently [27].

Dyslexic children, by contrast, have wide vocabularies. Nevertheless, the stringent RL match design has revealed phonological deficits across languages. Dyslexic children are not simply worse than CA matched children in phonological awareness tasks, but are worse than younger RL matched children. As development progresses, the picture changes. Intensive remediation focused on phonology can help to reduce differences with RL controls, until only differences with CA controls remain. Furthermore, depending on the orthography being acquired, dyslexic children can become indistinguishable from CA controls for measures of accuracy (although not speed). This is particularly noticeable for tasks measuring phonemic awareness [28], and is presumably because many phonemic awareness tasks can be solved on the basis of letter knowledge. Again, this is an environmental effect: learning to read a more consistent orthography helps to specify phonemes because of the 1:1 mapping from graphemes. Deficits that affect particular levels of phonology can also explain why a bilingual child can be dyslexic in one language (such as English) but not in another (such as Japanese) [29]. Japanese is a syllabic script whereas English is alphabetic; reading English requires sub-syllabic skills whereas reading Japanese does not.

Differences in the nature and timing of environmental input thus affect the development of phonological awareness and reading, which was our third test of theoretical stringency. More recently, such differences have been supported by longitudinal studies following the development of infants at risk for dyslexia by virtue of having two dyslexic parents [30,31]. These few studies are revealing differences in the rate and nature of phonological production in infancy (e.g. babbling is delayed, and fewer complex sounds are produced), as well as subtle auditory difficulties (e.g. temporal duration detection for non-speech sounds is impaired).

These recent longitudinal studies are the first examples of the neuroconstructivist research strategy in dyslexia (see [32] for SLI). The possibility that duration detection is impaired from infancy in dyslexia suggests that low-level auditory processing of supra-segmental temporal parameters may be important for the development of high-quality phonological representations. Deficits in processing auditory input are the most logical and parsimonious cause of the impaired development of phonological representations, yet have lost popularity as causal explanations of dyslexia. One reason may be that research has focused at the wrong temporal window (see Box 2). Meanwhile, the phonological core deficit model passes all of our criteria for a developmental model of a developmental disorder.
Box 1. The development of phonological awareness

Phonological awareness refers to the awareness of the structure of sounds within words. Studies across languages suggest a developmental progression, from phonological awareness of ‘large’ units of phonology (syllables, onsets and rimes) to phonological awareness of ‘small’ units (phonemes) (see Figure I, Table I). The onset in a spoken syllable comprises the consonant phonemes before the vowel, the rime is the vowel phonemes and any subsequent consonants (the coda), for example, in street, str- is the onset and -eet is the rime; similarly for spring. Words with two syllables have two onsets and two rimes, for example, w-ig-w-am. A phoneme is the smallest unit of sound that changes the meaning of a word, for example, cot and cat differ in their medial phoneme. Phonemic awareness does not seem to develop automatically with age, as adult illiterates perform poorly in phonemic awareness tasks [59]. Pre-literate children also rarely manage such tasks, but phonemic awareness usually develops rapidly once literacy instruction commences. Importantly, phoneme-based lexical representations do not seem to be present before learning to read [60–61].

The magnocellular deficit

There are of course other low-level deficits that may cause impoverished phonological representations. One is the impaired development of magnocellular neurones. The magnocellular theory originally argued for deficits specific to the visual magnocellular system. Dyslexic impairments in tasks measuring binocular vergence control and contrast sensitivity [33–34] suggested a deficit in coherent motion processing, indicating deficits in the ‘dorsal stream’ of visual processing served by magnocells. Motion detection is important for controlling eye movements, thus poor control can lead to unstable binocular fixation [35]. The causal argument made was that deficits in the dorsal stream reduced dyslexics’ ability to determine the spatial position of letters relative to each other. Letters appeared to move around during reading, causing mis-sequencing of letters and impaired reading. The magnocellular theory was the first to propose a route from the neural substrate to cognition.

We now know a great deal about the normative development of dorsal stream processing [36], enabling us to assess whether variation in the development of dorsal stream processing affects reading and spelling. Interestingly, the dorsal stream of visual processing appears to be more developmentally vulnerable than the ventral stream (this latter stream deals predominantly with object and face recognition and colour). Braddick and colleagues [36] report specific deficits in dorsal stream function in children with Williams syndrome, hemiplegic children with normal IQ’s, and in children with autism. Yet children with Williams syndrome develop reading (phonological recoding) skills commensurate with their verbal mental age [37], and many autistic children develop excellent recoding skills despite having very low IQs. [38]. This would not be expected if a dorsal stream vulnerability caused letter position confusions (see also [39]).

Our second criterion concerned experimental tasks and appropriate control groups. Most of the experimental tasks used to assess the magnocellular deficit are psychophysical ones, which means that attentional or IQ deficits will have serious effects on performance levels. It is therefore crucial to exclude children with attentional disorders and to match RL and CA control groups for IQ. Different sampling criteria may go some way to explaining failures to find magnocellular deficits across languages [40–41]. Few studies have included an RL control group, and magnocellular function in garden variety poor readers has not been studied.

Finally, we can ask about differences in the nature and timing of environmental inputs. Studies of infants suggest that early dorsal stream development is governed by progressive myelination. Adult motion coherence thresholds are not reached until around 8 – 10 years of age. This yields a large developmental window during which environmental events could influence binocular control, including aspects of early reading acquisition (for

Table I. Examples of phonological awareness tasks for children

<table>
<thead>
<tr>
<th>Phonological level</th>
<th>Instructions</th>
<th>Example</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Syllable</td>
<td>Tap once for each beat in the word</td>
<td>valentine</td>
<td>3 taps</td>
</tr>
<tr>
<td>Syllable</td>
<td>Do these words share a sound at the beginning?</td>
<td>hammer, hammock</td>
<td>yes</td>
</tr>
<tr>
<td>Onset – rime</td>
<td>Which is the odd word out?</td>
<td>cot, pot, hat</td>
<td>hat</td>
</tr>
<tr>
<td>Onset – rime</td>
<td>Do these words share a sound at the beginning?</td>
<td>broom, brand</td>
<td>yes</td>
</tr>
<tr>
<td>Phoneme</td>
<td>Do these words share a sound at the beginning?</td>
<td>rail, snap</td>
<td>no</td>
</tr>
<tr>
<td>Phoneme</td>
<td>Tap once for each sound in the word</td>
<td>steak, sponge</td>
<td>yes</td>
</tr>
<tr>
<td>Phoneme</td>
<td>Delete the first sound from this word</td>
<td>twist, brain</td>
<td>no</td>
</tr>
</tbody>
</table>

Figure I. The linguistic units within the monosyllabic word ‘street’ presented in terms of hierarchical level. The syllable level is the highest in the hierarchy and the phoneme level – the smallest unit that changes the meaning of a word – is the lowest.

Phonological level

- Syllable
- Onset / rime
- Onset / vowel / coda
- Phonemes

Figure I
The cerebellar deficit

Another low-level deficit proposed to explain the phonological deficit is cerebellar. The cerebellar deficit explanation originated in the idea that ‘automaticity’ might provide a new framework for dyslexia research [44]. Dyslexic children were said to be generally impaired in the non-conscious processes whereby repeatedly performed behavioural or cognitive sequences become fluent. For example, they showed apparently striking problems in areas of motor skill such as balance [45–46]. The automatisation deficit was claimed to stem from a cerebellar deficit, because the cerebellum is important for the automatisation of motor skills [44].

Unfortunately for the application of criterion 1, little is known about the normative development of cerebellar-related behaviours in children. Adults with acquired cerebellar damage display clinical signs such as hypotonia (e.g. hand wobble after arm shake), unusual fatigability following repetitive movements, and difficulties in controlling the rate and force of voluntary movements [47]. Importantly, there are no reports of dyslexia following acquired cerebellar damage [48]. Tasks typically used to assess cerebellar patients, like speeded reaching with visual guidance or speeded toe tapping, have yet to be
Karmiloff-Smith argued that longitudinal brain imaging studies of auditory deficits across languages. Finally, research strategies are required, as are longitudinal supra-segmental temporal level. Stringent developmental persuasive, and so the research focus should shift to the temporal processing deficits in dyslexia have not been essential for the field to move beyond studies of adult data. Meanwhile, the rejection of a causal role for low-level causal explanations of developmental dyslexia on current The low-level impairments proposed by the magnocellular dyslexia in comparison with these other disorders. Furthermore, attempts to find poor balance in dyslexic children across languages have largely failed. Importantly, these replications have tended to exclude dyslexic children with ADHD [18,51,52]. Only one study to date has included all the important control groups, namely dyslexic children without ADHD, ADHD children without dyslexia, and children with neither ADHD nor dyslexia [53]. This study found balancing difficulties only in the dyslexic + ADHD children, and the children with ADHD. The children with ADHD only were not poor readers. Regarding criterion 3, the environment plays an important role in automatisation, as the deficit is defined by failure to automatise even after long practice. All motor skills improve with practice, and dyslexic children appear no exception, for example apparently acquiring sports skills as well as their peers. For cerebellar function, it has been pointed out that the cerebellum is especially sensitive to problems that occur during neurodevelopment. In fact, cerebellar abnormalities are found in schizophrenia, autism and ADHD. As noted earlier, ADHD-only and autistic children have good single word reading skills. Finally, no longitudinal studies of children with cerebellar soft signs are available to assess their reading development. Further information on the time course of cerebellar neurogenesis and maturation might help to distinguish the specific role, if any, that the cerebellum plays in dyslexia in comparison with these other disorders.

Conclusion
The low-level impairments proposed by the magnocellular and cerebellar explanations of dyslexia are present in many developmental disorders, and cannot be accepted as causal explanations of developmental dyslexia on current data. Meanwhile, the rejection of a causal role for low-level auditory explanations is premature. However, it is essential for the field to move beyond studies of adult ‘developmental dyslexies’. The data supporting rapid temporal processing deficits in dyslexia have not been persuasive, and so the research focus should shift to the supra-segmental temporal level. Stringent developmental research strategies are required, as are longitudinal studies of auditory deficits across languages. Finally, Karmiloff-Smith argued that longitudinal brain imaging studies should be used for developmental disorders. Such studies should be commenced, focusing in detail on lower-level deficits. Cross-sectional brain imaging studies have begun, including training studies, and all support the core phonological deficit explanation of dyslexia [54–58].

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