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Dyslexia as a case study of the application of the principles of neuroconstructivism to developmental disorders

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In this section, we consider the example of developmental dyslexia as an illustration of the principles of neuroconstructivism in the context of developmental disorders. In terms of progressive specialisation, we will encounter the idea that reading system emerges in part from specialisation of more general visual object recognition system that happens to have the correct properties for recognising written words. We will see evidence for an absence of this specialisation in dyslexia. In terms of competition / co-operation, we will encounter the idea that the reading system divides labour between lexical and semantic routes for naming; that the division of labour depends to some extent on the language in which reading is being learnt; and that atypical neurocomputational constraints may disrupt the normal division of labour in the acquisition of reading. In terms of partial representations, we will see that different partial representations of a word are used to drive naming, and that developmental anomalies in different partial representations (phonological, orthographic) may lead to overlapping behavioural deficits in reading. In terms of interactivity, we will see how

new knowledge of orthography alters the structure of phonological representations at the onset of literacy. In terms of restriction of fate, we will see that phonological interventions for dyslexia are more effective in aiding literacy development if they occur at the pre-school stage. Finally, in terms of ensocialment, we will see that the ease of learning to read depends on two factors: the granularity of the pre-existing phonological representations engendered by the individual's native language prior to onset of literacy, and the subsequent complexity of mapping from phonology to orthography for that language. The overt emergence of disorder of literacy crucially depends on the latter complexity. Thus the cultural specification of the reading task influences both normal and atypical development. In this chapter, we will once more consider convergent evidence from several disciplines, including psychology, neuroscience, genetics, and computational modelling.

The reading system in normal development: the specific from the general

In discussing the cognitive and neural systems underpinning reading, it is important to remember that in historical terms, reading is a recent cultural invention reaching back just a few thousand years. Indeed, a significant proportion of humans still do not read. It is highly unlikely, then, that evolution will have had time to produce domain-specific constraints to guide the development of the relevant cognitive systems (see, e.g., McCandliss, Cohen, & Dehaene, 2003). If in an adult there is a reading system per se, it must be a specialisation of a more general system for relating visual forms to concepts and to speech. Such specialisation will be driven by interaction with a

particular kind of environment, namely, a culture that demands expertise in reading and mandates the investment of educational time in its acquisition.

The Visual Word Form Area

In the literate adult, brain-imaging research suggests that the reading system recruits up to a dozen different areas of the brain (Dehaene, 2003). Dehaene, Cohen and colleagues (e.g., L. Cohen et al., 2000; L. Cohen et al., 2002; Dehaene et al., 2002; McCandliss, Cohen, Dehaene, 2003) investigated one such region, the Visual Word Form Area in the left inferior temporal region and specifically the left fusiform gyrus. This area falls within the ventral stream typically involved in visual object recognition. In imaging studies, the visual word form area has been found to be most active in response to written word forms. However, although activation levels are higher for orthographically legal letters strings than illegal letter strings, activation levels are equal for words and pseudowords (made-up letter strings that are plausible words). Therefore the area appears to recognise visual stimuli that look like words prior to establishing whether they possess a meaning (Dehaene et al., 2002). Interestingly, the location of the visual word form area appears highly consistent across individuals, falling within approximately 5mm irrespective of the language (and therefore script) that the individual has to learn (Dehaene, 2003; McCandliss et al., 2003). McCandliss et al. (2003) argued that this consistency derives from the constrained nature of the task that script recognition demands of the visual system. They pointed to work by Malach and colleagues (Malach et al., 2002) indicating the existence of a computational gradient across ventral visual cortex such that regions furthest from the centre of the brain respond to fine-scale detailed images while those closer to the centre respond to larger scale images involving peripheral visual fields.

The result is a gradient of areas that preferentially respond – moving respectively from the outer part of ventral visual cortex towards the centre – to objects and words, then faces, then buildings, and then outdoor scenes.

The area that eventually comes to specialise in the recognition of visual word forms is the area that has the appropriate computational constraints for the recognition of visual stimuli of this particular size and detail. These are the dual requirements for fine foveal discrimination and invariant recognition of letter- and word-sized units (McCandliss et al., 2003). In addition, the area must comprise representations at a level of abstraction where the appropriate perceptual invariances required of script recognition can be computed. Fluent reading requires that the relevant partial representations of the visual input should be insensitive to changes in the font, size, case and position of words in the visual field.

Although the position of the visual word form area depends on the location in the brain where the appropriate information and computational constraints come together, Dehaene (2003) also argues that the range of constraints in the visual system of the pre-literate human brain would also limit the cultural variation of scripts that humans have invented. That is, human societies will only invent scripts to be written and read that are learnable given the constraints that the visual system contains. Humans and their cultural environment therefore co-specify each other via the external expression of the computational constraints of the cognitive system.

Nevertheless, the fact that a specialised reading function emerges across development from a more general system is underscored by brain imaging evidence that the activation of the visual word form area correlates with expertise in reading (B. A. Shaywitz et al., 2002). In this study, activations levels were correlated with grapheme-phoneme decoding skills even when age was controlled for, implying that

changes in the function of the visual word form area depend on expertise rather than maturation.

Cross-linguistic comparisons

While the computational capacities of the human visual system have constrained the invention of written forms, humans have not always made things easy for themselves. Presumably for rather involved historical reasons, some languages selected by human cultures appear to be a lot harder to read than others. The level of difficulty depends on two factors. The first is the appropriateness of the discriminations available in the pre-literate child's phonological representations, in terms of sound distinctions generated during acquisition of the language's spoken vocabulary, to which subsequent letters or letter clusters (graphemes) can map. The second is the consistency of the mapping between graphemes and component word sounds (phonemes) of a given language (Goswami, 2003). Let us consider these two factors in turn.

During language development prior to literacy, the requirement to learn increasing numbers of similar sounding spoken word forms forces the phonological representations to acquire increasing levels of granularity (Metsala & Walley, 1998). According to the 'lexical restructuring theory', word forms are initially stored as undifferentiated wholes, but the acquisition of new similar (and therefore confusable) words pushes phonological representations to store words according to their component syllables (e.g., 'seesaw' => 'see-saw'). Subsequently, the acquisition of new similar (and therefore confusable) syllables pushes phonological representations to breakdown syllables into onset and rhyme components (e.g., 'see' => 's-ee', where s is the onset and ee the rhyme).

When children are required to learn to read, the task (particularly for reading novel words) is to learn the general relation between graphemes and phonemes. For languages like Italian and Spanish that have many simple consonant-vowel syllables, a decomposition of words into syllables and syllables into onsets and rhymes is equivalent to splitting a word into component phonemes (e.g., 'casa' => 'c-a-s-a') (Goswami, 2003). Children learning these languages are well placed to learn the relation of graphemes to components of their existing phonological representations. However, in other languages such as German and English, many words have complex syllables ending in codas (e.g., the syllable 'cat' => 'c-a-t', where t is the coda). This means that with the onset of literacy, re-organisation of phonological representations is required to a further level of granularity beyond onset and rhyme to distinguish codas as separate entities, before graphemes can be mapped to component phonemes (Goswami, 2003).

Turning to complexity, some languages such as English and French have inconsistent mappings between phonemes and graphemes. Here the context of the letters surrounding a grapheme can alter the phonemes to which it corresponds. The amount of context may vary from the adjacent letters to the whole word. For example, in English, the letter 'i' in 'bit' maps to a different phoneme than the 'i' in 'bite', but to the same phoneme in 'sieve', but to a different phoneme in 'pint' and in 'aisle', and so on. This complexity presents additional challenges to deriving the relation between visual and spoken word forms, because the nascent reading system is required to develop and operate at multiple levels of granularity.

The consequence of the different computational challenges of each language is that cross-linguistic studies of reading have revealed differences in the rates at which literacy emerges. In all cases, learning about letters appears to cause a re-organisation

of the mental lexicon into phoneme-based representations. For languages with consistent mappings between graphemes and phonemes, the re-organisation is rapid, with grapheme-phoneme decoding ability close to ceiling within the first year of learning to read; in inconsistent languages like English, this process can take up to 3 years (Goswami, 2002, 2003). Here we see a clear example of interactions between visual and auditory representations, mediated by the complexity of the task present in the individual's cultural environment.

Developmental disorders of the reading system: atypical constraints

Between 5% and 17% of the school age population exhibit a reading disability, depending on how the disorder is defined (Shaywitz & Shaywitz, 1994). Two main sub-types of developmental dyslexia have been identified (e.g., Castles & Coltheart, 1993; Manis et al., 1996), although many dyslexics exhibit a 'mixed' pattern with symptoms of each type. In phonological developmental dyslexia, there is particular difficulty in reading novel or pseudowords. In surface developmental dyslexia, there is a particular difficulty in reading irregular words (such as 'aisle', 'quay', and 'yacht' in English). These two patterns of reading deficit are also found in healthy adults after some kinds of brain damage (see Thomas Karmiloff-Smith, 2002a, for discussion).

Genetics and dyslexia

There appears to be a genetic contribution to developmental dyslexia, which can be assessed by twin studies (see Pennington, 1999; Plomin & Dale, 2000, Plomin & Rutter, 1998, for discussion). While monozygotic (MZ) twins share the same genome, dizygotic (DZ) twins are on average no more genetically similar than siblings. To the

extent that a developmental disorder has a genetic cause, one should expect MZ twins to be more likely to share the disorder than DZ twins. The 'concordance' rate of a disorder is defined as the proportion of twin pairs in which both members of the pair meet some cut-off criterion for possessing the disorder. When DeFries and Alarcon (1996) examined 200 pairs of MZ twins and 150 pairs of DZ twins in which at least one member of each pair met strict criteria for reading disability, they found MZ twins were 68% concordant as compared to 38% concordant for DZ. To the extent that once can assume that twins tend to have much the same environment for learning to read, have roughly the same motivation to read and consequently, roughly the same level of experience with the task, the implication is that reading deficits are substantially due to genetic factors.

If the reading system is a recent, culturally induced specialisation of more general systems involved in visual object recognition and spoken language processing, it follows that we should not expect a genetic variation that is entirely specific to the reading system. Nevertheless, stories in the media have appeared proclaiming the discovery of the "gene behind dyslexia".[1] One such story was prompted by work searching for quantitative trait loci (QTLs) correlating with developmental disorders. QTLs are areas of chromosomal similarity in individuals who exhibit a developmental deficit and represent candidate locations for genes implicated in contributing to the disorder. The assumption behind QTLs is that many genes may contribute different quantitative amounts to the probability of having a given disorder but that these genes may be involve in multiple functions (see Plomin and Dale, 2000, for an introduction). The media story was prompted by a study by Fagerheim et al. (1999) who studied a Norwegian family in which dyslexia was

common and identified a region of chromosome 2 as a possible contributor. (Other work has pointed to regions of chromosome 6 and chromosome 15; see Pennington, 1999, for discussion).

The media story in question accepted that the processes involved in reading and writing might involve several genes. However, it viewed the identification of a gene for dyslexia as a potential breakthrough, since this would permit early screening and identification of dyslexics, followed by intervention to correct the impairment when the 'brains of children are most plastic', an idea to which we will return in the computational modelling section. Interestingly, the media story also quoted the chief executive of a national dyslexia association as arguing that the 'gene for dyslexia' shouldn't be 'removed or tampered with' because 'people with dyslexia, who are forced to think in alternative ways, are often revealed as geniuses'. The supporting empirical evidence for this claim comprised the case studies of a famous war leader, a famous businessman, a famous TV presenter, and a famous comedian, all of whom are dyslexics and all of whom are apparently geniuses (even the TV presenter).

The specificity of cognitive differences and brain differences in dyslexia

In our general discussion on developmental disorders, we argued that genetic effects on brain development are rarely region specific, and that in disorders of a genetic origin brain differences are frequently widespread. If the computational constraints of the developing brain have been altered by a genetic mutation in developmental dyslexia, we should not expect deficits to be reading specific but to show up in other cognitive domains. Perhaps the reading task particularly exacerbates computational constraints that are anomalous in the wider visual system, affecting the ability to

[1] "Gene behind dyslexia is discovered" London Metro, Tuesday September 7, 1999

recognise visual word forms; or particularly exacerbates computational constraints that are anomalous in the spoken language system, affecting the ability to create the necessarily granularity in phonological representations. Nevertheless, we should expect to find behavioural deficits elsewhere. The specificity of developmental dyslexia at both the cognitive level and the brain level, then, is highly relevant. However, currently there is little consensus in the literature at either level.

For example, at the cognitive level, it has been argued that individuals with dyslexia show deficits beyond reading and phonology, including variously auditory deficits (Tallal, 1980), visual deficits (Lovegrove, Bowling, Badcock, & Blackwood, 1980), tactile deficits (Stoodley et al., 2000), motor deficits (Fawcett, Nicolson, & Dean, 1996), learning deficits (Nicolson & Fawcett, 2000), attention deficits (Hari & Renvall, 2001). Dyslexia has also been portrayed as a general sensorimotor syndrome (Stein & Walsh, 1997). However, some have argued that developmental dyslexia is specifically phonological, with other features as co-incidental co-occurring factors or indirect side effects (e.g., Ramus, 2002; Ramus et al., 2003).

At the brain level, the last 5 years have seen around 50 scientific articles reporting the results of brain imaging experiments exploring brain anomalies associated with developmental dyslexia. The differences identified in the dyslexic brain show a significant degree of variability. In terms of functional magnetic resonance imaging (fMRI), core dysfunction is claimed to reside in and around the angular gyrus of the left hemisphere, but with associated compensation in posterior regions in the right hemisphere and left inferior gyrus (Milne et al., 2002). Claims are also made for anomalies in the organisation of the cerebellum (Rae et al., 2002), but so too for abnormalities of the magnocellular component of the visual stream specialised for processing fast temporal information (Stein & Walsh, 1997). On the

basis of imaging evidence, dyslexics are claimed to differ from controls both in letter rhyming (phonological) and visual letter matching tasks (orthographic) (E. Temple et al., 2001) and indeed not just in reading tasks but also tasks restricted to auditory language processing (Corina et al., 2001). Simos et al. (2000) make the precise claim that dyslexia results from aberrant patterns of functional connectivity between ventral visual association cortex and temporo-parietal areas in left hemisphere (see also Paulesu et al., 1996). Structural imaging suggests that dyslexia is associated with marked rightward cerebral asymmetry and marked leftward asymmetry of the anterior lobe of the cerebellum (Leonard et al., 2001), decreased grey matter in the temporal lobe (Eliez et al., 2000), and lowered corpus callosum in posterior regions (Robichon et al., 2000). McCandliss et al. (2003) point to evidence from several studies indicating a reduced tendency to activate the visual word form area during reading in dyslexics (fMRI: Paulesu et al., 2001; S. E. Shaywitz et al., 1998; MEG: Helenius et al., 1999), arguing that this is evidence that there is an absence of emergent specialisation of this region, through lack of relevant reading experience. In short, there is no consensus about whether structural differences are focused or widespread, or whether the range of tasks on which individuals show atypical brain activation patterns is narrow or diverse.

In terms of the two sub-types of developmental dyslexia, only phonological has generated a consensus on the underlying cognitive cause. In the 'phonological representations' hypothesis (see Goswami, 2000; Snowling, 2000, for reviews), the initial representations of speech sounds that children develop are deficient, especially in terms of the emergence of the onset and rhyme level of granularity. When children come to learn to read, their phonological representations are ill fitted for learning the relationship between phonemes and graphemes, because the relevant speech sounds

for the graphemes to connect to are not present, or at least their emergence is much delayed. As a consequence, the reading of novel words is impaired. Under this hypothesis, the problem pre-dates literacy, so it should also be apparent in tasks that do not involve visual (orthographic) information, such as short-term memory for words, nonword repetition, or speeded picture naming tasks (e.g., Swan & Goswami, 1997; see Goswami, 2003, for review of relevant literature).

Importantly, the impact of initial phonological representations with reduced granularity has a differential impairment on the emergence of dyslexic symptoms depending on the structure of the target language. Thus, consistent languages like Spanish and Italian mitigate the symptoms, while inconsistent languages like English and French exaggerate the problems (Paulesu et al., 2001). Nevertheless, non-orthographic tasks reveal spoken language deficits irrespective of language, and reading latencies are slower. At the brain level, Paulesu et al. (2001) found that Italian individuals recruited on the basis of slower reading speed and defective phonological processing who nevertheless had high levels of reading accuracy demonstrated the same altered brain activation patterns as English and Italian individuals with dyslexia. In this case, the common 'dyslexic' features were reduced activation in the left middle, inferior, and superior temporal cortex and in the middle occipital gyrus.

In terms of surface developmental dyslexia, the picture is less clear. Studies have tended to agree that individuals with this sub-type have failed to develop specific knowledge about word spelling, and that these individuals appear to demonstrate no deficits in their phonological representations, as assessed by tasks of phonological awareness (Castles & Coltheart, 1996; Goulandris & Snowling, 1991; Hanley, Hastie, & Kay, 1992; Manis et al., 1996; see Ans, Carbonnel, & Valdois, 1998, for discussion). Goulandris and Snowling (1991) found that individuals with surface

dyslexia had poor performance on tasks of visual memory, although instances of surface dyslexia in the absence of visual impairments have also been reported (e.g., Castles & Coltheart, 1996). Since many of the reading errors diagnostic of developmental surface dyslexia have also been found in younger typically developing children (Bryant & Impey, 1986), some have doubted its validity as a distinct developmental dyslexia syndrome (see Pennington, 1999, for discussion). However, the view that surface dyslexia is simply delayed reading development fails to explain why delay should selectively strike the reading system. Moreover, as we saw in Chapter X, the pattern of errors in a disorder is in part constrained by the nature of the task domain in any case, so that some similarities between early normal development and disordered development may be inevitable. The notion of 'delay' remains ill-specified at a mechanistic level and indeed, as we shall later see in Figure Y, computational modelling demonstrates that there are many constraints that produce slower learning as a side effect when those constraints become atypical.

Causal models of dyslexia

Let us assume for a moment that in individuals with developmental dyslexia, anomalies are not specific to brain areas involved in reading and that there are deficits beyond the task of reading. At least two causal models are then possible for how this state of affairs came about. First, at the neural level, there could have been a reasonably restricted anomaly in brain development caused by a genetic mutation, followed by knock-on effects in the development of other brain areas. Alternatively, there could have been a more widespread effect of the genetic mutation simultaneously affecting multiple brain areas. These two possible accounts would also exist at the cognitive level: narrower-becomes-more-widespread-with-development

vs. more widespread to begin with. At the cognitive level, however, there are the additional complications that some structural brain anomalies may have a greater or lesser computational consequence, and some cognitive processes may rely more or less on the computational properties that are affected by structural anomalies. The link between brain, neurocomputational properties, and behavioural deficits may be far from transparent.

In the face of these possibilities, the "actual" cause of a reading deficit depends to some extent on whether there is only one way to disrupt the nascent reading system to produce phonological dyslexia and only one way to produce surface dyslexia. If there are multiple routes to each (as indeed will be suggested by the later computational modelling section), then sub-types may be causally heterogeneous. That is, experimental groups may be clusters of individuals drawn together by virtue of sharing a particular (impaired) behaviour (Thomas, 2003b).

A recent proposal allows us to contrast the two causal models. Ramus (2002, 2003) suggested that at a cognitive level, phonological developmental dyslexia is a consequence of a specific phonological deficit. However, in addition the deficit is sometimes accompanied by a sensorimotor syndrome with variable manifestations but little additional impact on phonology and reading. Under this hypothesis, the original cause at the neural level are cortical layer ectopias and microgyri (focal anomalies of neural migration in the outer layer of the cortex), which are located mainly in the left peri-sylvian areas (Galaburda et al., 1985; Humphreys et al., 1990). These focal migration anomalies are viewed as being of genetic origin, based on evidence from mouse studies (Sherman et al., 1990). However, secondary to the cortical anomalies, thalamic anomalies arise in the magno-cells of the lateral and medial geniculate nuclei of the thalamus, which produce visual and auditory deficits respectively (Livingstone

et al., 1991; Galaburda et al., 1994). Based on animal studies, Ramus argued that this secondary effect only arises when the cortical anomalies coincide with excessive testosterone concentrations during brain development (Fitch et al., 1997; Herman et al., 1997; Rosen et al., 1999). This would explain why behavioural genetic studies indicate that the phonological deficit in dyslexia is highly heritable, while auditory and visual deficits are not (e.g., Bishop et al., 1999). Ramus invoked this account to explain why in a group of 16 adults with developmental dyslexia, a careful battery of tests revealed that all 16 had a phonological deficit, but 10 also had an auditory deficit, 4 a motor dysfunction, and 2 visual problems. Five, however, had a phonological deficit alone (Ramus et al., 2003).[2]

This account, linking a behavioural deficit with early genetic effects on brain development is speculative. Nevertheless, it illustrates our first causal model: that the initial genetic deficit is restricted to left peri-sylvian areas underlying subsequent phonological processing, but under some conditions there are secondary effects on brain development that spread the impact of the initial anomaly.[3] Ramus's theory currently lacks two aspects: an account of the consequences of the ectopias and microgyri for the computational properties of peri-sylvian areas, and a developmental account of why an attempt to acquire phonological representations in a system with these anomalous computational properties should lead to the deficits we see in dyslexia. At it stands, the theory incorporates a significant leap from focal neural migration anomalies in the neonatal brain to adults who show a reading impairments.

[2] The study was not designed to evaluate Ramus' claims about the role of testosterone in mediating the specificity of the deficit, a claim that in any case would apply to the conditions that held in early brain development rather than in adults with dyslexia.

[3] See Frith and Happé (1998) for an example of a cognitive level version of the narrower-becomes-more-widespread-with-development causal story with respect to dyslexia.

Ramus' (2003) hypothesis also highlights two of the issues raised in our earlier general discussion of developmental disorders. First, we argued that developmental disorders need to be viewed in terms of atypical limits on plasticity, given that healthy children with focal lesions do not usually show domain-specific deficits when they are older but instead exhibit recovery (Thomas, 2003a). If early brain anomalies in a dyslexics are indeed as focal as Ramus' account maintains, an explanation of subsequent behavioural deficits needs to address why there is no compensation of the cognitive system from other undamaged brain areas across development, sufficient to allow recovery. Thomas and Karmiloff-Smith (2002a) identified several candidate computational constraints that would explain the isolated atypical development of individual functional components against a background of normal function, a condition they labelled 'Residual Normality' (RN). Identification of such constraints is crucial if apparently selective behavioural deficits and an absence of compensatory recovery are to be fitted into a developmental theory of disorders. The computational constraints for RN are as follows: (1) strong structure-function correspondences, (2) strong competition, (3) early irreversible commitment, (4) inflexibly guided specialisation, and (5) resource limitations.

In order to explain the mapping between early focal deficit and domain-specific endstate deficit, Ramus (2002, 2003) appealed to two of the RN-preserving constraints, strong structure-function correspondences and strong competition. He proposed firstly that certain anatomical modules are unique in their ability to support certain cognitive modules – i.e., only peri-sylvian areas have the computational structural properties to compute the functions required in the domain of phonological processing; and secondly, that these areas are unique in their initial input and output connectivity – no other areas can compete to take over the input-output mappings of

phonological processing (even if they had broadly appropriate computational properties) because initial connectivity is strongly biased against them. They are not provided with the relevant inputs and outputs to start to compete to perform the function. Together, these two constraints would ensure that no other area could effectively compensate for inefficiencies in peri-sylvian areas. Of course, such a claim does not imply that individuals with dyslexia must demonstrate zero compensation compared to the normal reader in attempting to overcome phonological processing difficulties. Thus we saw in Chapter X how brain-imaging studies of dyslexics revealed differences compared to controls in the activation of both occipital and frontal areas during reading tasks, consistent with attempts of other brain areas to compensate for inefficient phonological processing (see Casey, Thomas, & McCandliss, 2001). But by virtue of being defined as dyslexics, we know that any such compensation must be insufficient to produce reading behaviour in the normal range. If individuals who had initial focal neural migration anomalies in peri-sylvian areas (or some other brain deficit) achieved successful compensation, then they would not have appeared in the dyslexic samples in any of these studies. This brings us to the second issue raised in our earlier general discussion of developmental disorders.

In Chapter X, we discussed the thorny question of the relationship between atypical variability in developmental disorders and other forms of cognitive variability. In this case, the key issue is the relationship between individual variability (general and specific 'intelligences') and atypical development. In developmental disorders that are defined on behavioural grounds alone, the sample of individuals recruited into a study necessarily conflates individual variation with atypical variation due to the disorder. For example, let us assume that individuals show independent variation in their levels of auditory, visual, and motor abilities, simply as part of the

individuation variation present in the normal population. Assume, too, that developmental dyslexia were to be caused (contra Ramus) by a genetic mutation with widespread effects in early brain development across auditory, phonological, visual, and motor areas. However, in this scenario, certain dyslexic individuals with above average performance in one or other of these abilities could show behaviour in the normal range on some subset of visual, motor, and auditory processing. (They could not show performance in the normal range in phonological processing because, by definition, they are recruited as dyslexics). The consequence would be individuals unified by sharing a phonological deficit, but varying in the other deficits they exhibit. This hypothesis would explain the same pattern of empirical data reported by Ramus et al. (2003), but appeal to the alternative causal model we identified earlier, that is, the simultaneous presence of more widespread genetic effects on brain development.

In terms of individual differences, low general intelligence can produce poor reading scores, but in this case against a background of low performance in other cognitive domains. It remains to be seen whether a reading deficit that is part of a domain-general pattern should be explained in terms of variations in different underlying neurocomputational parameters than in the case of domain-specific reading disability. Do individual variation and atypical variation converge on the same behavioural deficit? The same cognitive deficit? Fletcher et al. (1999) have argued that poor reading through generally low IQ and poor reading as an apparently domain-specific disability do not differ radically in the cognitive factors with which they are associated, such as poor phonological processing. A full account of cognitive variability in the developmental realm must one day unify domain-general and domain-specific accounts of poor performance (see Thomas & Karmiloff-Smith, 2003b, for discussion of this question).

Although Ramus's (2002, 2003) hypothesis omits a story of how initial computational deficits can lead to behavioural problems across development, other researchers have been active in applying computational models to addressing this question. We now turn to consideration of these models.

Computational modelling of developmental dyslexia

The importance of constructing developmental models of reading is highlighted by insufficiencies in static, hand-wired computational models of the adult reading system. For example, one such model proposes two mechanisms for reading, one that relies on information about whole words, linking whole visual word forms to their pronunciations (the lexical route) and another that relies on finer granularity information, linking graphemes to their corresponding phonemes (the nonlexical route) (Coltheart et al., 2001). If you attempt to extend this static model to explain developmental dyslexia, one story you can end up with is as follows: "Some children might be acquiring the components of the lexical route at a normal rate, but be having difficulty with [developing] one or more components of the nonlexical route. Such children would have a selective difficulty in reading nonwords aloud. This is developmental phonological dyslexia… Other children might be acquiring the components of the nonlexical route at a normal rate, but be having difficulty with [developing] one or more components of the lexical route. This is development surface dyslexia" (Coltheart et al., 2001, p.??).

In response to such an account one must ask, Why is there no compensation in the disorder? If only one of the two mechanisms is initially compromised, why doesn't the other initially intact mechanism compensate for it across development?

One can't simply assume that no between-route compensation would take place in dual-route models. Indeed Thomas and Karmiloff-Smith (2002a) demonstrated a dual mechanism system where precisely this kind of compensation and recovery takes place after initial damage to one of the mechanisms. Coltheart et al.'s claim only holds if, when the model is extended to the developmental realm, it embodies some of the computational constraints that lead to Residual Normality (listed in the previous section). Without specification of the developmental process, speculative claims about selective developmental deficits cannot be evaluated.[4]

However, the developmental causes of reading deficits have been the focus of a large body of developmental modelling work. We discuss these models in the following paragraph. First, however, a couple of caveats. Thus far, few of the models of developmental dyslexia have been based on a model of reading acquisition that captures all stages through which children pass when learning to read. Currently the normal models of development are somewhat limited. They tend to omit the early stages of the reading process when the child is using partial visual cues to recognise whole words, and when the child's visual system has yet to reach the correct perceptual invariances required to recognise letters. (For example, p, q, b, and d are confused because the child has to learn that normal perceptual invariances for visual objects such as rotation should not apply in the specialist domain of recognising letters; Dehaene, 2003). Nevertheless, the existing models are sufficient to give an idea of the implications of various computational constraints in learning the relation between written and spoken words. The second caveat is that to date, there has been a

[4] In contrast to Coltheart et al.'s proposal, Ramus (2003a) conversely argues that researchers now accept that there cannot be a specific developmental deficit in one of the two reading routes without affecting development in the other route. However, he maintains that there can be a specific deficit in

relative lack of cross-linguistic modelling that would permit an exploration of the interaction of atypical computational constraints with the particular language to which the system is exposed. The final caveat is that, due to lack of space, this discussion omits a consideration of the relative merits of the competing normal models on which atypical manipulations are based. The reader is directed to discussions in Ans et al., (1998) and Coltheart et al. (2001).

Connectionist models of the typical and atypical reading system

Developmental models of reading have tended to appeal to connectionist architectures, employing two-layer and three-layer networks, usually with distributed representations. Some models have included attractor networks and cycling activation, so that the system settles into stable solution states.

Connectionist models of reading assume that the computational problem in this domain is to learn to map between representational codes of the written form of a word, the spoken form of a word, and the word's meaning (Plaut, McClelland, Seidenberg, & Patterson, 1996; Seidenberg & McClelland, 1989). Typically, this involves three connectionist networks, one to map from orthography to phonology, one to map from orthography to semantics, and one to map from semantics to phonology (although in many models only the first of these networks is implemented; see Harm & Seidenberg, 2001, for an exception). Usually, each of these networks is assigned a three-layered structure comprising an input layer, an output layer, and an intermediate layer of hidden units.

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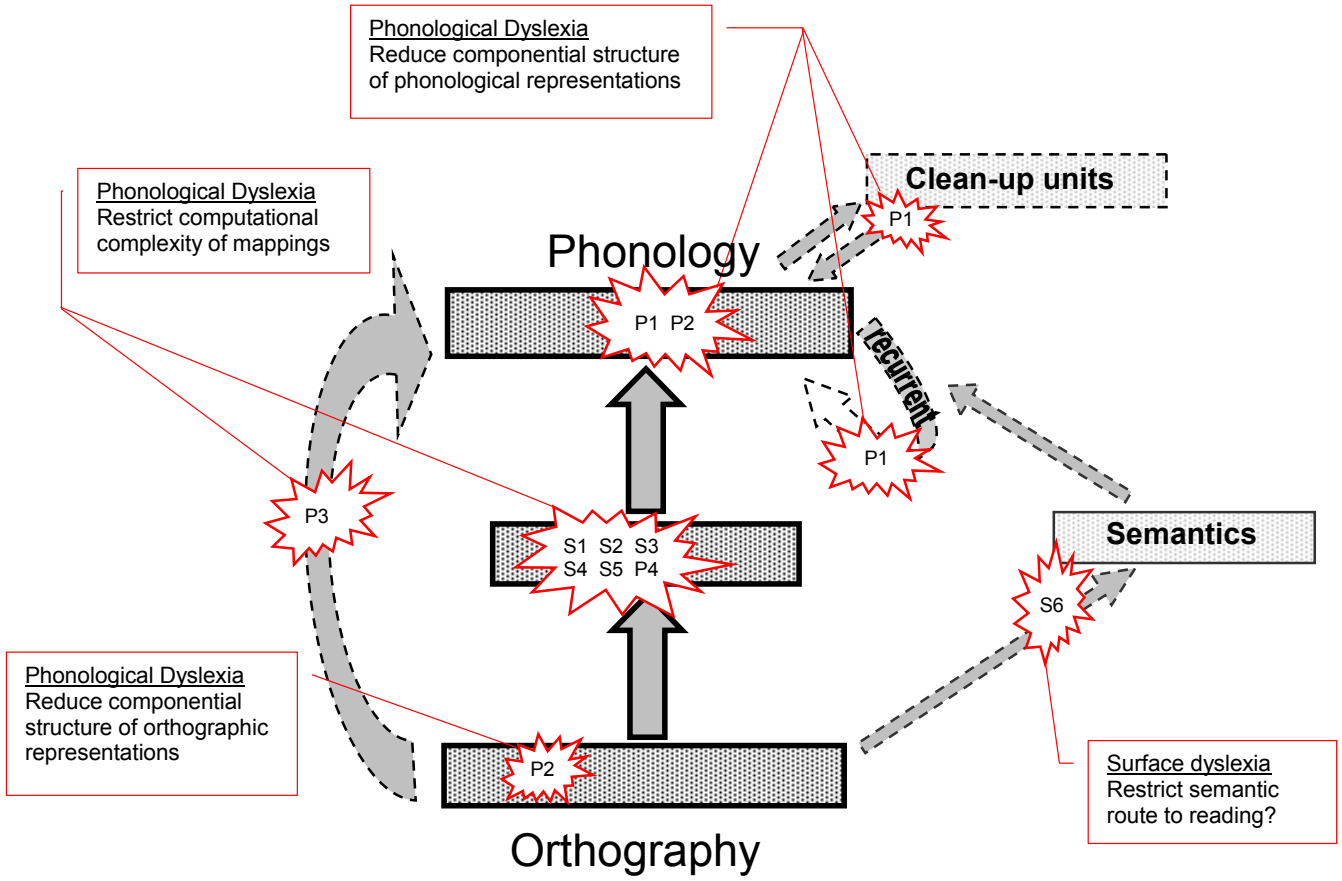
the development of phonological representations without affecting development elsewhere in the reading system. For discussion of this distinction, see Thomas and Karmiloff-Smith (2002b).

Some models employ recurrent connections to allow phonological representations to settle into stable output (attractor) states, while a fully implemented network might allow cycling interactions between all three representational codes. Sometimes a layer of 'clean-up' units is connected to the phonological layer to aid the settling process (see e.g., Harm & Seidenberg, 1999, 2001). In other cases, orthography is connected to phonology via two routes, one with direct connections the other involving an intermediate hidden layer (Zorzi, Houghton & Butterworth, 1998a). In such a dual route network, the Direct connections favour computations based on components of word representations (graphemes and phonemes) while the Indirect, hidden-unit-mediated route favours computations based on a larger granularity (combinations of graphemes or combinations of phonemes) (see Thomas & Karmiloff-Smith, 2002a, for discussion of this architecture and emergent specialisation). Ans et al. (1998) used a similar two-route architecture but with localist hidden unit representations (in their terminology, a multiple-trace episodic memory). A localist representational format is one where the activity of a single unit corresponds to the representation of a single entity and there is no similarity between the representations of each entity. In the Ans et al. model, the localist units were separately constrained to represent word components or whole words. Mappings between orthography and phonology could independently use one or other of these sets of processing units, pre-specified as two processing routes (or 'modes').

A composite architecture summarising all the various connectionist models is depicted in Figure X. In line with the general theoretical framework for the simulation of developmental deficits outlined in Chapter X, the sub-types of dyslexia were simulated in these models by altering certain initial computational constraints and

then exposing the system to a training set of written words and their pronunciations. Figure X also demonstrates the constraints that were changed in various models.

Figure X. Composite architecture of connectionist models of the reading system. The core assumption is a network mapping between orthography and phonology. Different models add other components (dotted outlines), such as (1) a semantically mediated route between these codes; (2) Direct vs. Indirect connections from orthography to phonology; (3) attractor networks for each representational code, include recurrent connections at output and/or clean-up units. P# stands for the site of startstate manipulations designed to simulate developmental phonological dyslexia. S# stands for the site of startstate manipulations designed to simulate developmental surface dyslexia (see text for references).



Simulating the sub-types of dyslexia

Surface dyslexia, an impairment in reading exception words, has been simulated by altering any initial constraints that reduce the general ability of the network to learn the relation between orthography and phonology (manipulations are labelled as 'S#' in Figure X). Exception words are the first to suffer from this degradation, since they are inconsistent with most of the knowledge gained from exposure to reading words. Constraints that have this effect have included a reduction in the initial number of hidden units in this network (S1: Bullinaria, 1997; Harm & Seidenberg, 1999; Plaut et al., 1996; Seidenberg & McClelland, 1989), a less efficient learning algorithm (S2: Bullinaria, 1997), less training (S3: Harm & Seidenberg, 1999), and a slower learning rate (S4: Harm & Seidenberg, 1999). In models that employ two routes to connect orthography and phonology, proposed manipulations have focused on impairing the Indirect (hidden-unit-mediated) route (S5: Ans et al, 1998; Zorzi, Houghton & Butterworth, 1998a). This route is able to process the larger granularity mappings required to encode whole word exceptions. Eliminating it reduces the network's ability to learn such exceptions using the remaining Direct orthography-phonology route.

Plaut et al. (1996) argued that reading may take place either via connections between orthography and phonology, or via a semantic route. They argued that a division of labour would be negotiated between these two routes by a competition during learning (unimplemented in their model). They speculated that exception words (especially of a low frequency) might preferentially rely on the semantically mediated route. Simulations in the related domain of inflectional morphology, which is also characterised by a partially regular mapping problem, support this idea. Joanisse and Seidenberg (1999) and Thomas and Karmiloff-Smith (2003a) have

demonstrated that where word-specific information such as a meaning is available at input, networks learning partially regular input-output mappings between uninflected and inflected phonological forms will exploit the word-specific information during learning to support exception mappings but not regular mappings. Moreover, Thomas and Karmiloff-Smith (2003a) found that removal of word-specific information from the startstate produced differentially delayed acquisition of the exception mappings compared both to regular mappings and generalisation. It therefore seems likely that in the general reading architecture in Figure X, a processing impairment (in connectivity, speed of processing, or quality of representations) in the semantically mediated route would also produce symptoms of developmental surface dyslexia compared to a normal system able to exploit this route (labelled S6 in Fig. X). Without the semantic route, the additional resource to support exceptions is no longer available. (Simulations in the past tense domain suggest that an absence of word-specific information impairs low frequency exceptions more than high frequency exceptions, thereby exaggerating the frequency by regularity interaction – a pattern sometimes reported in surface dyslexia, see Ans et al., 1998; Plaut et al., 1996, for discussion.)

Phonological dyslexia, an impairment in reading pseudowords, has been simulated in two main ways. Both methods cause the network to develop an insufficiently general function relating orthography to phonology. The first method reflects the claim we encountered earlier that phonological dyslexia corresponds to phonological representations (and perhaps orthographic representations as well) that have developed with insufficient componentiality (Manis et al., 1993; Plaut et al., 1996). Harm and Seidenberg (1999) implemented this proposal by restricting the initial computational properties of the phonological component of their model (the

phonological output layer, its recurrent connections, and its clean-up units). Their manipulations occurred prior to the onset of literacy and included (1) the initial removal of the clean-up units and severing half the recurrent connections between the phonological units, or (2) restricting the size of the weights in the recurrent connections, or (3) making computations within the phonological component more noisy. All of these manipulations resulted in poorer nonword naming, and some of them impacted on exception word reading as well (P1 in Fig. X). Brown (1997) also demonstrated that when both orthographic representations and phonological representations are deliberately constructed with reduced componentiality, reduced nonword reading results at the end of training (P2 in Fig. X).

The second method of simulating developmental phonological dyslexia seeks to constrain the nature of the computational function that can be learnt between orthography and phonology, rather than the input or output representations themselves. In models with two routes connecting orthography and phonology, manipulations have focused on impairing the Direct route, which is better suited to learning relations between individual graphemes and phonemes (Ans et al, 1998; Zorzi, Houghton & Butterworth, 1998a). Initial removal of this route compromises the network's ability to learn a simple function relating orthography and phonology applicable to novel words, and so generalisation is reduced (Zorzi, Houghton & Butterworth, 1998b) (P3). Brown (1997) used another constraint on the computational function by employing several three-layer networks with reducing numbers of hidden units and comparing them when performance on regular and exception words was matched. Necessarily, this meant that the networks with fewer hidden units had experienced more training. Networks with fewer hidden units were unable to learn a

robust function linking orthography and phonology and so showed poor nonword reading (P4).

What can we deduce from this set of results? The variety of network architectures along with the variety of manipulations makes comparisons difficult – would a manipulation that succeeds in one architecture necessarily succeed in another? Before drawing some general conclusions, it will be useful to consider a recent systematic exploration of the range of developmental deficits that can be generated in these types of associative learning models, by Thomas & Karmiloff-Smith (2003a). Although the following simulations were carried out in the domain of inflectional morphology (specifically, English past tense formation), the simulations systematically examined developmental deficits along similar dimensions to those of reading models but in a single base architecture. The simulations therefore allow for convergent evidence on the implications of various sorts of computational deficits for an associative model attempting to acquire a partially regular domain.

In past tense formation, a phonological representation of each verb stem must be related to a phonological representation of the inflected past tense form. Along with each verb stem, word-specific (semantic) information is provided, so that the model can either learn to generate past tense forms based on the meaning or based on emerging regularities between phonological input and output forms. In the English past tense, there is a majority pattern (the add -ed rule, e.g., 'talk' => 'talked'), along with a minority of exceptions or irregulars ('think' => 'thought', 'go' => 'went', 'hit' => 'hit'). Generalisation to novel strings should extend the add –ed rule. A developmentally disordered model can be assessed on whether it shows (1) a differential impairment in learning irregular past tense forms (analogous to surface dyslexia); (2) an impairment in generalising the add –ed rule to novel strings

(analogous to phonological dyslexia); and, (3) an overall delay in acquiring both regular and exception past tense forms (analogous to delayed reading development). The analogy to the domain of reading breaks down in that, unlike in reading, both input and output representations are in the same modality, and therefore alterations to them must be yoked. In reading, orthography and phonology can in principle vary independently.[5]

Figure Y summarises the effects of a wide range of startstate manipulations to a normal developmental model along of these 3 dimensions (plotted from Table 1 in Thomas & Karmiloff-Smith, 2003a). The manipulations include changes in the initial network architecture, the initial numbers of hidden units, the processing unit activation function (i.e., the ability of processing units to make fine discriminations), processing noise, the learning algorithm, the learning rate, the presence or absence of word-specific information, and the structure of the phonological information (where reducing phonological 'similarity' is equivalent to reducing componentiality). Broadly, the manipulations fall under just two groupings: the manipulation either changes the computation problem (specified by input and output representations) or changes the computation power of the learning system (via changes in processing routes, processing resources, activation dynamics, or plasticity).

[5] The domains also diverge with regard to what constitutes regularity. In the reading, regularity is consistent with similarity (the same graphemes in different words are pronounced in the same way). In the English past tense, regularity ignores similarity (all regular verbs form their past tense by adding –ed to the verb root irrespective of any (dis)similarity between those verb roots).

Figure Y. The atypical developmental effects of altering various computational parameters in the startstate of a network that is learning a partially regular cognitive domain analogous to reading, past tense formation (Thomas & Karmiloff-Smith, 2003a). (S) => Delayed irregular acquisition is analogous to Surface developmental dyslexia. (P) => Reduced generalisation to novel strings is analogous to phonological developmental dyslexia.

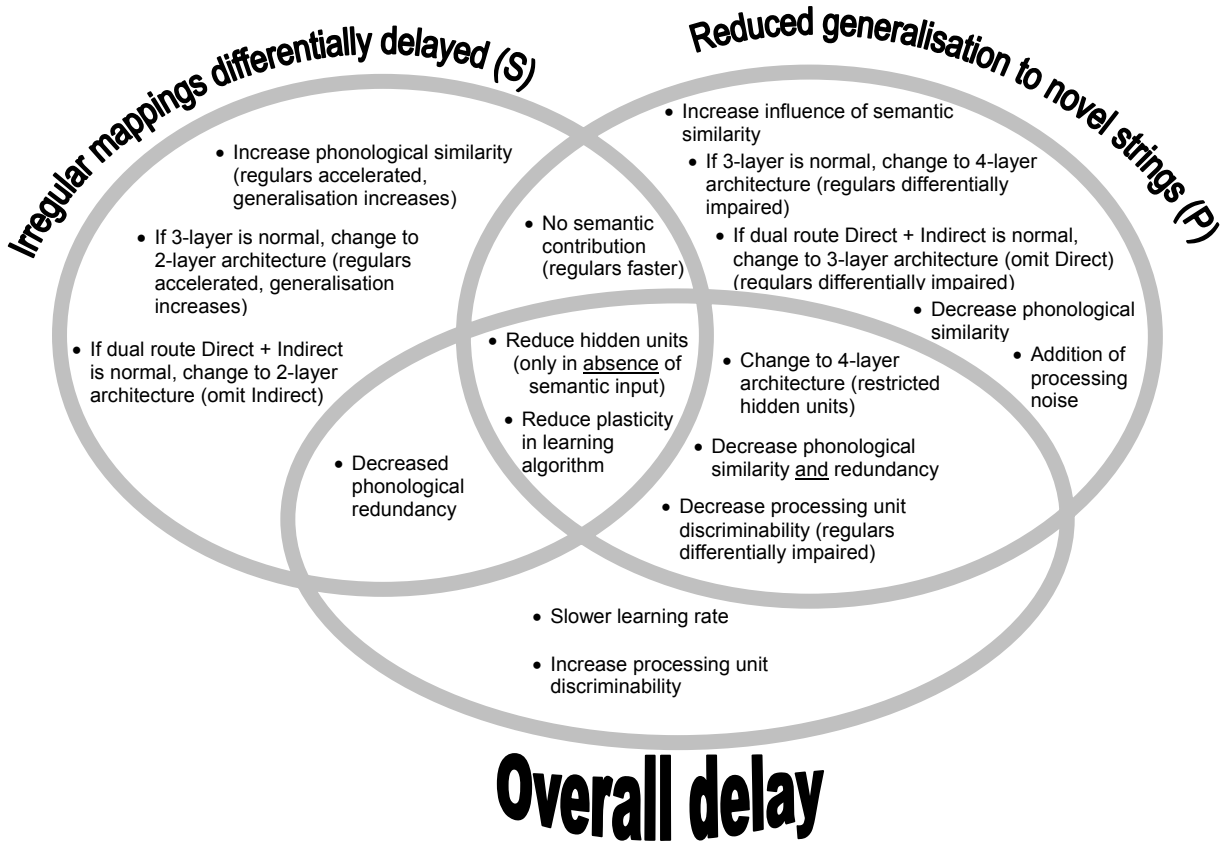


Figure Y replicates and expands on the pattern of results found in the models of reading acquisition. There are five main conclusions. (1) There are multiple ways of simulating each of the three deficits. Multiple causality of developmental deficits appears to be a strong prediction of both reading and past tense models. (2) Some manipulations produce unique effects on only one dimension, whilst other manipulations produce effects on two or all three dimensions, allowing both 'pure' and 'mixed' patterns of errors. (3) The presence of word-specific information such as meaning, aids the acquisition of exception forms. So too does the presence of a processing route that permits a larger granularity of processing. Initial deficits to either resource impair 'exception' acquisition. (4) The past tense simulations suggest that one finding from the reading models – that reduced numbers of hidden units particularly hurts exception mappings – may be an artefact of using models without an implemented semantic route. In the past tense model, the presence of semantic information allowed the system to overcome the differential effect of hidden unit numbers on exception mappings. In a reading model with a semantic route, particularly under assumptions of division of labour, reduced resources in the orthography to phonology network would likely shift exception processing into the semantic route and allow compensation to recovery.[6] (5) Changes in generalisation to novel forms can be disrupted by reduced componentiality / similarity of input representations and of output representations, but also by a range of other factors that alter the mapping function that the system can learn. Note, too, that concentration on a word-specific or semantic route also impairs generalisation, since the relevant regularities are not encoded in the dimension of meaning (for reading, they are

[6] albeit in a mildly atypical reading system that would demonstrate exaggerated effects of semantic variables like imageability on the reading of exception words.

encoded in a systematic orthography-phonology relationship; for past tense, in a systematic phonology(stem)-phonology(past tense) relationship).

Sensitive periods in reading development

Two other computational models of reading are relevant to the neuroconstructivist principles identified in this book. First, in our discussion of the normal acquisition of reading, we saw the theoretical claim that the onset of literacy serves to restructure pre-existing phonological representations, enforcing a finer level of granularity. Harm and Seidenberg (1999) were able to investigate to the possible nature of this process via simulation work, comparing the structure of phonological representations with and without the influence of orthography. They demonstrated improved segmentation of phonological representations following literacy training, along with sharper representations of rhymes, and a divergence in the phonological representation of rhyming words that had different spellings (e.g., 'hair' and 'bear'). The model illustrates the interactive, activity dependent nature of a processing system required to link multiple modality-specific representations in driving behaviour.

Second, Harm, McCandliss and Seidenberg (in press) used the Harm and Seidenberg (1999) model under conditions of simulated phonological dyslexia, and evaluated the extent to which there were sensitive period effects in remediating the deficit. The dyslexic model was impaired in its initial representations of phonology, as per the Harm and Seidenberg model. The phonological impairment was then alleviated at different points during reading instruction to determine whether this led to improved reading. Harm et al. focused on the sensitivity of this improvement to the time at which the intervention was applied. In this case, intervention was simulated simply by removing the noise / architectural constraints impairing phonological

development, as a sort of miracle cure. In a second set of simulations, Harm et al. investigated the efficacy of an existing intervention programme for dyslexic children on a model that retained its atypical constraints. Harm et al. (in press) found that in their model, interventions targeting phonological representations had a potential for success in alleviating reading difficulties but only if they were introduced extremely early in learning. Repairing phonological representations once poor learning had become entrenched led to a much-reduced effect. As Harm et al. noted, this replicates data indicating that the degree to which phonological awareness training produces reading improvements critically depends on the time of the intervention with regard to the onset of literacy. Preschoolers show significantly greater benefits than kindergarten or primary school children in their subsequent reading abilities (Bus & Ijzendoorn, 1999). Thus the model demonstrates an increasing restriction of fate in the development of its representations, albeit into an atypical state that is insufficient to permit the subsequent normal acquisition of a behaviour based on those representations.

Why no compensation?

Computational models now allow us to address the 'Why No Compensation?' question for developmental dyslexia. There are two answers. First, the initial computational constraints of the nascent reading system may militate against compensation. In particular, the implemented models argue that structure-function correspondences exist in the system, so that particular structures (those biased to process particular granularity of mappings, those with access to word-specific information) may be particularly suited to acquiring parts of the reading domain. If these parts are initially restricted or disconnected, no other part may have the right (or

optimised) computational properties to replicate the function. Secondly, compensation may indeed occur. Other structures may attempt to take on the function of the compromised elements. However, they will not be able to compute the relevant functions as efficiently and, moreover, in attempting to compensate, they may interfere with the acquisition of their normal function. A clear example of compensation in computation modelling can be found in the dual-route simulations of Thomas & Karmiloff-Smith (2002a, Figure 6, p. 746), and in the human case, in the imaging data discussed by Casey, Thomas, & McCandliss (1999).

The crucial point on the prevalence of compensation is that the atypically developing system is defined as disordered only if its compensatory processes have been unsuccessful. In empirical studies, disordered participant groups will not include cases where compensation has been successful, unless there is an independent (e.g., genetic) basis on which the disorder can be diagnosed.

The Neuroconstructivist Principles applied to the example of dyslexia

Developmental dyslexia illustrates the neuroconstructivist principles in the following way. Reading is a specialisation of a more general system, driven by exposure to a particular environment and culture. The structure of reading is likely to have interacted historically with the constraints of the general system that is recruited, ensuring that scripts are learnable. The location of the emergent reading system in the healthy brain involves interactions between multiple areas, determined by which areas are supplied with the appropriate information and have suitable (often more domain-general) computational constraints required of the task. For instance, the visual word form area arises in a region equipped to resolve the appropriate level of visual detail

and compute the appropriate translation invariances. In dyslexia, there may be an absence of emergent specialisation, although whether this is a side effect of a lack of expertise or the cause of subsequent deficits is currently unclear. Different partial representations interact across development, for example as illustrated by the restructuring of pre-existing phonological representations at the onset of literacy.

The disorder of developmental dyslexia has a genetic component but it is likely that the genetic mutation(s) that causes dyslexia also generate anomalies beyond the nascent reading system, both at a neural and cognitive level. However, there is still debate as to the appropriate causal model for these wider anomalies, as well as on the relationship between normal individual variation and atypical development. Importantly, expression of the disorder depends in part on the nature of the reading environment to which the atypical system is exposed. Phonological dyslexia may represent a poorly developed system for representing spoken speech sounds present prior to literacy across languages, but marked reading deficits only emerge in a subset of the languages that have complex orthography-to-phonology mappings.

Computational modelling has illustrated the importance of evaluating the impact of a clearly specified developmental process in producing the behavioural deficits shown in a disorder. Although there is some way to go to ensure we are capturing the right development process in these models, these models nevertheless represent significant progression from notional extensions of static models to developmental deficits, extensions that fail to specify any mechanisms of change. Models have generated insight into division of labour (through competition) in multiple component systems, into inter-modality interactions across development, into

sensitive periods and progressive restriction of fate, and into the implications of multiple causality in behaviourally defined disorders.

Multiple causality, however, was not unbounded in the models. Broadly, reductions in the similarity or componentiality of phonological / orthographic representations (separately or together) led to reductions in generalisation to novel stimuli, while attenuation of the availability of word-specific or semantic information differentially affected exception word performance. But in addition, various changes to the nature of the function that the architecture could compute had overlapping effects. These findings might encourage one to identify signs of convergence between the modelling of dyslexia and empirical brain imaging data. Imaging work also appears to implicate phonological processing regions and visual processing regions, along with the connectivity between areas representing visual object identity and phonology. However, additional areas have been implicated by the imaging data with no apparent role in the models (e.g., the cerebellum).

Finally, computational models allowed us give a clearer answer to the key question in the study of developmental deficits – why, unlike in early acquired focal brain damage in healthy children, is there no compensation to recovery across development? The answer is that (1) there is compensation, which may produce (perhaps quantitatively, perhaps qualitatively) different cognitive systems; but (2) compensation is unsuccessful in achieving recovery. The reason it is unsuccessful can be found in a consideration of the computational constraints, typical and atypical, that shape the developmental process when the cognitive system is exposed to a particular – and in this case, cultural specified – environment.

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