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Discussion forum

Dyslexia, dysgraphia, procedural learning and the cerebellum

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1. Introduction

In this review we focus on the developmental disorders of dyslexia (a disorder of reading) and dysgraphia (a disorder of writing), considering their commonalities and differences with a view to reflecting on the theoretical implications. Interest in dysgraphia was stimulated by the distinction between phonological and surface dyslexia (Castles and Coltheart, 1993), which claimed that orthographic problems (spelling) were separable from phonological reading problems. While this distinction has received mixed support (Snowling et al., 1996; Stanovich et al., 1997) it led to a fruitful analysis not only of the underlying causes of orthographic difficulties, but also to the widespread recognition of developmental difficulties in handwriting control (Deuel, 1995; Manis et al., 1996; Sprenger-Charolles et al., 2000). The result of this theoretical and empirical progress is that there are two usages of the term dysgraphia. One takes dysgraphia to refer to errors of writing that are analogous to errors in reading (e.g., surface, phonological or deep dysgraphia corresponding to surface, phonological and deep dyslexia), the other relating to difficulties in handwriting control. Furthermore, despite these attempts at differentiation, there remains some controversy in the literature as to whether motor difficulties in handwriting should be subsumed under the label dyslexia.

This review attempts to tease out the different strands of theoretical research underlying these confusions by analysing explanations of dyslexia and dysgraphia at the cognitive level and the brain level, considering both cortical and sub-cortical systems. First we outline theoretical approaches to developmental dyslexia, introducing causal explanations at the cognitive level, followed by an outline of recent developments in research into motor difficulties in handwriting. We then note the prevalence of comorbidities between developmental disorders, suggesting that this presents both a challenge and a potential stimulus for the disciplines. We then investigate a brain level causal explanation for dyslexia in terms of cerebellar deficit, because it provides a potential explanation of the co-existence of motor skill deficits and phonological deficits in dyslexia. The framework has strengths, but was also strongly criticised by theorists who advocated cortical foci of deficit. A promising further framework that may integrate cortical and sub-cortical accounts and provides a natural explanation for heterogeneity and comorbidity is that of neural systems and procedural learning (Nicolson and Fawcett, 2007). We extend this framework by applying it to dysgraphia, and conclude by arguing that the neural systems level of explanation provides a fruitful unifying framework for the developmental disabilities.

2. Developmental dyslexia

There is still considerable debate over the diagnosis of developmental dyslexia, but the traditional definition is ''a disorder in children who, despite conventional classroom experience, fail to attain the language skills of reading, writing and spelling commensurate with their intellectual abilities'' (World Federation of Neurology, 1968). It is known to be one of the most common of the learning disabilities with a prevalence of at least 4%. Prevalence may rise to as high as 10% or more if overlaps with other disorders such as specific language impairment, attention deficit, and developmental coordination disorder (DCD) are taken into account (Shaywitz et al., 1990; Hill, 2001; Badian, 1984b; Bishop and Snowling, 2004).

Traditionally, it had been considered that dyslexic children showed a form of 'minimal brain dysfunction' associated with a general problem in learning. However, the demonstration

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(Vellutino, 1979) that dyslexia reflected a verbal deficit, specific to language, set the scene for the hugely influential phonological deficit framework for explanation and support (Bradley and Bryant, 1983; Snowling, 1987; Stanovich, 1988b). This framework provided an explanation of the behavioural symptoms (poor reading) in terms of the cognitive level concept of weak phonology, illustrating the value of distinguishing three levels of theoretical explanation: behavioural, cognitive and biological (Morton and Frith, 1995). It is now generally accepted that phonological deficits in dyslexia tend to co-occur with other cognitive level deficits including speed of naming (Wolf and Bowers, 1999). Following extensive discussions, a more recent definition is "*Dyslexia is a specific learning disability that is neurological in origin. It is characterised by difficulties with accurate and/or fluent word recognition and by poor spelling and decoding abilities. These difficulties typically result from a deficit in the phonological component of language that is often unexpected in relation to other cognitive abilities and the provision of effective classroom instruction*" (IDA, 2002). It is noteworthy that the reading and spelling problems have been retained, but that the handwriting problems are no longer included in this definition (see Lyon et al., 2003 for the theoretical justification in terms of phonological deficit). As a consequence, writing problems in developmental dyslexia remain under-recognised and under-treated (Berninger et al., 2008).

3. Cognitive level explanations of dyslexia

There is considerable heterogeneity in the skills of dyslexic children, as might be expected given the very large numbers involved. A key strength of the phonological deficit hypothesis is that almost all children with dyslexia do show a core phonological problem, with variable secondary problems (Stanovich, 1988a). Nonetheless, it is important to attempt to explain secondary problems also. Arguably the broadest cognitive level description of the general type of performance difficulty in children with dyslexia is that they have difficulties when required to undertake fast, fluent, overlearned skills, or novel skills that involve the blending of two actions. Naturally this description applies in the literacy domain, with lack of automaticity in reading often cited as one of the key problems (Stanovich, 1988a; Wimmer, 1993), and with evidence that dyslexic children require twice as long tachistoscopic presentation of a word as children matched for reading age (Yap and van der Leij, 1993). It also seems to hold, at least for subgroups of dyslexic children, in cognitive non-literacy skills such as mathematics (Ackerman and Dykman, 1995), in naming speed (Denckla and Rudel, 1976; Wolf and Bowers, 1999), in general speed of information processing (Nicolson and Fawcett, 1994a), and in motor skills (Fawcett and Nicolson, 1995; Rudel, 1985; Wolff et al., 1990).

In the light of the establishment of a wider range of difficulties, it is not surprising that two major theories expanded significantly on the phonological deficit hypothesis. The double deficit hypothesis (Wolf and Bowers, 1999) claims that dyslexia is characterised by a deficit not only in phonological processing but also in terms of speed of naming. Cognitive level explanations suggest diagnostic tests and can inspire remediation strategies but cannot determine biological causes, in that a range of brain mechanisms could lead to problems in speed, phonology and reading. Consequently, explanations moved to biological level accounts, with an influential approach being the magnocellular deficit account (Eden et al., 1996; Stein and Walsh, 1997; Tallal et al., 1993), which attributes the difficulties to the magnocellular visual or auditory sensory processing system.

The phonological and magnocellular deficit hypotheses have been well described in the literature and there is no scope within this paper to consider them further. A valuable overview is provided in Demonet et al. (2004); a wide-ranging account centred on phonological deficits is given in Vellutino et al. (2004); and recent reviews of the magnocellular deficit hypothesis are provided in Beaton (2004), Stuart et al. (2006) and Boden and Giaschi (2007).

4. Developmental dysgraphia

As noted earlier, dysgraphia is characterised by difficulties in writing. The diagnostic and statistical manual of mental disorders (DSM-IV) definitions dissociated learning disabilities into three categories: reading disability, mathematical disability and 'disorder of written expression', together with a fourth category, 'learning disability not otherwise specified'. Dysgraphia as the disorder of written expression is characterised by "*writing skills (that) …are substantially below those expected given the person's chronological age, measured intelligence, and age-appropriate education*" (American Psychiatric Association, 1994). There is a lack of clarity as to whether writing refers only to the motor skill of writing (which is the traditional interpretation) or whether it also includes the orthographic skill of spelling.

Studies of dysgraphia are sparse, especially if single case studies are discounted. Smits Engelsman and Van Galen (1997) investigated quality of motor control in groups of children with dysgraphia and control children. They concluded that dysgraphia was associated with poor motor control, reflecting greater 'noise' in movement production rather than poor letter knowledge, and that this impairment was not outgrown in the following year. Mather (2003) distinguished between adolescents with good reading and poor spelling (termed dysgraphia), poor reading and poor spelling (termed dyslexia) and control adolescents. Both groups of poor spellers showed a specific deficit under dual task conditions when having to tap with their right hand and judge line orientation at the same time. The author interpreted the findings as reflecting a left hemisphere processing limitation. A recent study (Adi-Japha et al., 2007) assessed groups of boys with attention deficit hyperactivity disorder (ADHD) and writing difficulties but normal reading (termed dysgraphia) with control children. The authors concluded that the boys with (ADHD and) dysgraphia suffered primarily from motor planning errors rather than linguistic impairment. A small study (Ben-Pazi et al., 2007) of tapping ability and variability in a group of children indicated that poor quality handwriting was associated with abnormal tapping rhythm but that not all children with poor handwriting suffered from this 'dysrhythmia'. This finding provides an interesting link to cerebellar performance, which is discussed below.

In view of this apparent incidence of motor planning errors in dysgraphia, and the suggested overlaps between dyslexia, dysgraphia, ADHD and DCD), it is important to review first the incidence of (and explanations for) motor skill deficits in dyslexia and secondly, the evidence relating to comorbidity in the developmental disabilities. These analyses provide the framework for an integrative theoretical account of these findings.

5. Motor skill and dyslexia

From the first studies of dyslexia, there has been continuing evidence that mild clumsiness is associated with dyslexia. In a review of Orton's writings, Geschwind (1982) noted "… *He pointed out the frequency of clumsiness in dyslexics. Although others have commented on this, it still remains a mysterious and not adequately studied problem. It is all the more mysterious in view of the fact that many of these clumsy children go on to successes in areas in which high degrees of manual dexterity are absolutely necessary*."

There have been many subsequent anecdotal reports of sub-clinical motor difficulties in dyslexia, which when taken in conjunction with experimental evidence is suggestive of motor skill deficits. Augur (1985) documents several, including swimming and riding a bike. Data from the British Births cohort examined the skills of 12,905 children longitudinally (Haslum, 1989), identifying two motor skills tasks at age 10 which were significantly associated with dyslexia. These were failure to throw a ball up, clap several times and catch the ball and also failure to walk backwards in a straight line for six steps. Deficits in fine motor skills have also been identified, in terms of the characteristically poor handwriting (Benton, 1978; Miles, 1983), and copying in young children (Badian, 1984a; Rudel, 1985), coupled with difficulty in tying shoelaces (Miles, 1983).

For many years there has been evidence for 'soft neurological signs' in the motor skill deficits noted in dyslexia, including deficits in speed of tapping, rapid successive finger opposition, heel–toe placement and accuracy in copying (Denckla, 1985; Rudel, 1985). This led Denckla to argue for a 'non-specific developmental awkwardness', reflected in poor coordination even in relatively athletic dyslexic children, which is normally outgrown by puberty (Rudel, 1985). Denckla (1985) notes particular impairments in acquiring new skills, followed by normal performance once acquired: "*the part of the 'motor analyzer' that is dependent on the left hemisphere and has been found to be important for timed, sequential movements is deficient in the first decade of life in this group of children whom we call dyslexic.*"

The major cognitive level explanatory framework for dyslexia that predicts motor skill deficits is the automatisation deficit hypothesis (Nicolson and Fawcett, 1990). This proposed that dyslexic children have difficulties making skills automatic (so that one no longer needs to think how to do the skill). Automaticity develops from long practice under consistent conditions, and underpins almost all of our highly-practised skills from speech to walking to arithmetic. Nicolson and Fawcett and their colleagues have maintained a strong interest in the causes and specificity of learning problems, with representative findings indicating that problems arise in most speeded actions (Nicolson and Fawcett, 1994b), in motor-sequence learning (Nicolson et al., 1999), in eye blink conditioning (Nicolson et al., 2002) and in overall learning rates for procedural skills (Nicolson and Fawcett, 2000).

The claim of motor skill deficits in dyslexia has, however, been hotly disputed. This is due in part to the further fractionation of dyslexia into dyslexia as a phonological deficit, and dyspraxia/DCD as a motor skill problem, with a substantial overlap between the characteristics of the two disorders (Kirby et al., 2008). Nevertheless, those researchers who have investigated motor skills in dyslexia have found evidence of impairment (e.g., Chaix et al., 2007; Iversen et al., 2005) with significant links between slow motor development and both language and reading speed deficits in children at familial risk for dyslexia (Viholainen et al., 2006). A particular issue has been the presence or otherwise of balance deficits. Nicolson and Fawcett have consistently argued that the majority of children with dyslexia show balance problems if they are assessed using sensitive measures. This perspective has been supported by a range of studies (Stoodley et al., 2005; Nicolson and Fawcett, 1990, 1994a; Fawcett and Nicolson, 1999; Yap and van der Leij, 1994; Moe-Nilssen et al., 2003), though the findings are dependent on the age of the participants, the balance task, and the sensitivity of the analyses (Needle et al., 2006). A meta-analysis of 17 studies (Rochelle and Talcott, 2006) concluded that balance deficits were indeed associated with dyslexia, but were probably not associated directly with reading problems.

Interestingly, however, several research groups attribute motor and/or balance difficulties to comorbidity with attention deficit (Denckla et al., 1985; Ramus et al., 2003; Wimmer et al., 1999; Raberger and Wimmer, 2003; Rochelle et al., 2009). For these theorists, the lack of a direct link to reading difficulties is a major issue. Several studies have certainly established that motor skill problems are not significantly associated with reading difficulties over and above phonological problems (White et al., 2006). However, as we shall show, secondary symptoms such as motor skill problems, and comorbidities between disorders, are of major significance when attempting to identify the underlying neural systems that may be impaired. We therefore turn to brain level accounts of dyslexia, focusing initially on our cerebellar deficit framework. The magnocellular deficit account mentioned above is a further influential approach.

6. Brain level explanations for dyslexia

Understanding of brain processes has been transformed by functional imaging, with a major outcome being the discovery (Ackermann et al., 1999; Desmond and Fiez, 1998; Leiner et al., 1989) that the cerebellum plays a key role in linguistic and cognitive skills in addition to its well-established role in motor skill and coordination. See Ito (2008) for a recent review of the evidence. The established link between cerebellum and automaticity, and the emerging link between cerebellum and language therefore made it natural to propose and test the cerebellar deficit theory of dyslexia (Nicolson et al., 1995, 2001). This provided a principled account of the cognitive level

problems in phonology, working memory and speed, in terms of the underlying brain processes.

Subsequent research on the functional role of the cerebellum has provided support for the cerebellar deficit framework (Nicolson and Fawcett, 2005), with developments in cognitive neuroscience confirming two-way anatomical and functional connectivity of the cerebellum with frontal cortex (Kelly and Strick, 2003; Middleton and Strick, 2001) and strongly supporting the role of the cerebellum in language-related tasks (Justus and Ivry, 2001; Marien et al., 2001) as well as in learning complex cognitive/motor skills such as tool use (Imamizu et al., 2003; Miall et al., 2000). Furthermore, imaging research has confirmed that the cerebellum is indeed active in reading (Fulbright et al., 1999; Turkeltaub et al., 2002) and a recent imaging study on rhyming (Booth et al., 2007) established that the cerebellum has reciprocal functional connections with both left inferior frontal gyrus and left lateral temporal cortex. Most directly, studies have established further evidence of functional and anatomical abnormality of the cerebellum in dyslexia (Leonard et al., 2002; Rae et al., 2002; Eckert et al., 2003; Vicari et al., 2003; Pernet et al., 2009). For independent reviews see Demonet et al. (2004), Habib (2004), Lozano et al. (2003) and Vlachos et al. (2007). These findings present compelling evidence in support of cerebellar anomalies in dyslexia.

Following the cerebellar deficit theory, we developed (Nicolson et al., 2001) an 'ontogenetic' framework that attempted to explain how cerebellar differences at birth might lead to the range of difficulties suffered by children with dyslexia (Fig. 1). It may be seen that the framework provides an outline of how a basic impairment in the cerebellum can lead to problems in our highest level cognitive skill – reading – 6 years later. Detailed discussion of how this framework provides a principled explanation of all three cognitive level theories (phonological deficit, speed deficit and automaticity deficit) is provided in Nicolson and Fawcett (2005, 2008).

For our purposes here, however, the important issue is the heterogeneity implicit within the framework. The framework accounts for the three criterial difficulties for dyslexia from three different 'routes'. The writing problem is attributed to motor skill difficulties. The initial reading problems arise primarily from problems in phonology. The spelling problems arise primarily from problems in skill automatisation, as do later problems in reading fluency. Of course, given the strong interdependence between these three skills in the development of literacy (and in the school environment), problems in any one skill may lead to problems in all three, with particularly strong interdependence between reading and spelling developments (Frith, 1986), and the impact of effortful handwriting on spelling.

A key point is that the regions of the cerebellum – and the neural systems involved – are different for each of the three routes. Consequently, an individual may have impairment in one, two or all three routes. The cerebellar deficit hypothesis claims only that the language-related regions of the cerebellum are affected in dyslexia. These are generally considered to be Lobule VI and VIIB in the neocerebellum – well away from the motor and balance regions in the cerebellum – though there is also some representation in the cerebellar vermis (Desmond and Fiez, 1998). Other cerebellar regions may also be affected, but this is not necessary. Consequently, associated difficulties in skills such as handwriting may, but need not, arise.

Despite the extensive converging evidence described above many influential researchers remain convinced that reading difficulties arise from abnormalities in frontal or parietal cortex, and the cerebellar account has been hotly disputed by phonological deficit theorists (Vellutino et al., 2004). For a balanced evaluation see Beaton (2002). A series of studies has suggested strongly that, although a high proportion of dyslexic children and adults do show both phonological and motor difficulties, as might be expected given the cerebellar deficit framework, approaching half the dyslexic children do not (Ramus et al., 2003; White et al., 2006). Furthermore, several theorists have suggested that in fact the cerebellum may be functioning normally, but is receiving impaired information from other brain regions, sensory or cortical, an issue colourfully described by Zeffiro and Eden (2001) as the 'innocent bystander problem'.

In view of the heterogeneity within dyslexia, the prevalence of motor skill problems, and the overlaps with ADHD, it is appropriate to highlight the extent of comorbidity between the developmental disabilities before continuing to an explanatory framework.

7. Comorbidities between the developmental disabilities

There is a considerable overlap between different developmental disorders, with an apparent 'comorbidity' between most (Bishop, 2002; Bishop et al., 2004; Bishop and Snowling, 2004; Fletcher et al., 1999; Gilger and Kaplan, 2001; Gillberg, 2003; Hill, 2001; Jongmans et al., 2003). Long-standing evidence for comorbidity derives from the literature on ADHD and dyslexia. School and clinic-based comorbidity rates for ADHD with dyslexia range from 25% to 40% (Semrud-Clikeman et al., 1992; Shaywitz et al., 1994; Willcutt and Pennington, 2000), with considerable variability deriving from differing inclusionary criteria. There are also clear links between dyslexia and DCD, both via links to articulatory control and to motor control. Both of these coordination difficulties might well be attributable to cerebellar dysfunction. Studies of cerebellar involvement in DCD are surprisingly sparse. Taking a sample of 31 young children diagnosed with DCD, O'Hare and Khalid (2002) established a high incidence of problems in phonology and (less surprisingly) in motor skills such as heel-to-toe walking, which they attributed to cerebellar dysfunction. A study of 7 children with DCD revealed abnormal adaptation to distortion of visual feedback on a computer drawing task (Kagerer et al., 2004), whereas a study of prism adaptation (Cantin et al., 2007) yielded equivocal results, with 6 of the 9 children showing some abnormality of adaptation but no significant between-group differences on any specific aspect of adaptation.

The relationship between ADHD and specific language impairment (SLI) is less clearly understood or researched, although children with SLI show phonological deficits, which are a core problem for children with dyslexia. An early study (Love and Thompson, 1988) claimed that 75% of children

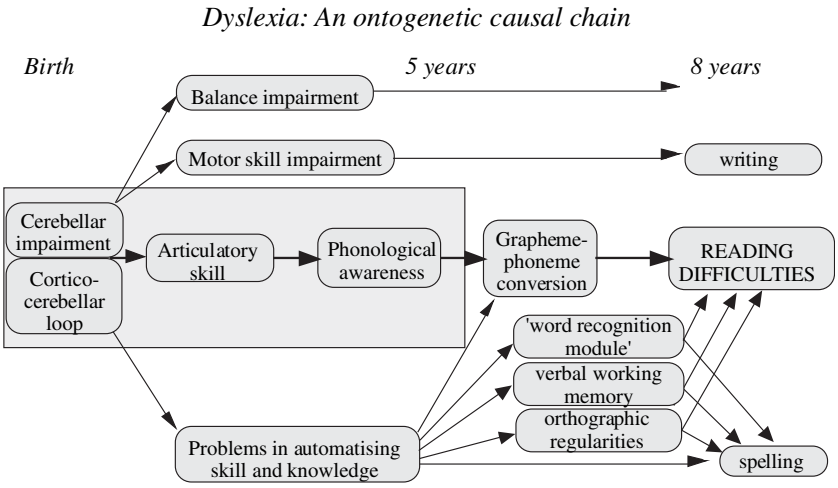

Fig. 1 – A hypothetical causal Chain. The horizontal axis represents both the passage of time (experience) and also the ways that difficulties with skill acquisition cause subsequent problems, leading to the known difficulties in reading, writing and spelling. The text provides a fuller explanation of the processes involved. Of particular interest is the progression highlighted as a central feature. Cerebellar abnormality at birth leads to mild motor and articulatory problems. Lack of articulatory fluency leads in turn to an impoverished representation of the phonological characteristics of speech, and thence to the well-established difficulties in phonological awareness at around 5 years that lead to subsequent problems in learning to read. Other routes outline the likely problems outside the phonological domain, and indicate that the difficulties in learning to read, spell and write may derive from a number of inter-dependent factors.

referred for language disorder also had ADHD, compared with only 16% with language disorder alone. Finally, reviving the concept of 'minimal brain dysfunction' from the 1970s, there appears to be a surprisingly high incidence of minor motor delays in ADHD, dyslexia and specific language impairment in addition to developmental coordination and the dyspraxias (Gillberg, 2003; Hill, 1998).

A study in Calgary (Kaplan et al., 2001) is particularly challenging. The authors studied a population-based sample of 179 children receiving special support, and established that the incidence of ADHD was 69%, the incidence of dyslexia was 64%, and the incidence of DCD was 17% (and that of oppositional defiant disorder was 23%). If a child met criteria for dyslexia, the chance of having at least one other disorder was 51.6%. If the child met criteria for ADHD, the chance of having at least one other disorder was 80.4%. Similar results were found recently in a large clinical sample of 886 children with normal range intelligence (Mayes and Calhoun, 2007). The authors concluded that attention, graphomotor, and speed weaknesses were likely to coexist, and that the majority of children with autism and with ADHD had weaknesses in all three areas. As Gilger and Kaplan (2001) argue, "*in developmental disorders comorbidity is the rule not the exception*".

One promising explanation at the genetic level for the overlap between developmental disorders is the 'generalist genes' hypothesis (Plomin and Kovas, 2005) which argues that one gene may have multiple potential effects via pleiotropy. Plomin and Kovas speculate (p. 200) that it is likely that a gene may affect several regions of the brain, which in turn affect various cognitive abilities. Consequently, genes that affect one learning disability are likely to affect another.

In our view, the genetic approach provides a valuable line of potential converging evidence in generating a theoretical understanding of the developmental disorders, but it cannot succeed alone. If the geneticists are to make progress, the cognitive neuroscientists and developmental theorists need to be able to provide high quality data. At the behavioural level, what is the phenotype that should be used? Reading is such a complex skill that a problem almost anywhere in the brain will lead to failure to acquire fluent reading. The same applies for spelling. A vague phenotype will necessarily lead to an impression of gene pleiotropy. The problem is exacerbated by difficulties in reaching a firm diagnosis when criteria for the different disorders in the DSM-IV are overlapping, with ADHD particularly difficult to pin down. At the brain level, we know that the different brain regions are multiply connected, with a series of major neural networks underpinning learning in at least four different domains (declarative, procedural, motor and sensory). If we are to make progress we need to undertake more systematic analyses that integrate insights from genetic, brain, cognitive and behavioural levels. Below we present an initial effort in this direction.

8. The neural systems framework

In a recent analysis (Nicolson and Fawcett, 2007) we have proposed a possible integration between the varied and apparently incompatible frameworks for dyslexia and other learning difficulties by undertaking an analysis of the neural systems underlying the different types of learning.

Five perspectives come together in this analysis. We started from our own analysis of learning, automaticity and the cerebellum in dyslexia, and the difficulties of disentangling the effects of impaired cerebellum from impaired components of the cortico-cerebellar circuits. The 'innocent bystander'

problem is eliminated if one considers as a whole the neural systems that act together in learning.

Second, we added to this the intriguing comorbidity between different developmental disorders that suggested that with current diagnostic methods primary symptoms of one disability were often secondary symptoms of another, and vice versa.

The third perspective is that of neural systems, starting with the long-standing distinction (Squire, 1987) between the declarative memory system (that is for learning facts, meanings and other consciously accessible information) and the procedural memory system (that is for skills, habits, and other not-consciously-accessible procedures).

The fourth perspective derived from the work of Doyon and Ungerleider and their colleagues in investigating the neural systems underlying the various stages in learning motor skills over the period from minutes to days (Doyon and Benali, 2005; Doyon et al., 2003; Doyon and Ungerleider, 2002). The authors proposed that there are two distinct motor learning circuits, a cortico-striatal system and a cortico-cerebellar system. The cortico-striatal system is particularly involved in learning sequences of movements, whereas the cortico-cerebellar system is particularly involved in adapting to environmental perturbations. However, the key point is that all three brain regions – motor cortex, basal ganglia and cerebellum (and also the frontal cortex for explicit skill monitoring in the early stages) are involved in the initial ('fast learning') stage of motor skill acquisition, whereas the roles of the cortico-striatal and cortico-cerebellar systems diverge as we approach the automatisation stage.

For us the crucial step forward was taken when Ullman (2004) highlighted the fact that in addition to the well-known procedural learning motor system (for motor skills such as handwriting), there is also a procedural learning system for language skills and habits, such as our implicit knowledge of language rules. It comprises the basal ganglia; frontal cortex, in particular Broca's area and pre-motor regions; parietal cortex; superior temporal cortex, and the cerebellum. The system has clear commonalities with the cortico-striatal and cortico-cerebellar-motor learning systems, the difference being that the language-based system interacts with the language-based regions of the frontal lobe, whereas the motor skill system interacts with primary motor cortex. Both systems include pre-motor regions. Normally, of course, these systems work together to provide optimal learning and performance – as in the case of spoken language, for example - but in the event of one system working sub-optimally, it is likely that the other will compensate (Ullman, 2004) by working harder.

While Doyon and Ungerleider derived their framework from studies of motor skill development, it seemed appropriate, given the similarities in procedural and declarative neural circuits for language skills, to extend their framework to include procedural language learning as well.

Combining the five disparate frameworks referred to above, we proposed that developmental dyslexia arises specifically from impaired performance of the 'procedural learning system for language', a neural system that includes prefrontal cortex around Broca's area, parietal cortex, and sub-cortical structures including the basal ganglia and cerebellum. A subset of dyslexic children will also show difficulties in the motor procedural learning system, but this is not a requirement for a diagnosis of dyslexia.

We find on the combined model of procedural learning, therefore, that impairment in any one brain region – cerebellum, motor cortex or basal ganglia – would lead to an impaired ability initially to acquire the skills associated with that region. This may lead to either a developmental delay or developmental deficit, dependent on the neural circuits involved and the degree of impairment. Impairment in any component of the procedural learning system would lead to some impairment in the initial acquisition of motor skill. While this deficit might be overcome subsequently, there would be a developmental delay in its acquisition. For those with impairment in the cortico-striatal circuit there would be long-term problems in motor-sequence activities (corresponding to a slight clumsiness). For those with abnormality in the cortico-cerebellar circuit there would be long-term problems in skills such as balance and adaptive timing.

The framework provides a novel and potentially fruitful integration of a range of perplexing problems which are hard to explain using less broad approaches. It also provides a clear clarity of linkage to learning and to intervention. For instance, if one child has good declarative learning but weak language-based procedural learning it is likely that the appropriate intervention will be qualitatively different from that for a child with the opposite profile. To our knowledge, no educational theorist or practitioner has investigated this possibility.

We addressed the various different learning disabilities within this framework, suggesting (in line with Ullman, 2004) that the majority could be described in terms of five neural systems and their interaction (Fig. 2).

It may be seen that we were able to attribute a range of learning disabilities to different branches of the 'tree' with generalised learning difficulties attributed to problems in the Declarative Learning System, and the remainder to problems in one branch or other of the procedural learning system. In particular, we allocated SLI to the striatal-language branch, DCD to the striatal-motor branch, and dyslexia to the cerebellar-language branch. We allocated ADHD (inattention subtype) to the same branch as dyslexia, but it might equally be allocated to a striatal branch, in that there is gathering evidence (Redgrave et al., 1999) that the basal ganglia are strongly involved in response selection and inhibition. We did not specify a disorder for the cerebellar-motor branch of the procedural learning system. Given that the cerebellar branch is likely to be involved in adaptation of movement parameters such as size of handwriting and in rhythmic coordination, it seems appropriate to allocate this branch to dysgraphia.

Of course this tree analysis considerably oversimplifies the situation. One method of accommodating the effects of putative magnocellular problems in dyslexia would be to introduce the further major neural systems, the parvocellular and magnocellular systems.

Lest one think that the framework is too vague to be testable, a recent study (Brookes et al., 2007) provides intriguing support. A group of participants with dyslexia and a group with DCD were compared with controls on their ability to adapt to the visual field displacement caused by prisms – a task thought to be cerebellar (Baizer et al., 1999). It turned out that 6 of the participants with DCD had comorbid dyslexia.

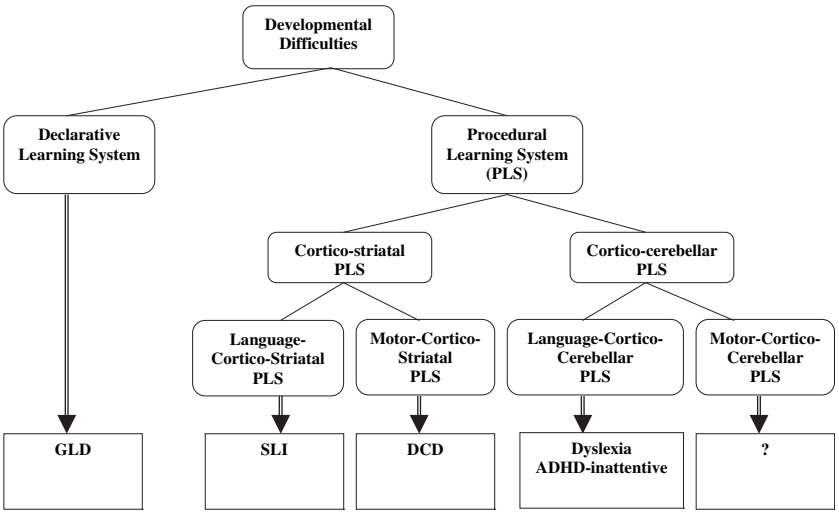

Fig. 2 – A neural systems typography for learning difficulties.

The dyslexic group showed significantly slower adaptation than the controls, with 10 out of 14 showing an individual impairment. Even stronger deficits obtained for the DCD group, with all 14 participants showing individual impairments, regardless of their reading abilities. The results therefore support the framework in several ways: in particular showing that a task designed to tap the function of the cerebellar procedural learning circuits can identify problems in its function, and that these problems transcend the DSM-IV classificatory system. On the other hand, according to the model, the children with DCD and no reading problems should not show cerebellar difficulties! It may nonetheless be that these children show comorbid cerebellar-motor procedural Learning problems, as in the dysgraphia branch of the tree. All the children in the dyslexic and dyspraxic groups showed mild to severe fluency problems on the dyslexia screening test (DST) 1 min writing test (Fawcett and Nicolson, 1996), but unfortunately we no longer have access to records of the quality of their handwriting performance.

The study of adaptation to prisms when throwing at a target referred to earlier (Cantin et al., 2007) is particularly interesting in this regard. The authors compared 9 children with DCD with age-matched controls. They correctly described their results as 'equivocal' in that one third of the children showed problems in immediate prism adaptation, a further third showed abnormal after-effects of prism removal, and one third performed within normal parameters, even though all showed abnormal throwing ability. Interpretation is hampered by considerable between-participant variability, by a relatively high age range between the participants, and lack of information about reading ability. Nonetheless, the heterogeneity established does lend support to the view that there is a range of potential underlying causes, and that these can be teased out by appropriate 'brain-based' assessments.

An additional key point, however, is that this tree structure should not be seen in terms of all-or-none branches. It is more appropriate to consider degrees of impairment. So, one might think of 'pure' dyslexia as having an overachieving Declarative Learning System at say 20% above normal, an underachieving Cerebellar-Language Procedural Learning System at say 70% of normal efficiency, and an underachieving Cerebellar-Motor Procedural Learning System at say 90% efficiency, whereas dysgraphia might be Declarative Learning System at 20% above normal, Cerebellar-Language Procedural Learning System at 90% and Cerebellar-Motor Procedural Learning System at 70% efficiency. A full analysis of the rationale for multiple deficits in developmental disorders is beyond the scope of this article, but it should be noted that the case has been made convincingly by Pennington (2006), though he does not consider the neural systems perspective.

9. Summary and conclusions

In summary, we have taken an extended tour through dyslexia and other learning disabilities, focusing on the commonalities and differences between dyslexia and dysgraphia. We have put forward the viewpoint that both of these reflect lack of automaticity at the cognitive level, attributable to impairment of procedural learning circuits involving the cerebellum at the neural circuits level, with 'pure' dysgraphia involving impairment of the cerebellar-motor circuit and 'pure' dyslexia involving impairment of the cerebellar-language circuit.

This framework provides natural twin explanations of the comorbidities between a variety of developmental disorders. First, the various procedural and declarative circuits are all involved in the early stages of skill learning, and therefore impairment in any one of them will lead to sub-optimal acquisition of a range of skills, with primary impairment in the skills specific to the impaired circuit, and secondary impairments in the other skills. Second, for disabilities attributable to abnormal brain development in gestation, it is likely that abnormalities will be relatively widespread. Indeed, studies of the effects of pre-natal and peri-natal lesions reveal that abnormalities can occur well away from the lesion site (Block et al., 2005). It is also worth noting that the protracted development of the cerebellum creates a special susceptibility to disruptions during embryogenesis (Wang and Zoghbi, 2001). Furthermore, developmental

studies of acquired damage to the cerebellum have highlighted the impact on motor function in children (Konczak and Timmann, 2007). Motor development and cognitive development have been shown to be fundamentally inter-related, leading to abnormalities in both the prefrontal cortex and the cerebellum in 'cognitive' developmental disorders (Diamond, 2000). The framework therefore explains the ubiquity of comorbidity in developmental disorders.

The framework also provides a strong case for abandoning the all-or-none approach to classification cogently criticised by Newell (1973). As Newell predicted, the initial distinctions, originally clearcut, have become points on a continuum following further investigation. This point has been emphasised in the dyslexia literature where it is now acknowledged that the combined effects of a range of risk factors and protective factors need to be considered when attempting to account for reading performance (Snowling et al., 2003) – see also Pennington (2006). This analysis must also be applied to the other developmental disabilities, where the implication of the framework is that several developmental disorders may share a similar set of risk factors and strengths. This suggests in turn that it may be more fruitful to characterise a given individual's profile of strengths and weaknesses, rather than the current approach of identifying the 'primary' weakness. If this was politics, it would involve moving from a 'winner takes all' system to a proportional representation system!

Possibly most important, the strong implication of the framework is that the neural systems level of description provides an under-explored classificatory system that complements, or may even replace, the current DSM-IV symptom-based diagnostic system. In our view this would lead to several significant advantages. First, emphasis on the underlying cause (at the neural systems level) rather than its behavioural manifestations is of immediate value in providing a common framework for understanding the developmental disorders; for investigating commonalities as well as differences; for linking to current conceptual frameworks in cognitive neuroscience; and arguably for providing a level of analysis that may be more fruitful for genetic investigations. Second, the emphasis on neural systems provides an important emphasis on the importance of considering the interplay between cortex and sub-cortex in scaffolding learning and expediting action. Third, the change of focus from differences in attainment to differences in learning ability and disability provides a potential methodology for bridging the chasm between neuroscience and education – the start of a discipline of pedagogical neuroscience (Fawcett and Nicolson, 2007). Fourth, the framework provides a set of immediate fruitful challenges; foremost among which is the development of a battery of tests designed to tease out the functionality of the many different neural learning systems.

It is important to conclude with a caveat. The neural systems approach is still in its infancy, and currently lacks the necessary empirical evidence with regard to the different developmental disabilities to provide solid foundations. As it is systematically tested, the framework structure should become clearer, but we recognise that at the present time further evidence is needed to support the ideas put forward. Nonetheless, the perspective provides a bridge between education, cognitive neuroscience and learning disorders. Development of the necessary tools to investigate and refine the framework is a worthy task for collaboration between geneticists, cognitive neuroscientists and learning disability theorists over the coming years.

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