A *TINS* debate – Hindbrain versus the forebrain: a case for cerebellar deficit in developmental dyslexia

Progressive improvement in reading and writing skills, through school and beyond, is something that many of us take for granted. However, for people suffering from dyslexia, these skills are not acquired in the usual manner. For many years it has been thought that brain differences in the cortical areas related to language are the likely source of the problems. However, it has recently been established that the problems associated with this syndrome go beyond reading-related problems: balance, motor skills and sensory processes can also be affected. An explanation for this multitude of seemingly disparate problems has proved elusive. Roderick Nicolson, Angela Fawcett and Paul Dean believe that a deficit in cerebellar performance might provide a complete explanation, and it is this argument, presented in the first article below, that forms the focus of this debate. But can cerebellar deficit explain all deficits experienced by dyslexics? Are those that experience cerebellar damage later in life similarly affected? Is the cerebellum the sole contributor to dyslexia?

Two pairs of experts in this field, Thomas Zeffiro and Guinevere Eden, and Richard B. Ivry and Timothy C. Justus, discuss these and other questions. It becomes clear during the debate that the acquisition of reading-related skills requires the co-ordination of many areas of the brain involved in visual, motor and cognitive activities, and that an increased understanding of dyslexia could provide insights far beyond the disorder itself. The conclusion to this debate is provided by Roderick Nicolson and his colleagues.

Developmental dyslexia: the cerebellar deficit hypothesis

Roderick I. Nicolson, Angela J. Fawcett and Paul Dean

Surprisingly, the problems faced by many dyslexic children are by no means confined to reading and spelling. There appears to be a general impairment in the ability to perform skills automatically, an ability thought to be dependent upon the cerebellum. Specific behavioural and neuroimaging tests reviewed here indicate that dyslexia is indeed associated with cerebellar impairment in about 80% of cases. We propose that disorders of cerebellar development can in fact cause the impairments in reading and writing characteristic of dyslexia, a view consistent with the recently appreciated role of the cerebellum in language-related skills. This proposal has implications for early remedial treatment.

Developmental dyslexia is traditionally defined[1] as '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'. Dyslexia researchers have focused on two alternative hypotheses: the phonological deficit account[2–4], which holds that the reading difficulties derive initially from problems in breaking spoken words down into their constituent sounds (syllables or phonemes), and the magnocellular deficit account, which holds that the reading problems derive from impaired sensory processing, caused by abnormal auditory[5] and/or visual[6,7] magnocellular pathways.

Unfortunately, in spite of extensive research, these approaches have failed to account for the full range of difficulties established for dyslexic children. It is therefore timely to present the case for our alternative hypothesis that the full range of deficits might be accounted for in terms of cerebellar deficit. There is no space here to survey the rich evidence relating to alternative hypotheses. We fully expect the commentators to present cogent alternative data and views. In preview, we claim that:

(1) The behavioural symptoms of dyslexia can be characterized as difficulties in skill automatisation[8,9] (the process by which, after long practice, skills become so fluent that they no longer need conscious control).

(2) The pattern of difficulties in cognitive, information processing and motor skills is predicted by the cerebellar deficit hypothesis[9–12].

(3) Dyslexic adults showing the above behavioural manifestations of cerebellar impairment also show direct neurobiological evidence of cerebellar impairment[13]. This is consistent with other evidence of cerebellar abnormalities in dyslexia[14].

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(4) It is possible to present an ontogenetic causal model for the development of the reading-related problems and other problems of dyslexic children, with the major causal factor being impaired implicit learning as a result of cerebellar abnormality[15].

Empirical evidence

Behavioural symptoms of dyslexia

All major theories make a reasonable attempt at explaining the major behavioural symptoms – reading, writing and spelling. Consequently, crucial tests often derive from domains outside literacy. In the studies mentioned below, the dyslexic subjects are defined in terms of: significant reading delay (at least 18 months); IQ of at least 90; without attention deficit hyperactive disorder (ADHD) or serious emotional problems. Control subjects were matched with the dyslexic subjects for age and IQ, and had no reading delay. In early work we assessed the 'profile' of difficulties of dyslexic children by testing a range of skills, within and outside the literacy domain. Interestingly, we established that the dyslexic children tested showed difficulties 'across the board', in information processing speed, memory, motor skill and balance, in addition to phonological and literacy skill[9,16]. This pattern was obtained not only for group data but also for the individuals within the group. In particular, taking three disparate tests – balance (while also undertaking a secondary task), phonemic segmentation (e.g. "say 'stake' without the 't'") and picture naming speed – 90% of the dyslexic children had 'marked impairment' (at least one SD below normal performance) on at least two of the tests. We concluded that the data supported our 'dyslexic automatization hypothesis' – that dyslexic children have difficulties automatizing skill, whether or not the skill is in the literacy domain[8].

Behavioural tests of cerebellar function

Problems in automatisation point to the cerebellum, which has traditionally been considered a motor area[17–20]. We have established extensive multi-disciplinary evidence directly consistent with the cerebellar deficit hypothesis. An influential study[21] established that patients with acute cerebellar damage show a characteristic dissociation between time estimation and loudness estimation, with a significant deficit only for the former. We established that the same dissociation occurred for our dyslexic panel[10]. Next, we reimplemented the classic clinical tests of 'cerebellar signs' – both dystonia and dyscoordination, described in Ref. 22, and applied them to our panels. The dyslexic children showed highly significant impairments on all the cerebellar tests, and significant impairment compared even with reading-age controls on 13 of the 14 tasks, with effect sizes equivalent to those found on the earlier literacy-related tests[11]. The study was subsequently replicated with a larger sample of dyslexic children taken from the whole cohort of 8–16-year-olds at a special school for dyslexic children[12]. A similar pattern of difficulties again occurred, with highly significant deficits on balance and muscle tone comparable in magnitude to their reading and spelling deficits, and greater than their deficits on segmentation and nonsense word repetition. Particularly noteworthy was that 51 of the 59 dyslexic children were markedly impaired on muscle tone.

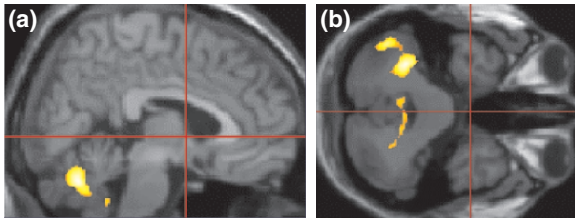

Fig. 1. Regions of significantly greater activation when learning a new sequence. Location of significant differences in activation ($P < 0.01$, corrected for multiple comparisons at $P < 0.05$) between dyslexic and control subjects for the comparisons of New sequence with Rest. The images are integrated sagittal and horizontal projections of the statistical parametric maps (SPMs), using an 8 mm smooth. Images produced by SPM96 (Wellcome Dept of Cognitive Neurology, 1996). The areas of significant difference are shown as light blobs superimposed on a T1-weighted magnetic resonance image normalized to the same standard stereotactic space. The axis lines on each image indicate horizontal and vertical planes that project through the position of the anterior commissure. Standard radiological convention is used [right hemisphere in (a)]. The figure shows the regions where the dyslexic group showed significantly less relative activation compared with controls. The only regions of significantly different relative activation are the right hemisphere of the cerebellum, together with the cerebellar vermis.

Direct tests of cerebellar function

The above tests of cerebellar function were necessarily indirect. In considering the design of a direct test, we wished to implement a functional imaging study. However, rather than select one in the reading-related domain (for which differences in performance affect interpretation of imaging data) we preferred to study a task outside the literacy domain in which there was clear evidence of strong cerebellar activation in normal subjects. Fortunately, a PET study[23] provided a perfect opportunity. Jenkins and colleagues had their subjects learn a sequence of eight button presses by trial and error using a four-key response board with one key per finger. They established clear increases in cerebellar activation (compared with rest), both when the subjects were executing a previously overlearned (automatic) sequence of presses and also when they were learning a new sequence of presses. We undertook a precise replication, using the oldest members (now adult) of our dyslexic and control panels. Compared with the control subjects, our dyslexic subjects showed significantly less cerebellar activation in the ipsilateral (right) hemisphere. Interestingly, similar results were obtained for both tasks – executing the previously overlearned sequence, and learning the new sequence (Fig. 1). Overall the dyslexic group showed barely any increase in activation in the right cerebellar hemisphere and

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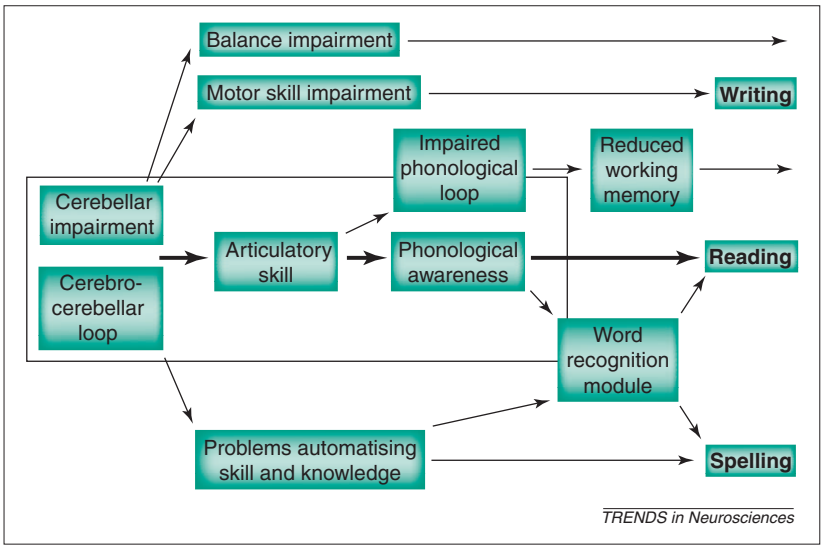

Fig. 2. A hypothetical causal chain. The abscissa represents both the passage of time (experience) and also the ways that difficulties with skill acquisition cause subsequent problems, leading to the known difficulties in reading, writing and spelling. The text provides a fuller explanation of the processes involved. Of particular interest is the progression highlighted as a central feature by the box. 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 subsequently to the well-established difficulties in phonological awareness at ~5 years that lead to subsequent problems in learning to read. Other routes outline the probable problems outside the phonological domain, and indicate that the difficulties in learning to read, spell and write might derive from a number of inter-dependent factors.

vermis (~10% of the controls). This PET study therefore confirmed that the behavioural cerebellar signs of these subjects did indeed reflect abnormal cerebellar function, and therefore lends weight to the above behavioural studies.

Converging direct evidence of cerebellar dysfunction is also provided by a recent study[14] of metabolic abnormalities in dyslexic men. Rae and colleagues obtained localized proton magnetic resonance spectra bilaterally from the temporo-parietal cortex and cerebellum of 14 dyslexic men and 15 control men of similar age. Bilateral MR spectroscopy indicated significant differences in the ratio of choline-containing compounds to N-acetylaspartate (NA) in the left temporo-parietal lobe and the right cerebellum, together with lateralization differences in the cerebellum of the dyslexic men but not the controls. The authors concluded that 'These differences provide direct evidence of the involvement of the cerebellum in dyslexic dysfunction'.

Toward a causal explanation

The above analyses indicate a correlation between dyslexia and abnormal cerebellar function in ~80% of the dyslexic children tested. A key question that arises is whether cerebellar impairment can provide a causal explanation of the development of the specific cognitive difficulties of dyslexic children.

Figure 2 (adapted from Ref. 15) outlines one hypothetical ontogenetic causal chain, linking cerebellar problems, phonological difficulties and eventual reading problems. Note that the three criterial difficulties of writing, reading and spelling are all accounted for in different ways. It might be useful to distinguish between direct and indirect cerebellar causation. Cerebellar deficit provides a natural, direct, explanation of the execrable quality of handwriting frequently shown by dyslexic children. Handwriting, of course, is a motor skill that requires precise timing and coordination of diverse muscle groups. Literacy difficulties arise from several routes. The central route is highlighted. If an infant has a cerebellar impairment, initial direct manifestations will be a mild motor difficulty – the infant might be slower to sit up and to walk – and crucially, the direct effect on articulation would suggest that the infant might be slower to start babbling, and, later, talking. Even after speech and walking emerge, one might expect that the skills would be less fluent, less 'dextrous', in infants with cerebellar impairment. If articulation is less fluent than normal, then one indirect effect is that it takes up more conscious resources, leaving fewer resources to process the ensuing sensory feedback. An additional indirect effect is that reduced articulation speed leads to reduced effective 'working memory' as reflected in the 'phonological loop'[24]. This, in turn leads to difficulties in language acquisition[25]. Furthermore, reduced quality of articulatory representation might lead directly to impaired sensitivity to onset, rime, and the phonemic structure of language[26] – in short, one would expect early deficits in phonological awareness. Cerebellar impairment would therefore be predicted to cause, by direct and indirect means, the 'phonological core deficit'[27] that has proved such a fruitful explanatory framework for many aspects of dyslexia. For spelling, the third criterial skill, problems arise from several indirect routes – over-effort in reading, poor phonological awareness and difficulties in automatising skills.

It is valuable to consider how the above framework relates to alternative theoretical formulations for dyslexia. Note that there is a qualitative difference between the three hypotheses discussed above: the magnocellular deficit and cerebellar deficit hypotheses are both phrased in terms of an underlying neural substrate – the 'biological' level, whereas the phonological deficit hypothesis is framed in terms of a non-biological theoretical mechanism – the 'cognitive' level[28].

At the biological level, cerebellar deficit is an alternative, or perhaps parallel, mechanism to magnocellular abnormality. It is possible that dyslexic children might show either or both of these abnormalities. This remains an open research issue. From the behavioural and functional data that we have established, it would appear probable that the majority of dyslexic children suffer from abnormal cerebellar function.

At the cognitive level of explanation we have outlined how cerebellar deficit accounts naturally for phonological deficit and for automatisation deficit. It also provides a natural explanation of the more recent

'double deficit' hypothesis[29]. This is based on the established difficulties that dyslexic children have on 'rapid automatised naming' tasks[30], in which the child has to name as rapidly as possible a page full of common pictures or standard colours, and suggests that dyslexia is characterized by a deficit not only in phonological skills but also in naming speed (reflecting a lower speed of processing). Naming speed difficulties are precisely those predicted by the cerebellar deficit hypothesis, given its established role in speech, inner speech and speeded processing. Consequently, all three cognitive level hypotheses appear to be directly consistent with, and indeed, subsumed by, the cerebellar deficit hypothesis.

Summary and conclusions

In summary, we have argued the following points.

(1) A high percentage of diagnosed dyslexic children show behavioural evidence of abnormal cerebellar function – in skill automatisation, in time estimation, balance and the classic cerebellar signs of dystonia.

(2) In the dyslexic adults tested, the behavioural evidence of cerebellar abnormality was accompanied by direct evidence of abnormal cerebellar function, both for executing an 'automatic' sequence of button presses and for learning a new sequence of button presses.

(3) The difficulties in skill automatisation correspond directly to the traditional role of the cerebellum[31,32]. The hypothesised role of the cerebellum in articulation-related cognitive skills is directly consistent with recent evidence of its role in speech-related cognitive tasks.

(4) Finally, we provided a plausible, albeit speculative, causal analysis that explains the difficulties in reading, writing and spelling within a consistent and coherent developmental framework. Furthermore, two of the major alternative cognitive-level explanations of dyslexia, namely the phonological deficit hypothesis and the double-deficit hypothesis, might be integrated naturally within this framework.

We would like to conclude by emphasizing that the cerebellar deficit hypothesis should be seen as speculative at this stage, because the dyslexia-related data provided are mostly from small scale studies in our own laboratory. One important research requirement therefore is to establish the extent to which other groups of dyslexic children show 'cerebellar signs'. The approach raises many further theoretical questions: are there subtypes of dyslexia corresponding to different loci of abnormality in the cerebellum; to what extent do cerebellar and magnocellular deficits co-occur; and how do these specific issues relate to underlying genetic endowment[33,34]? We consider these are all potentially fruitful research issues, and we consider their investigation will continue to illuminate the complex interplay between the brain, the environment and behaviour, in both normal and abnormal development.

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The cerebellum and dyslexia: perpetrator or innocent bystander?

Comment from Thomas Zeffiro and Guinevere Eden to Nicolson *et al.*

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The behavioral manifestations of developmental dyslexia are notably complex, including deficits in reading, voluntary movement and early sensory processing. Although extensive behavioral research has carefully characterized the often subtle character of these abnormalities, a clear understanding of the specific neural mechanisms, whose dysfunction could explain the myriad behavioral manifestations of dyslexia, has been elusive. Although many believe the core deficit in dyslexia to involve language dysfunction[1], others have emphasized the possible role of disrupted sensory processing mechanisms[2], or an overall disorder involving sensorimotor coordination evidenced by deficits in performance of speeded, voluntary movement[3]. Focusing on the known abnormalities in voluntary movement and skill acquisition, the authors of the target article argue for a primarily cerebellar localization of the pathophysiology of dyslexia. Although this is an intriguing and novel hypothesis that is consistent with much of the known behavioral phenomenology of this disorder, it is worthwhile to briefly review this argument in the context of current knowledge concerning the behavioral effects of cerebellar disease and cerebrocerebellar functional organization.

Cerebellar dysfunction

What are the consequences of acquired cerebellar damage in man? As elegantly described by Holmes[4] the motor abnormalities consequent from focal or neurodegenerative damage to the cerebellar cortex and its nuclei are profound and might be explained with reference to four core deficits. First is hypotonia, the reduction in resistance encountered during external perturbation or passive movement of an extremity. Second is asthenia, a condition in which the affected individual becomes unusually fatigued following repeated movement. Third are disorders of voluntary movement manifested by difficulty in controlling the rate, range and force of repetitive movement. This problem is characterized not so much as an abnormality-initiating movement as in the control of its dynamic phase. Last are disorders of associated movements, such as the unconscious swinging of the arms during normal locomotion. These fundamental components of cerebellar dysfunction were believed, by Holmes and many others since his time, to explain the commonly observed clinical manifestations of cerebellar disease including, dysmetria, dysdiadochokinesia, rebound, intention tremor, nystagmus and dysarthria.

Comparing cerebellar damage and developmental dyslexia

If we are to accept the contention that cerebellar disorder causes the behavioral manifestations of developmental dyslexia, we might also expect dyslexics to resemble patients with acquired cerebellar damage. Therefore, it is reasonable to examine the concordance between clinical signs and symptoms seen in individuals with either cerebellar damage or developmental dyslexia. As discussed previously, the cardinal signs of acquired cerebellar damage include hypotonia, asthenia and fatigability, disorders of the rate, force and regularity of voluntary movement and a disturbance of associated movements. These deficits are most clearly seen acutely following large lesions to the cerebellum or in individuals with a variety of neurodegenerative disorders affecting the cerebellar cortex, its deep nuclei and its brainstem connections. The voluntary movement disorder (ataxia) most characteristic of cerebellar disease can be observed under a variety of conditions and is easily elicited by having the patient perform a speeded reaching task with visual guidance. Therefore, it seems strange that individuals with developmental dyslexia, if that disorder results from a primary cerebellar lesion, do not exhibit more florid manifestations of the classic cerebellar clinical syndrome. Conversely, if normal cerebellar function is crucial to normal reading, why do patients with substantial cerebellar disease, either focal or diffuse, not exhibit prominent abnormalities in reading and phonological processing? The behavioral overlap between cerebellar syndromes and dyslexia appears to be small, limited to the sorts of shared abnormalities in motor learning and movement automatization described in the target article. These facts would appear to present a problem for any theory placing the principal locus of dyslexic pathophysiology in the cerebellum.

One answer to this apparent paradox might come from consideration of the behavioral consequences of developmental as contrasted with acquired neural system dysfunction. In the former case, abnormal development of the cerebellum might result in macroscopically undetectable effects on cerebellar morphology and only mildly dysfunctional cerebellar functioning, revealed only through careful and focused behavioral testing. Although this possibility is supported by the lack of evidence for microscopic or

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macroscopic morphologic abnormalities in individuals with dyslexia, it is rather unsatisfying to base a theory of dyslexic pathophysiology on forces and effects unobserved.

'…it seems strange that individuals with developmental dyslexia…do not exhibit more florid manifestations of the classic cerebellar clinical syndrome.'

Another possibility is that, even though the cerebellum is dysfunctional in dyslexia, the primary pathology is located elsewhere in the brain, exerting its effects via a modulatory influence on cerebellar processing. This is a more probable explanation for the subtle cerebellar signs and symptoms seen in many individuals with dyslexia. The cerebellum is part of multiple circuits that include thalamic nuclei, neocortical regions, pontine, and inferior olivary nuclei[5]. Normal function of each of these circuits depends upon integrity of the entire system and it does not require a great leap to imagine that cerebellar processing could be adversely affected by disrupted sensory input from corticopontine channels, thereby allowing dysfunction in cerebral portions of the cerebrocerebellar circuit to mimic primary cerebellar disease. For example, smooth oculomotor pursuit is a function that requires intact cerebellar function. Although cerebellar damage can cause abnormalities in smooth pursuit, it is also possible that manipulation of the visual input channel from extrastriate cortex could cause a similar deficit in sensorimotor integration. Through this mechanism, abnormalities in visual motion processing[2,6] could contribute to the observed deficits in smooth pursuit seen in individuals with developmental dyslexia[7] by providing the cerebellar oculomotor control mechanisms with inaccurate information concerning movement of the visual background.

Therefore, the cerebellum might stand unfairly accused, an innocent bystander in the processes responsible for disordered motor control in developmental dyslexia. As regards the neuroanatomical locus of the problem, there is now microscopic anatomical, macroscopic anatomical, and electrophysiologic evidence that the actual culprit might be located in persylvian neocortical regions[8,9]. These areas receive projections from the cerebellum via the thalamus and project to the cerebellum via corticopontine projections to the pontine tegmentum. It might be that the 'cerebellar' signs and symptoms associated with developmental dyslexia reflect a remote effect of neocortical perisylvian damage on cerebellar function.

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A neural instantiation of the motor theory of speech perception

Comment from Richard B. Ivry and Timothy C. Justus to Nicolson *et al.*

In their target article, Nicolson *et al.* propose a novel causal hypothesis concerning the neural roots of dyslexia. Rather than focus on the cortical areas traditionally associated with language processing, they suggest we turn our attention to the cerebellum. To those who have not followed the recent literature implicating the cerebellum in higher cognitive function, this hypothesis is sure to be surprising. Traditionally viewed as part of the motor system, the cerebellum would not be expected to be associated with a disorder defined as a linguistic deficit.

Revisiting the motor theory of speech perception

Nonetheless, their hypothesis redirects our attention to one of the most influential theories of speech perception, the motor theory of speech perception[1]. As the name implies, the central premise of the motor theory is that the recognition of the phonological units of speech is based on inferring the articulatory gestures of the speaker. This theory was proposed to account for our ability to perceive the invariant articulatory events that form the speech stream, in spite of the great variability in the acoustic signal.

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When seen from this perspective, the target article provides a link between the motor theory of speech perception and the developmental disorder of dyslexia[2]. The psychological processes outlined in the causal chain depicted in Fig. 2 of the target article builds on one well-established psychological perspective, namely that a core deficit in dyslexia involves noisy phonological representations[3], creating a situation in which the individuals are taxed in their ability to map these representations onto arbitrary visual symbols. In Nicolson *et al.*'s proposal, the phonological deficit results from an articulatory problem, thus connecting back to the motor theory. However, this version of the motor theory is primarily ontological. The intimate connection between production and perception is essential for the development of phonological knowledge.

'…the abnormal metabolic picture in the dyslexic group might be related to their hypothesised phonological impairment.'

What is most novel about the theory is the idea that a cerebellar impairment is the triggering agent for this causal chain of events. Nicolson *et al.* hypothesise a set of functional pathways through which the cerebellar dysfunction could contribute to the behavioural signs associated with dyslexia, including language-based problems with writing, reading, and spelling, as well as a more general problem found in tasks requiring rapid naming[4]. The writing problems are thought to be the linguistic manifestation of clumsiness. The reading, spelling and rapid naming deficits arise from the articulatory-based phonological impairment, in addition to an impairment in automatisation. Although the cerebellum is frequently associated with automatisation, this vague term is best thought of as a descriptive label for a behaviour rather than the specification of a process. It is possible that the rapid naming problem might be another manifestation of a noisy articulatory/phonological system.

The role of the cerebellum in articulatory rehearsal and phonological representation

The primary evidence associating the cerebellum with language comes from neuroimaging studies[5]. Even when overt movement is controlled for, increased activation within the cerebellum has been observed in numerous studies involving verbal short-term memory and semantic retrieval. This activation has been hypothesised to reflect the contribution of the cerebellum to articulatory preparation and/or to covert rehearsal. The Nicolson *et al.* model builds on this interpretation, although as noted above, they might interpret these activations to also reflect a cerebellar role in accessing well-learned (automated) representations[6]. The preliminary PET data reported in the target article are taken as evidence for a general problem in skill automatisation. However, the reduced cerebellar activity in the dyslexics could reflect reduced use of verbal rehearsal in the trial-and-error motor learning task. Such a hypothesis would account for the fact that blood flow to the cerebellum was reduced in the dyslexic group not only during the production of learned sequences, but also during the initial acquisition phase. We do not wish to argue that the cerebellum is not involved in non-verbal skill acquisition. Rather the point here is the abnormal metabolic picture in the dyslexic group might be related to their hypothesised phonological impairment.

A different approach for exploring the role of the cerebellum in language comes from the study of patients with acquired cerebellar lesions. Although the evidence is relatively sparse[7,8], the clinical picture indicates that speech perception is essentially normal in these patients. Similarly, there are no reports of acquired dyslexia following cerebellar damage. In the context of the Nicolson *et al.* model, the role of the cerebellum in dyslexia would be motoric in nature: as part of the articulatory network, the cerebellum helps establish phonological representations during development. Once established, these representations might be accessed without the cerebellum. The neural locus for a phonological store remains unclear, although superior temporal[9] or inferior parietal[10] cortices are probable candidates. Here we see a distinction between the motor theory as used for on-line perception and its account of development. In the former sense, articulation and phonology remain coupled[1]. In the latter, articulation is especially important during the acquisition of phonological knowledge, even though the two are considered independent to some degree[11].

A single neural locus for dyslexia?

The dissociation of articulatory rehearsal and phonological representation also makes clear that there could be multiple neural bases for dyslexia. Poorly developed phonological representations could result from (1) an impairment in articulation, (2) the consolidation of such representations, or (3) the access of these representations from systems involved in the conversion of orthography to phonology. The striking degree of clumsiness in dyslexia[12], in addition to the impairments on temporal processing tasks associated with the cerebellum[13] are consistent with the idea that an articulatory problem is at the head of the causal chain.

However, it might also be that these children exhibit a host of subclinical neural abnormalities, only one of which involves the cerebellum. Unlike acquired language disorders such as alexia without agraphia in which, by definition, the deficit is restricted, people with developmental dyslexia typically exhibit below normal performance on a host of linguistic and non-linguistic tasks. Even if future imaging studies were to identify structural

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abnormalities in the cerebellum, it is probable that abnormalities will also be found in other brain areas. Anatomical studies using *in vivo* MRI have identified cerebellar hypoplasia in several psychiatric disorders including autism[14], schizophrenia[15], and attention deficit and hyperactivity disorder[16]. Correspondingly, causal accounts based on the cerebellar abnormalities have been proposed for autism and schizophrenia. Nonetheless, the pathology does not appear to be restricted to the cerebellum: structural differences have also been found in cortical regions.

What then are we to make of this revisionist literature in which the cerebellum is suddenly being elevated from the low-level slave of the motor system to the key link of disorders as varied as schizophrenia and dyslexia? A sceptical position would be to argue that the cerebellar impairment is a correlate of the disorders, but not causal. For unknown reasons, the cerebellum is especially sensitive to problems that occur during neurodevelopment, but abnormalities in other, cortical regions are pre-eminent. Interestingly, the hypoplasia in autism, schizophrenia, and attention-deficit hyperactive disorder (ADHD) is associated with different cerebellar lobules. Knowing the time course of cerebellar neurogenesis and maturation might provide clues to windows during which the development of the CNS goes awry[17].

Alternatively, it is an important enterprise to consider causal accounts that include the cerebellum. The target article offers a fine example of how these can be developed and tested. Nicolson *et al.*'s working hypothesis for dyslexia builds upon a well-specified psychological model and, by emphasizing articulation as a skilled motor process, connects with more-traditional views of the cerebellum. As such, the ideas resonate with the central premise of the motor theory of speech perception and this theory's more recent progeny (e.g. mirror neurons, see Ref. 18). Perception and action are intimately linked, interactive processes. Our knowledge, be it linguistic, perceptual, or conceptual is constrained by the actions we can produce, for it is their production, as well as our ability to understand the actions of others, that renders this knowledge adaptive.

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Dyslexia, development and the cerebellum

Discussion by Nicolson *et al.* on commentaries by Ivry and Justus, and Zeffiro and Eden

We concluded our target article with four general points.

(1) A high proportion of dyslexic children show behavioural evidence of abnormal cerebellar function.

(2) In a neuroimaging study of dyslexic adults, there was evidence of abnormal function largely specific to the cerebellum, both for learning and in 'automatic' performance.

(3) The difficulties shown in skill automatization and in speech-related cognitive tasks are directly consistent with current conceptualizations of the role of the cerebellum.

(4) We provided a developmental schema of the problems likely to arise given cerebellar abnormality from birth. The schema accounts for the established problems of dyslexia and provides a principled explanation of why phonological deficits and speed deficits arise.

We are particularly encouraged that the two sets of commentaries (Zeffiro and Eden, and Ivry and Justus) support the central theme of the article, that the range of difficulties suffered by dyslexic children are consistent

with cerebellar deficit. Nonetheless, both commentaries suggest alternative and interesting interpretations of these and further data. In terms of our four conclusions, the only specific query was in fact point (3): Ivry and Justus suggest that the lack of cerebellar involvement in the learning phase might be attributable to a failure to adopt a normal 'verbal mediation' strategy. This could be an important factor in the learning phase, but in the 'automatic' phase (where they were tested on a very highly practised sequence that they had learned for two hours that morning) we ensured that each subject was able to perform concomitantly a serial digit span task or to participate in normal conversation while continuing to execute the prelearned sequence without deterioration in sequence performance[1]. We consider that the absence of cerebellar activation in the automatic condition indicates that verbal labelling alone is not a sufficient explanation.

In their interpretation of our data (and further data), the commentators make related points but offer different alternative conclusions. Both raise the parallel with acquired cerebellar lesions. Zeffiro and Eden ask why dyslexic children do not show the clinical signs of cerebellar damage, and why adults with cerebellar lesions do not show reading problems. In fact, we have found that dyslexic children do manifest the classical symptoms of cerebellar damage[2], but not in the 'florid' way envisaged by Zeffiro and Eden. No doubt this is because a child with a developmental disorder (from birth) makes adaptations that minimise the difficulties suffered. Our 'conscious compensation' hypothesis[3] proposes one such adaptation. However, the more important point relates to reading in patients with cerebellar damage. Such patients do indeed show articulatory difficulties. It is probable, however, that although articulation is crucial in the early stages in learning to read, it is not needed in skilled reading. Consequently, cerebellar involvement might be important for 'scaffolding' the learning but not for skilled performance. The point is made clearly by Ivry and Justus '… the cerebellum helps establish phonological representations during development. Once established these representations might be accessed without the cerebellum'. Therefore, it is not at all surprising that adults with acquired cerebellar lesions do not exhibit reading problems.

A further important issue is the thorny of one of correlation versus cause. Zeffiro and Eden eloquently suggest that 'the cerebellum might stand unfairly accused, an innocent bystander in the processes responsible for motor control in developmental dyslexia…[whereas] the actual culprit might be located in the perisylvian cortical regions'. Similarly, Ivry and Justus point out that the cerebellum has been implicated in several other disorders, including autism, attention deficit and schizophrenia, and in each of these disorders structural differences have been found in (different) cortical regions. At this stage, we consider the cerebellum the prime suspect, and have good reason to do so, but we would certainly not wish to rule out the possibility of abnormalities in other brain regions. Indeed we see these issues as being particularly fruitful, suggesting new and important research agenda that might help clarify the role of cortical and subcortical structures, not only in a range of developmental disorders but also in cognitive performance more generally.

In short, as concluded in the target article, our research and developmental schema should be seen as just the first of a set of causal models for dyslexia and for other developmental disorders. We trust that subsequent investigations will provide a much more detailed picture of the complex interactions between sensory, cortical and sub-cortical structures interacting with experience, skills and genetics. It seems probable that these investigations will lead to the establishment of different sub-types of dyslexia (and other developmental disorders) based on the particular regions of the cerebellum (or other structures) that are affected. We hope and expect that these investigations will lead to a change from the current symptom-based diagnoses of developmental disorder to a more consistent and more valuable 'brain-based' diagnostic methodology.

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