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, schizophrenia, and attention- 
deficit hyperactivity disorder. 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 
maturational might provide clues to windows during 
which the development of the CNS goes awry.

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.

Dyslexia, development and the cerebellum

Discussion by Nicolson et al. on commentaries by 
Ivry and J ustus, 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 J ustus) 
support the central theme of the article, that the range 
of difficulties suffered by dyslexic children are consistent

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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. 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, 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 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 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 subcortical 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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