Should there really be a ‘Dyslexia debate’?

As its name suggests, the book *The Dyslexia Debate* by Julian Elliott and Elena Grigorenko aims to generate a debate on the concept of developmental dyslexia. Contrary to some misguided detractors of dyslexia (see text box below) these authors do not attempt to deny the existence of children with a specific reading disorder, nor do they deny that it has a strong underlying biological basis, even less do they try to convince the reader that dyslexia is just an excuse for bad teaching or a synonym of social disadvantage. Indeed, they can be commended for being very up-to-date on current research on the topic.

The largest part of this book (Chapters 2, 3 and 4) is dedicated to a thorough review of the dyslexia literature, in its cognitive, neural, genetic and educational/therapeutic aspects. The breadth of research covered is remarkable, and its treatment is accurate, if not perfectly well balanced. One may indeed regret that, for the sake of its argument, the book prefers to emphasize inconsistencies and disagreements, rather than providing a more constructive synthesis by focusing on the (admittedly scarce) converging lines of evidence and points of broad agreement.

Most importantly, the comprehensive review of the literature is only a means to the book’s real purpose, developed in the first and the last chapters: to demonstrate, step by step, that the notion of dyslexia has no validity, and that we should prefer instead the notion of reading disability. The dispute is not just about whether the notion of specific reading disability should be called dyslexia. The authors argue that there is no good reason to distinguish specific reading disability (dyslexia) from reading disability (or poor reading). Let us begin by clarifying this apparently sibylline point about the definition of dyslexia.

**How to define dyslexia?**

According to the International Classification of Diseases (ICD-10), developmental dyslexia (under the name of specific reading disorder) is a specific and significant impairment in the development of reading skills that is not solely accounted for by mental age, visual acuity problems, or inadequate schooling (World Health Organization, 2011). Similarly, DSM-5 insists that ‘the learning difficulties are not better accounted for by intellectual disabilities, uncorrected visual or auditory acuity, other mental or neurological disorders, psychosocial adversity, lack of proficiency in the language of academic instruction, or inadequate educational instruction’ (American Psychiatric Association, 2013).

For most experts in the field, these definitions capture the essential feature of dyslexia, i.e. that it is a specific cognitive disorder, one that cannot be explained by more general factors. Hence the interchangeable use of developmental dyslexia and specific reading disability or disorder in the present review and elsewhere.

The most controversial exclusion criterion is the one concerning intelligence. In practice, many practitioners and researchers reserve the diagnosis of dyslexia to children who have either an IQ above a certain threshold, or a certain discrepancy between their reading skills and their IQ, or both. The logic is very straightforward: children with low IQ have difficulties learning about everything. Their difficulties with learning to read just follow from their broader difficulties. Children whose reading skills are significantly below their general intellectual abilities, on the other hand, require a
different, more specific, explanation. Does it make sense to lump all poor readers together, whether having a specific reading disability, low IQ, uncorrected hypermetropia, or not being properly schooled or taught?

Elliott and Grigorenko will have none of this. They review the many definitions of dyslexia that have been proposed (beyond international classifications), and argue that there is no widespread consensus about the right one, and no good reason to prefer one over another. They rebut international classifications’ deceptively simple logic by arguing that: (i) there is no evidence that different kinds of poor readers differ in terms of the underlying cognitive deficits; (ii) there is no evidence that they differ in the underlying neural basis; (iii) the evidence on the genetic basis of dyslexia remains too preliminary to contribute to the debate; (iv) most importantly, the evidence-based interventions that have proven to have some efficacy for reading disability are just the same for all kinds of poor readers; and (v) given that it has no therapeutic value, and that it has a number of adverse effects, the use of the term ‘dyslexia’ does overall more harm than good and should be discontinued. Let us now review these points step by step.

Elliott and Grigorenko review the numerous cognitive theories of dyslexia that have been proposed, and declare that there is no consensus. How could there be? Is there any known cognitive or mental disorder on which there is a consensus? Although they acknowledge the importance of the phonological deficit, they will not go as far as admitting that there is hardly a study in the literature that does not report a phonological deficit in a majority of dyslexic children. Concerning low-IQ poor readers, it is true that there is no evidence that they differ in the specific cognitive deficits characteristic of dyslexia. Indeed, they show the hallmarks of the phonological deficit as well (Hoskyn and Swanson, 2000).

Why would they not, if their low IQ affects their cognitive abilities uniformly, including their phonological skills? Even poor readers due to poor visual acuity or poor teaching may also have poor phonological skills, since in the course of development reading skills influence phonological skills as much as the other way around.

However, following this line of argument would imply believing that the phonological deficit is only a consequence of poor reading, which would ignore evidence from longitudinal studies showing that poor phonological (and more generally language) skills predict poor reading skills several years ahead, well before reading instruction (Puolakanaho et al., 2007). While this predictive relationship might hold in low-IQ poor readers as well, it cannot be the case for low visual acuity or poor teaching. Thus it is obvious that phonological deficits play a causal role in certain types of reading disability, but not in all of them. A similar point could be made for other subtypes of dyslexia with distinct cognitive deficits (visual, or visuo-attentional). The problem, however, is that whereas it is clear that not all dyslexics have a phonological deficit, there are many theories of non-phonological subtypes, and none of them has gained widespread acceptance. This argument therefore awaits further research.

Regarding the neural basis of dyslexia, I will skip discussion of functional neuroimaging, which invariably gives answers consistent with those of cognitive tests (Tanaka et al., 2011), and which does not address the underlying cause of dyslexia. Concerning the neuroanatomical basis of dyslexia, I have to agree that the current picture is far from clear. An optimistic review of voxel-based morphometry studies may lead one to think that dyslexic individuals are characterized by lower grey matter volume across most of the reading network (Richardson and Price, 2009). However, proper meta-analyses find relatively little consistency across studies (Linkersdörfer et al., 2012; Richlan et al., 2012), and a recent large-scale study with a sample size totaling almost the cumulated sample size of all previous studies failed to replicate all these results (Jednoróg et al., submitted for publication). Regarding diffusion imaging, studies are too scarce and use too diverse imaging and processing methods to allow one to draw clear conclusions from the existing literature (Vandermosten et al., 2012; Zhao et al., submitted for publication). And again, there is no evidence that brain differences observed in dyslexia are any different than those associated with low-IQ or other forms of poor reading. To be more precise, there is no evidence at all, since such comparisons have never been reported. Furthermore, some neuroimaging studies concern specific reading disability, others poor reading at various severity thresholds. Thus it may be that apparent inconsistencies result from confusing dyslexia with poor reading, and are indeed the outcome of the very brain differences whose existence Elliott and Grigorenko deny! The best way to test this hypothesis would be for future meta-analyses to consider selection criteria as a moderator.
At any rate, it is likely that some of the neuroanatomical differences characterizing dyslexia will be shared with other poor readers. Indeed, quantitative measures of neural tissue such as local grey matter volume and fractional anisotropy have been shown to change with experience, therefore the observed group differences, if reliable, might simply reflect differential reading experience, and would thus characterize all types of poor reading.

However, such reasoning may not hold for all brain correlates of dyslexia. In one study, cortical thickness in the visual word-form area differed between dyslexics and both age-matched and reading-matched controls (Altarelli et al., 2013), which suggests that this difference does not reduce to reading experience and will not necessarily be observed in all kinds of poor readers. Furthermore, some observations on dyslexic brains concern phenomena that are very precocious and unlikely to be affected by reading experience. The best example is the disruption of neuronal migration observed in the post-mortem studies by Galaburda and collaborators (1985), which however await confirmation. Nevertheless, the same studies also found a reversed asymmetry of the planum temporale, which has recently been confirmed (Altarelli et al., 2014), and which is unlikely to be a late consequence of reading acquisition. As similar research on the morphometry of brain landmarks in dyslexia progresses, it is likely that more robust and early differences will be found, and will differ between varieties of poor reading, as suggested at the functional level by Peyrin et al. (2011).

This appears to be the central piece of argument in Elliott and Grigorenko’s book. This is because, even if the arguments outlined above on cognitive deficits and their neural basis turn out to be wrong in the long run, never mind the causes, ‘all poor readers benefit from the same kind of evidence-based reading interventions’, so the argument goes, therefore it is counter productive to try and distinguish them in practice. Again, they are partly right. In the current state of the evidence, the best interventions for reading disabilities are phonics-based teaching programmes that are particularly intensive, systematic and explicit. And they have apparently been applied with equal (but moderate) success to all kinds of poor readers. Of course, it is an exaggeration to state that ‘all poor readers benefit’, when in fact there is always a substantial minority of children who do not seem to benefit much. Nevertheless, it is true that these children do not form a recognizable subtype of poor readers, and in particular IQ does not seem to be a good predictor of response to intervention (Stuebing et al., 2011).

Again, is this picture likely to remain the same in the long run? Have enough efforts been made to actually understand the nature of the problems of those who resist common interventions? Have enough efforts been made to test different types of reading intervention on different types of poor readers? Let us consider a few special cases. Does poor reading due to low visual acuity benefit from common types of reading intervention? Well, corrective glasses would certainly help more. Let us go back to the issue of the potential subtypes of dyslexia. Most theories that attempt to explain cases of dyslexia without a phonological deficit appeal to a visual or a visual-attentional deficit. We do not know for sure what the right theories of visual dyslexia are, but surely such cases exist. Are they likely to benefit as much from standard reading interventions as from treatments that target their symptoms more specifically? To give an example, one particular theory of visual dyslexia is that of a reduced visual attention span (Bosse et al., 2007). There is some preliminary evidence that children with such a deficit do indeed benefit from an intervention targeting their visual attention span (Valdois et al., 2014).

Admittedly, the evidence that different kinds of poor readers require different types of intervention is scarce. But the claim that one intervention fits all is also totally premature and bound to turn out to be wrong. It is already obvious that it cannot be true that all poor readers have the same problem, and that they all benefit from the same intervention. What we need is much more research on specific subtypes of dyslexia, and on what specific interventions best suit each kind of poor reader and each type of dyslexia. Is this going to be achieved by eliminating the concept of dyslexia and promoting confusion between all causes of reading disability as a matter of principle?

It is one thing to say that the concept of dyslexia is not valid and serves no purpose. It is yet another to argue that it is actually harmful. The accusations are well-known: stigmatizing, freeing teachers from the responsibility of good teaching, etc. What matters of course is not whether there are potential costs (there are always costs associated with any policy), but whether they are outweighed by greater benefits or not, and what the alternatives are. Many testimonies provide evidence of the positive effects that a diagnosis of dyslexia can have, so diagnosis does not have only negative consequences. It is also worth thinking whether it would be possible to reduce some of the costs by more efficient means than just suppressing dyslexia. For instance, one legitimate worry is that the term dyslexia, being more associated with biological factors than poor reading is, gives teachers the wrong impression that it is less prone to remediation and therefore that there is little they can do. While a generalized substitution of dyslexia with poor reading might help mitigate this problem (provided we manage to keep the secret that the whole range of reading ability is under biological influence...), another solution would simply be to educate teachers better about biological influences, brain plasticity and to explicitly target false beliefs about determinism. This would actually benefit many more children than just those with dyslexia.
classifications such as ICD and DSM attempt to delineate sets of individuals that seem to have similar problems at some level of description. This is primarily a scientific venture seeking to discover natural entities, i.e. sets of symptoms and underlying traits that tend to go together. While it is hoped that patients with similar disorders will benefit from the same treatment, this cannot be taken for granted, and most importantly this is never a condition for the definition of the diagnostic category. For instance, speech and language therapy may benefit children with speech sound disorder, specific language impairment, autism, Down’s syndrome, as well as adults with aphasia. Should they all be lumped into a single diagnostic category? Similarly, antidepressants can be useful to people with depression, as well as some with bipolar disorder, obsessive compulsive disorders, and many more. Yet psychiatrists and international classifications find it useful to distinguish these disorders. Finally, methylphenidate improves the attention skills of many children with ADHD, but also of medical students, and indeed of just about everyone else. Does it follow that the ADHD diagnostic category serves no purpose and that children with ADHD should be lumped with the rest of the population as having ‘limited attention’? Obviously, there is much more to diagnostic categories than just to respond to a similar treatment. Seen under this broader perspective, even if the claim that all poor readers benefit from the same intervention was correct, the conclusion that no distinction should be drawn among them simply would not follow. Diagnostic criteria and recommendations for intervention are just two different questions.

For the purpose of guiding efficient policy on reading disability on the basis of the current evidence, I would be inclined to recommend, just like the authors, not to worry too early about diagnostics, and rather to provide evidence-based educational interventions to all poor readers as a first intention, regardless of putative causes. However, adequately helping those who do not benefit enough from first-intention educational interventions likely requires a more thorough understanding of the nature of their problem, and eventually a diagnosis of dyslexia or of another disorder for some of them.

Overall, the arguments of the authors rest considerably on ‘there is no evidence’ statements. But ‘absence of evidence’ is not ‘evidence of absence’. Although the burden of proof lies with those making a claim (that there is some evidence), in the present case it is already clear that absence of evidence will not last forever. Then is it really worth eradicating the dyslexia concept from the face of the Earth if one has to backtrack a few years later?

What we need is to focus more research on the different kinds of reading disability, on their distinct causes, on the interventions that are optimal for each of them, and to study more specifically those children who seem to resist each form of intervention. One wonders how this will ever be achieved, if one imposes the dogma that all poor readers are alike.

By now the reader must have perceived that I disagree with the conclusions and recommendations of this book. Nevertheless this does not detract from the quality of the literature review. For this reason I would still be happy to recommend The Dyslexia Debate to students seeking a good overview of current dyslexia research, as long as they also have access to another perspective on the conclusions, such as this review.

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