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Cross-linguistic neuroimaging and dyslexia: A critical view

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ABSTRACT

Recent neuro-cognitive theories of dyslexia presume that all dyslexics have the same type of brain abnormality irrespective of the particular writing system their language uses. In this article, we indicate how this presumption is inconsistent with cross-linguistic investigations of reading and dyslexia. There are two main issues. First, the information-processing requirements of reading vary greatly across different orthographies. Second, it is known that even within a single orthography there are different subtypes of dyslexia. Consequentially, it cannot be the case, not even within a single orthography let alone across orthographies, that all dyslexics have the same type of brain abnormality. Neuro-cognitive theorizing about dyslexia cannot afford to ignore these issues.

1. The definition problem

Developmental dyslexia is a complex disorder involving specific difficulty in the acquisition of reading and writing skills (Critchley, 1970; World Health Organization, 1993). It appears to vary in prevalence across languages with different writing systems (Goswami, 2002). Apart from entirely behavioural definitions, dyslexia has also been viewed as a neurological disorder with genetic origin (Grigorenko, 2001; Smith et al., 1998). However, the definition and explanations of dyslexia (for reviews see Demonet et al., 2004; Habib, 2000) have long been problematic (Solity, 1996).

A causal modelling framework involving three distinct levels of description – behavioural, cognitive, and biological (Jackson and Coltheart, 2001) – can shed light on some seemingly intractable problems and conundrums. Dyslexia can then be defined as a neuro-developmental disorder with a biological origin that is the basis for abnormalities at

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a cognitive level, which are the immediate causes of the behavioural signs of dyslexia. These hypothesized cognitive deficits are subject to an ongoing debate, but serve as a starting point for testable predictions at both the behavioural and the biological levels. At all three levels interactions with the properties of writing systems must occur. These interactions will have a major impact on the clinical manifestation of dyslexia, the handicap experienced by the dyslexic, and the possibilities for remediation (Frith, 1999; Siok et al., 2004).

2. Neuroimaging studies of dyslexia

The use of sophisticated cognitive tests in combination with a variety of neuroimaging methods, including functional Magnetic Resonance Imaging (fMRI), Positron Emission Tomography (PET), Electroencephalography (EEG) and Magnetic Source Imaging (MSI) can substantially contribute to our understanding of dyslexia, especially with respect to its neurobiological bases. Most neuroimaging studies converge in showing that in individuals with dyslexia there is much more underactivations of, and fewer connections between, the key neural network structures than is observed in nondyslexics (Goswami, 2008).

More specifically, recent neuroimaging studies investigating structure–function relationships with alphabetic languages have detected several brain regions with atypical function and anomalous structure in individuals with dyslexia (Siok et al., 2004). These include the left temporoparietal areas, the left middle-superior temporal cortex and the left inferior temporo-occipital gyrus.

The left temporoparietal areas are often thought to be involved in letter-to-sound conversion during reading (e.g., Aylward et al., 2003; Brown et al., 2001; Eden et al., 2004; Eden and Moats, 2002; Hoeft et al., 2006, 2007; Horwitz et al., 1998; Johansson, 2006; Price and Mechelli, 2005; Temple et al., 2003). Nevertheless, a recent study by Ben Shalom and Poeppel (2008) showed that these left temporoparietal areas (especially the supramarginal gyrus) are probably involved more in sensorimotor transformations at the phoneme level rather than letter-to-sound conversion.

The left middle-superior temporal cortex is especially important for speech sound analysis (Aylward et al., 2003; Brambati et al., 2004; Brown et al., 2001; Eckert, 2004; Paulesu et al., 2001).

Finally, the left inferior temporo-occipital gyrus seems to function as a rapid visual word recognition system (Brambati et al., 2004; Brown et al., 2001; Kronbichler et al., 2008; Shaywitz et al., 2002; Silani et al., 2005; Simos et al., 2002). Just as we reported in this section, Wimmer et al. (2010, this issue) also found left occipito-temporal underactivation. However, this was accompanied by overactivation in more posterior medial occipital regions, possibly reflecting prolonged or repeated low-level visual processing of the letter string information. Wimmer et al. (2010, this issue) conclude that as opposed to healthy readers, dyslexic individuals do not engage the so-called Visual Word Formation Area (Cohen et al., 2002), but instead tend to rely on these overactivated posterior occipital regions, and also on left frontal premotor and motor regions (for a more detailed description of these results, see Wimmer et al., 2010, this issue).

The neuroimaging results so far support a neurophysiological model of reading skill acquisition and its disorders according to which dyslexia is linked to atypical structural and functional development of posterior brain systems (e.g., Eden et al., 2004; Eden and Moats, 2002; Hoeft et al., 2007; Horwitz et al., 1998; Johansson, 2006; Price and Mechelli, 2005; Shaywitz et al., 1992).

3. Neuro-cognitive theories of dyslexia

Neuro-cognitive theories of dyslexia have been developed (e.g., Paulesu et al., 2001; Silani et al., 2005) that make the presumption that all dyslexics have the same type of brain abnormality irrespective of the properties of the orthographic system used to write their language. However, the neural circuits involved in reading and reading disorders would be expected to vary substantially across languages, because of differences in how a given writing system links print to spoken language (Eden et al., 2004; Goswami, 2002, 2006; Perfetti et al., 2005; Price and Mechelli, 2005; Schlaggar and McCandliss, 2007; Siok et al., 2004). Moreover, it is known and widely accepted that even within a single orthographic community there are different subtypes of dyslexia (e.g., Castles and Coltheart, 1993; Heim et al., 2008; King et al., 2007; Lorusso et al., 2004). We thus argue here that the neuro-cognitive theories that were developed recently by Paulesu et al. (2001) and Silani et al. (2005) are at least misleading, if not wrong, approaches to cross-linguistic investigations of dyslexia. We will first discuss the most important critical issues and deficiencies of these theories before venturing into what should be improved in future neuro-cognitive considerations of dyslexia and constructions of new theories.

4. Critical issues: the danger of being average

First and foremost, the papers by Paulesu et al. (2001) and Silani et al. (2005) assume that developmental dyslexia is a cognitively homogeneous condition, i.e., all dyslexics have the same type of reading difficulty. However, as we have indicated above this is known to be false: there are various different forms of developmental dyslexia (Jackson and Coltheart, 2001; Slaghuis, 2007). One form of developmental dyslexia is characterized by reading responses where letter position errors occur: this is developmental letter position dyslexia (Friedmann and Gvion, 2005; Friedmann and Rahamim, 2007). Another form of developmental dyslexia is developmental neglect dyslexia, in which reading errors occur only with respect to one end of the word, typically the left (Friedmann and Nachman-Katz, 2004). Then there is developmental attentional dyslexia (Friedmann et al., 2010, this issue; Rayner et al., 1989) in which letters from words in parafoveal vision intrude into the processing of the currently fixated word. Other dyslexics have a specific difficulty in learning the rules according to which letters are converted to sounds; a condition known as developmental phonological dyslexia (see e.g., Campbell and Butterworth, 1985; Temple and Marshall, 1983). Others, on the other hand, have a specific difficulty in building

up a large sight vocabulary for rapid and automatic recognition of words as wholes; this is developmental surface dyslexia. And these subtypes fractionate even further: Friedmann and Lukov (2008) have shown that developmental surface dyslexia itself occurs in three different forms.

It is not possible that these many different varieties of developmental dyslexia are all characterized by the same type of neural impairment. Hence, averaging imaging data across a heterogeneous group of individuals with dyslexia not selected according to subtype of dyslexia, as done by Paulesu et al. (2001) and Silani et al. (2005), cannot yield data from which meaningful conclusions can be made, because of the gross biological and cognitive heterogeneity of any group chosen in this way.

Quite apart from this completely general point, there were specific methodological difficulties with these studies. The method by which Italian dyslexics in Silani et al. (2005) were identified was quite different from the method by which English and French dyslexics were identified: the French and English subjects were people who volunteered for the study because they had in the past received a formal diagnosis of dyslexia, whereas the Italian subjects were selected from a large sample of 1200 students who were given group tests of spelling and stress assignment. Thus the different groups of dyslexic individuals were not comparable across different languages, as deemed necessary in the past (Frith, 1999; Siok et al., 2004).

In addition, the two papers assert that all individuals with dyslexia have a phonological deficit, which is not the case. There are examples in the literature where individuals with dyslexia did not show any phonological impairment (e.g., Shankarnarayan and Maruthy, 2007), and indeed of the five subtypes of developmental dyslexia we described above only one (developmental phonological dyslexia) seems to be at all consistently associated with the presence of phonological deficits.

If dyslexia had a universal pathophysiological basis in the brain, then dyslexic individuals whose scripts are not alphabetic (e.g., Chinese, Korean, Japanese, some Indian scripts) would show the same neural deficit as their alphabetic counterparts. Because the cognitive mechanisms required to read non-alphabetic scripts are very different from those required for reading alphabetic scripts, this seems highly unlikely; and it is in fact not true (Siok et al., 2004). Moreover, it seems that due to brain plasticity, differences between properties of reading systems can induce substantial neuroanatomical differences between populations reading alphabetically-written versus ideographically-written languages (Kochunov et al., 2003).

Consider, for example, the fact that in readers of alphabetic scripts localized brain damage in previously literate people can selectively affect nonword reading whilst sparing the reading of real words (acquired phonological dyslexia; for a review of this condition see Coltheart, 1996). This must mean that there is a specific brain region serving as the neural substrate for reading aloud via letter–sound correspondences. The brains of readers of ideographic scripts (such as Chinese, or Japanese kanji) or syllabic scripts, such as Japanese kana or Devanagari (as used for writing various Indian languages) will of course contain this specific brain region, but in such readers this region will certainly not subserve the function of letter-to-sound translation, because there are no letters in ideographic or syllabic scripts.

Even when one considers just readers of alphabetic scripts, the same point can be made. One of the problems which readers of English have to solve when using letter–sound rules to read aloud is that in English a phoneme is sometimes represented by two or more letters, such as the SH in SHIP or the IGH in HIGH. So it has been proposed (see e.g., Coltheart, 1985) that the English reading system contains a *graphemic parser* whose job is to parse a letter string into graphemes (the orthographic elements corresponding to phonemes). However, there are languages in which phonemes are only ever represented as single letters (Serbian and Bosnian, for example), and for such languages graphemic parsing is never needed. Hence when children learn to read English they will have to acquire a graphemic parsing system as a part of their reading systems, whereas children learning to read Serbian or Bosnian will not.

Another quirk of the English writing system is that sometimes a phoneme is represented by non-contiguous letters, such as the vowel phoneme in the word RACE: here we might refer to the A_E corresponding to the vowel phoneme as a "split grapheme". Italian and English differ from Serbian in that there are multiletter graphemes in both Italian and English; but Italian and English differ from each other in that English has split graphemes and Italian does not.

The French and English orthographies both require graphemic parsing and both include split graphemes; nevertheless, they still differ at an orthographic level in that accents are used in written French but not in written English.

Thus although English, French, Italian and Serbian are all written alphabetically, the reading systems of skilled readers of these languages will nevertheless be nonidentical because each language employs the alphabetic principle in unique ways (Seymour et al., 2003). This allows for the possibility – indeed, the probability – of language-specific forms of developmental dyslexia. Children learning English have difficulty learning letter–sound rules involving split graphemes, for example, but that difficulty cannot be seen in children learning to read Italian or Serbian.

5. Conclusion

Neuroimaging methods have played a role in identifying the neural correlates of reading impairments that are typically associated with dyslexia. However, a major deficiency of neuroimaging investigations is that they have too often failed to establish contact with contemporary models of cognition (see e.g., Coltheart, 2006; Poeppel, 1996; Raichle, 1998). Recent cross-linguistic neuroimaging studies of dyslexia have raised some important theoretical issues with respect to the neurobiological origin of the disorder; however, they failed to consider relevant conceptual and methodological shortcomings that were instead simply ignored. For instance, the neuro-cognitive theories introduced by Paulesu et al. (2001) and Silani et al. (2005) presume that, within any orthographic community, all dyslexics have the same type of brain abnormality. In this paper, we have discussed why these theories cannot be correct, or at least have major deficiencies

requiring substantial reconsideration of many vital arguments. Future neuro-cognitive theories and empirical studies should take these issues consistently into account in order to craft an adequate and up-to-date theory, as well as to develop better education possibilities for dyslexics reading various different orthographies.

Neuroimaging studies have the potential not only to identify the neural correlates of dyslexia, but also to offer a new set of constraints that will help in evaluating the adequacy of existing alternative theories in a cross-linguistic context. For this reason, we are optimistic that by seriously reconsidering the above-mentioned limitations, it should be possible to employ neuroimaging techniques to move beyond an oversimplified notion of the dyslexic brain, and to arrive at a unified cross-linguistic theory of reading disability that would reconcile a wide range of seemingly incompatible behavioural- and neuroimaging findings.

REFERENCES

Aylward EH, Richards TL, Berninger VW, Nagy WE, Field KM, Grimme AC, et al. Instructional treatment associated with changes in brain activation in children with dyslexia. *Neurology*, 61: 212–219, 2003.

Ben Shalom D and Poeppel D. Functional anatomic models of language: Assembling the pieces. *The Neuroscientist*, 14: 119–127, 2008.

Brambati SM, Termine C, Ruffino M, Stella G, Fazio F, Cappa SF, et al. Regional reductions of gray matter volume in familial dyslexia. *Neurology*, 63: 742–745, 2004.

Brown WE, Eliez S, Menon V, Rumsey JM, White CD, and Reiss AL. Preliminary evidence of widespread morphological variations of the brain in dyslexia. *Neurology*, 56: 781–783, 2001.

Campbell R and Butterworth B. Phonological dyslexia and dysgraphia in a highly literate subject: A developmental case with associated deficits of phonemic processing and awareness. *The Quarterly Journal of Experimental Psychology*, 37a: 435–475, 1985.

Castles A and Coltheart M. Varieties of developmental dyslexia. *Cognition*, 47: 149–180, 1993.

Cohen L, Lehericy S, Chochon F, Lemer C, Rivaud S, and Dehaene S. Language-specific tuning of visual cortex functional properties of the Visual Word Form Area. *Brain*, 125: 1054–1069, 2002.

Coltheart M. Cognitive neuropsychology and the study of reading. In Posner MI and Marin OSM (Eds), *Attention and Performance XI*. Hillsdale: Lawrence Erlbaum Associates, 1985.

Coltheart M. *Phonological Dyslexia*. Hove: Lawrence Erlbaum Associates, 1996.

Coltheart M. What has functional neuroimaging told us about the mind (so far)? *Cortex*, 42: 323–331, 2006.

Critchley M. *The Dyslexic Child*. London, UK: Heinemann Medical Books, 1970.

Demonet J, Taylor M, and Chaix Y. Developmental dyslexia. *Lancet*, 363: 1451–1460, 2004.

Eden GF, Jones KM, Cappell K, Gareau L, Wood FB, Zeffiro TA, et al. Neural changes following remediation in adult developmental dyslexia. *Neuron*, 44: 411–422, 2004.

Eden G and Moats L. The role of neuroscience in the remediation of students with dyslexia. *Nature Neuroscience*, 5: 1080–1084, 2002.

Eckert M. Neuroanatomical markers for dyslexia: A review of dyslexia structural imaging studies. *The Neuroscientist*, 10: 362–371, 2004.

Friedmann N and Gvion A. Letter form as a constraint for errors in neglect dyslexia and letter position dyslexia. *Behavioural Neurology*, 16: 145–158, 2005.

Friedmann N, Kerbel N, and Shvimer L. Developmental attentional dyslexia. *Cortex*, 46(10): 1216–1237, 2010.

Friedmann N and Lukov L. Developmental surface dyslexias. *Cortex*, 44: 1146–1160, 2008.

Friedmann N and Nachman-Katz I. Developmental neglect dyslexia in a Hebrew-reading child. *Cortex*, 40: 301–313, 2004.

Friedmann N and Rahamim E. Developmental letter position dyslexia. *Journal of Neuropsychology*, 1: 201–236, 2007.

Frith U. Paradoxes in the definition of dyslexia. *Dyslexia*, 5: 192–214, 1999.

Goswami U. Phonology, reading development, and dyslexia: A cross-linguistic perspective. *Annals of Dyslexia*, 52: 139–163, 2002.

Goswami U. Neuroscience and education: From research to practice? *Nature Reviews Neuroscience*, 7: 406–413, 2006.

Goswami U. Reading, dyslexia, and the brain. *Educational Research*, 50: 135–148, 2008.

Grigorenko EL. Developmental dyslexia: An update on genes, brains, and environments. *Journal of Child Psychology and Psychiatry*, 42: 91–125, 2001.

Habib M. The neurological basis of developmental dyslexia: An overview and working hypothesis. *Brain*, 123: 2373–2399, 2000.

Heim S, Tschierse J, Amunts K, Wilms M, Vossel S, Willmes K, et al. Cognitive subtypes of dyslexia. *Acta Neurobiologiae Experimentalis*, 68: 73–89, 2008.

Hoeft F, Hernandez A, McMillon G, Taylor-Hill H, Martindale JL, Meyler A, et al. Neural basis of dyslexia: A comparison between dyslexic and nondyslexic children equated for reading ability. *The Journal of Neuroscience*, 26: 10700–10708, 2006.

Hoeft F, Meyler A, Hernandez A, Juel C, Taylor-Hill H, Martindale JL, et al. Functional and morphometric brain dissociation between dyslexia and reading ability. *Proceedings of the National Academy of Sciences USA*, 104: 4234–4239, 2007.

Horwitz B, Rumsey JM, and Donohue BC. Functional connectivity of the angular gyrus in normal reading and dyslexia. *Proceedings of the National Academy of Sciences USA*, 95: 8939–8944, 1998.

Jackson N and Coltheart M. *Routes to Reading Success and Failure*. Hove: Psychology Press, 2001.

Johansson BB. Cultural and linguistic influence on brain organization for language and possible consequences for dyslexia: A review. *Annals of Dyslexia*, 56: 13–50, 2006.

King WM, Giess SA, and Lombardino LJ. Subtyping of children with developmental dyslexia via bootstrap aggregated clustering and the gap statistic: Comparison with the double-deficit hypothesis. *International Journal of Language and Communication Disorders*, 42: 77–95, 2007.

Kochunov P, Fox P, Lancaster J, Tan LH, Amunts K, Zilles K, et al. Localized morphological brain differences between English speaking Caucasians and Chinese speaking Asians: New evidence of anatomical plasticity. *NeuroReport*, 14: 961–964, 2003.

Kronbichler M, Wimmer H, Staffen W, Hutzler F, Mair A, and Ladurner G. Developmental dyslexia: Gray matter abnormalities in the occipitotemporal cortex. *Human Brain Mapping*, 29: 613–625, 2008.

Lorusso M, Facoetti A, Pesenti S, Cattaneo C, Molteni M, and Geiger G. Wider recognition in peripheral vision common to different subtypes of dyslexia. *Vision Research*, 44: 2413–2424, 2004.

Paulesu E, Démonet J-F, Fazio F, McCrory E, Chanoine V, Brunswick N, et al. Dyslexia: Cultural diversity and biological unity. *Science*, 291: 2165–2167, 2001.

Perfetti CA, Liu Y, and Tan LH. The lexical constituency model: Some implications of research on Chinese for general theories of reading. *Psychological Review*, 112: 43–59, 2005.
Poeppel D. A critical review of PET studies of phonological processing. *Brain and Language*, 55: 317–351, 1996.
Price CJ and Mechelli A. Reading and reading disturbance. *Current Opinion in Neurobiology*, 15: 231–238, 2005.
Raichle ME. Behind the scenes of functional brain imaging: A historical and physiological perspective. *Proceedings of the National Academy of Sciences USA*, 95: 765–772, 1998.
Rayner K, Murphy L, Henderson J, and Pollatsek A. Selective attentional dyslexia. *Cognitive Neuropsychology*, 6: 357–378, 1989.
Schlaggar BL and McCandliss BD. Development of neural systems for reading. *Annual Review of Neuroscience*, 30: 475–503, 2007.
Seymour PHK, Aro M, and Erskine JM. Foundation literacy acquisition in European orthographies. *British Journal of Psychology*, 94: 143–174, 2003.
Shankarnarayan VC and Maruthy S. Mismatch negativity in children with dyslexia speaking Indian languages. *Behavioral and Brain Functions*, 3: 36, 2007.
Shaywitz SE, Escobar MD, Shaywitz BA, Fletcher JM, and Makuch R. Evidence that dyslexia may represent the lower tail of a normal distribution of reading ability. *The New England Journal of Medicine*, 326: 145–150, 1992.
Shaywitz BA, Shaywitz SE, Pugh KR, Mencl WE, Fulbright RK, Skudlarski P, et al. Disruption of posterior brain systems for reading in children with developmental dyslexia. *Biological Psychiatry*, 52: 101–110, 2002.
Silani G, Frith U, Demonet J-F, Fazio F, Perani D, Price C, et al. Brain abnormalities underlying altered activation in dyslexia: A voxel-based morphometry study. *Brain*, 128: 2453–2461, 2005.
Simos PG, Fletcher JM, Bergman E, Berier JI, Foorman BR, Castillo EM, et al. Dyslexia-specific brain activation profile becomes normal following successful remedial training. *Neurology*, 58: 1203–1213, 2002.
Siok WT, Perfetti CA, Jin Z, and Tan LH. Biological abnormality of impaired reading is constrained by culture. *Nature*, 431: 71–76, 2004.
Slaghuis WL. Letter to the editor. *Vision Research*, 47: 1976–1978, 2007.
Smith SD, Kelley PM, and Brower AM. Molecular approaches to the genetic analysis of specific reading disability. *Human Biology*, 70: 239–256, 1998.
Solity J. Discrepancy definitions of dyslexia. *Educational Psychology in Practice*, 12: 141–151, 1996.
Temple CM and Marshall JC. A case study of developmental phonological dyslexia. *British Journal of Psychology*, 74: 517–533, 1983.
Temple E, Deutsch GK, Poldrack RA, Miller SL, Tallal P, Merzenich MM, et al. Neural deficits in children with dyslexia ameliorated by behavioral remediation: Evidence from functional MRI. *Proceedings of the National Academy of Sciences USA*, 100: 2860–2865, 2003.
Wimmer H, Schurz M, Sturm D, Richlan F, Klackl J, Kronbichler M, et al. A dual-route perspective on poor reading in a regular orthography: An fMRI study. *Cortex*, 46(10): 1284–1298, 2010.
World Health Organization. ICD-10. *The International Classification of Diseases*, vol. 10. Geneva: World Health Organization, 1993.