Neuroimaging sheds new light on the phonological deficit in dyslexia

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A new study reports that activations of superior temporal regions for speech are normal in dyslexia, although being less well connected to downstream frontal regions. These findings support the hypothesis of a deficit in the access to phonological representations rather than in the representations themselves.

Developmental dyslexia is a specific difficulty in learning to read that cannot be attributed to intellectual deficiency, sensory disorders, or inadequate schooling. It is well established that the underlying cause at the cognitive level is, for most dyslexic individuals, a phonological deficit; that is, a deficit affecting the representation and/or processing of speech sounds. For most researchers in this area, the most parsimonious hypothesis is that dyslexics' phonological representations are somewhat degraded (i.e., less precise, less well specified, less categorical, and/or noisier).

However, 10 years ago Gayaneh Szenkovits and myself initiated a series of experiments tapping the phonological deficit in dyslexia and, against our expectations, none of them was consistent with the hypothesis of degraded representations. It also appeared to us that the published literature was not as supportive as it seemed. Thus we tentatively proposed that dyslexic individual phonological representations were intact and that perhaps it was the ability to access them that was impaired [1]. Although this alternative hypothesis was not couched in neural terms, a straightforward translation would be that the neural networks supporting the representations (in auditory cortex and nearby regions) function normally, whereas the connections with other brain regions taking auditory representations as input are disrupted. The issue with the hypothesis of a deficit in phonological access is that it is very difficult experimentally to tease it apart from the alternative hypothesis. Indeed, all cognitive tasks that tap phonological representations inevitably require access to them, and vice versa. Attempts to design tasks that independently manipulate representational versus access demands have only been partly successful. Thus, a few years later, it was interesting to observe that several studies had reported data more consistent with an access than with a representation deficit [2]. However, not one of them was in a position to claim to have unambiguously settled the case.

Now, a study by Bart Boets and collaborators published in *Science* in December 2013 marks an important step in that direction [3]. These authors have capitalized on recently developed 'brain decoding' methods [here, multivoxel pattern analysis (MVPA)] to identify brain responses to sequences of bisyllabic pseudowords. By computing correlations between multivoxel patterns of activation for each pseudoword, depending on their phonological similarity, they quantified the degree to which a given cortical region shows activation patterns that are reliably similar for identical pseudowords and different for pseudowords differing by one consonant, one vowel, or both. Not surprisingly, they found that regions whose activation patterns best reflect phonological similarity include the primary auditory cortices, superior and middle temporal gyri, and supramarginal gyri. Against their expectations, they found that dyslexic and control participants did not differ in any way – not in the regions evoked by the stimuli, in the number of voxels activated in each region, or in each region's sensitivity to phonological similarity, even after running several alternative analyses.

Boets *et al.* also conducted a functional connectivity analysis between six bilateral seed regions showing peaks of activation for speech. They found that bilateral temporal regions were strongly coactivated between themselves as well as with the left inferior frontal gyrus (Broca's area). Connectivity patterns were very similar between the two groups, differing only in that they were weaker in dyslexics between Broca's area on the one hand and the left superior temporal gyrus and right primary auditory cortex on the other hand (Figure 1). This functional connectivity difference was reinforced by a structural connectivity difference in the left arcuate fasciculus shown in the authors' previous work [4] and extended in the present study.

This finding is particularly interesting because Broca's area is situated at a higher level in the speech sound-processing hierarchy, involved especially when phonological working memory or speech production is required [5]. For such tasks, there is a need for Broca's area to efficiently access input phonological representations hosted in superior temporal regions. Thus the results of Boets *et al.* suggest that the same phonological representations of dyslexic individuals' that are found intact using MVPA may be less accessible by Broca's area for certain purposes. This is compatible with earlier findings that, in complex phonological tasks as well as for reading aloud, dyslexic participants show not only reduced activations in temporal

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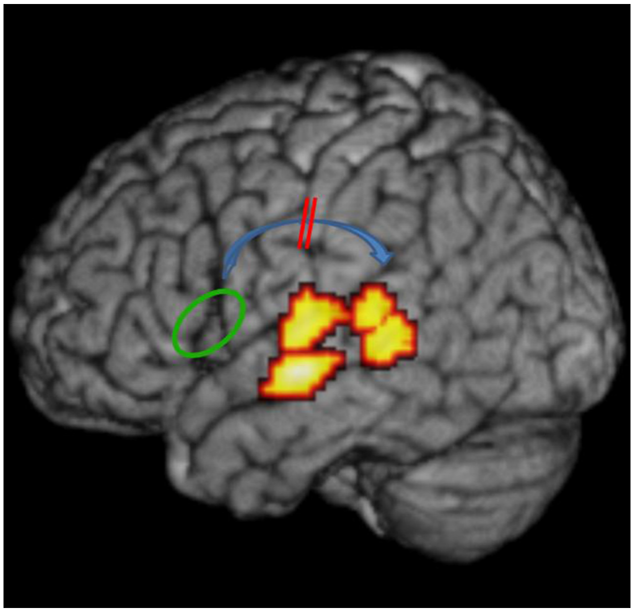

Figure 1. Schematic illustration of the cortical regions showing activations that are equally sensitive to phonological similarity in dyslexic and in control individuals (yellow). The green oval is centered on the left inferior frontal region, which is less well connected (blue arrow) with the superior temporal regions in dyslexic participants. Image by courtesy of Dr Bart Boets.

regions but also stronger activations in inferior frontal regions, interpreted as a form of effortful compensation [6,7]. It is also interesting that, whereas phoneme-discrimination performance in the scanner was at ceiling for both groups, dyslexic participants were reliably slower and their reaction times correlated with temporal–frontal connectivity, thus supporting the link between connectivity and access and suggesting that reduced connectivity impacts access time.

Of course, the most crucial finding, that of normal activations for phonological representations, is a null result and will need to be replicated. The main limitation of this result is the sensitivity of the MVPA technique to activation differences for subtle phonological contrasts. Although this sensitivity is largely unknown, the study shows that it is sufficient to differentiate patterns of activation for pseudowords that differ by only one consonant and demonstrates the expected left-hemisphere advantage for consonant but not for vowel contrasts. The lack of responsiveness of Broca's area to phonological contrasts in the MVPA analysis may seem surprising. However, this is expected given that the task was easy and purely perceptual, whereas Broca's area is thought to be part of an articulatory network that is recruited for perception only under demanding conditions [5]. Indeed some will argue that the phonological contrasts to be discriminated and the conditions of presentation made the task too easy to be sensitive to dyslexics' subtle deficits. I would, on the contrary, argue that this was a wise choice. Indeed, increasing task difficulty typically recruits additional working memory and metacognitive skills, increasing access as well as representational demands, thus making it impossible to tease apart the two hypotheses. If dyslexics' phonological representations are really degraded, it should be possible to demonstrate this with straightforward phonological contrasts under optimal listening conditions.

Although this study will certainly not put an end to this complex debate and will not convince everyone, it appears to be the best piece of evidence for the hypothesis of an access deficit since it was proposed 5 years ago. Remarkably, it may also be one rare case where neuroimaging seems to be better suited than experimental psychology to tease apart two hypotheses that were formulated strictly at the cognitive level.

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References

1 Ramus, F. and Szenkovits, G. (2008) What phonological deficit? *Q. J. Exp. Psychol.* 61, 129–141
2 Ramus, F. and Ahissar, M. (2012) Developmental dyslexia: the difficulties of interpreting poor performance, and the importance of normal performance. *Cogn. Neuropsychol.* 29, 104–122
3 Boets, B. *et al.* (2013) Intact but less accessible phonetic representations in adults with dyslexia. *Science* 342, 1251–1254
4 Vandermosten, M. *et al.* (2012) A tractography study in dyslexia: neuroanatomic correlates of orthographic, phonological and speech processing. *Brain* 135, 935–948
5 Hickok, G. and Poeppel, D. (2007) The cortical organization of speech processing. *Nat. Rev. Neurosci.* 8, 393–402
6 Shaywitz, S.E. *et al.* (1998) Functional disruption in the organization of the brain for reading in dyslexia. *Proc. Natl. Acad. Sci. U.S.A.* 95, 2636–2641
7 Brunswick, N. *et al.* (1999) Explicit and implicit processing of words and pseudowords by adult developmental dyslexics: a search for Wernicke's Wortschatz? *Brain* 122, 1901–1917