Brain abnormalities underlying altered activation in dyslexia: a voxel based morphometry study

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Voxel-based morphometry was used to assess the consistency among functional imaging and brain morphometry data in developmental dyslexia. Subjects, from three different cultural contexts (UK, France and Italy), were the same as those described in a previous PET activation paper, which revealed a common pattern of reduced activation during reading tasks in the left temporal and occipital lobes. We provide evidence that altered activation observed within the reading system is associated with altered density of grey and white matter of specific brain regions, such as the left middle and inferior temporal gyri and the left arcuate fasciculus. This supports the view that dyslexia is associated with both local grey matter dysfunction and with altered connectivity among phonological/reading areas. The differences were replicable across samples confirming that the neurological disorder underlying dyslexia is the same across the cultures investigated in the study.

Keywords: developmental dyslexia; MRI; PET; cultural context; phonological deficit; disconnection hypothesis; grey matter; white matter

Abbreviations: VBM = voxel-based morphometry

Introduction

Developmental dyslexia has been classically defined as a specific difficulty in the acquisition of reading and writing in spite of preserved general intelligence, learning opportunity, motivation or sensory acuity (Critchley, 1970; World Health Organization, 1993). The assumption that dyslexia is a developmental disorder of genetic origin with a neurological basis (Smith et al., 1998) is relatively recent. However, because of the variability of its behavioural manifestations, the nature of the neurological and cognitive basis of the disorder remains a matter of debate.

There have been a number of cognitive and neurological interpretations of the mechanisms responsible for dyslexia (reviews in Habib, 2000; Grigorenko, 2001; Ramus, 2003; Demonet et al., 2004). Most if not all the following hypotheses have received a certain amount of support from functional imaging investigations.

The strongest support has been obtained for the idea that dyslexia represents a type of language disorder specifically involving phonological processing (Liberman, 1973; Snowling, 1981; Bradley and Bryant, 1983; Frith, 1999). Brain areas involved in phonological processing (i.e. left perisylvian cortices, left middle and inferior temporal cortex) have been found to be dysfunctional in many studies (Rumsey et al., 1992, 1997, 1999; Paulesu et al., 1996, 2001; Salmelin et al., 1996; Shaywitz et al., 1998, 2002; Helenius et al., 2002; Ruff et al., 2002). Altered functionality of processes not directly involved in phonology has also been found in some studies. In particular, dysfunction of early sensory stages of the magnocellular visual and auditory pathways has been found (Tallal and Piercy, 1973; Livingstone et al., 1991; Eden et al., 1996; Demb et al., 1998; Temple et al., 2000); a dysfunctional visuospatial attentional system (Hari et al., 2001); and a disorder of the motor system including altered cerebellar physiology (Nicolson and Fawcett, 1990, 1994; Fawcett and Nicolson, 1992, 1994; Nicolson et al., 1999). Altered cortico-cortical connectivity of the language and...
visual areas (Paulesu et al., 1996; Klingberg et al., 2000) has also been invoked as a possible mechanism underlying dyslexia.

The anatomo-morphological correlations in dyslexia started with the post-mortem observations of Galaburda and colleagues (Geschwind and Levitsky, 1968; Galaburda et al., 1978, 1985; Galaburda and Kemper, 1979; Humphreys et al., 1990) who reported more symmetric plana temporale in dyslexic subjects. The analysis of the cortical pathology demonstrated the presence of a diffuse pattern of cortical scars, dyslaminations and ectopias.

A morphological post-mortem anatomical study is also available for the cerebellum, based on the same specimens from the Orton Society Brain Bank used by Galaburda and co-workers; in their study, Finch et al. (2002) identified a significantly larger mean cellular area in medial posterior and in the anterior lobe of the cerebellar cortex for the dyslexic subjects.

Several structural MR studies which followed have shown inconsistent results: for example, the observation of Galaburda has been replicated only in about half of the MRI studies (Habib, 2000; Grigorenko, 2001). Anatomical measures of other structures such as the corpus callosum have also provided variable results: enlarged splenium (Duara et al., 1991; Rumsey et al., 1996), shrinkage in the genu (Hynd et al., 1995), or abnormally shaped isthmus (Robichon and Habib, 1998; Robichon et al., 2000; von Plessen et al., 2002).

Recently, Brown et al. (2001), in their voxel-based morphometry (VBM) study, reported reduced grey matter in the orbital portion of the left inferior frontal gyrus and superior temporal gyrus, but also outside the classical language regions. The largest clusters were detected in the left temporo-parieto-occipital region, in the left inferior and middle temporal gyri, and inferior and superior frontal gyri, as well as superior cerebellar regions bilaterally, but only with low statistical thresholds. No differences in white matter densities were reported.

Leonard et al. (2001) reported four anatomical measures that differentiate their phonological dyslexic subjects from the reading-disabled and control subjects: marked rightward cerebral asymmetry, marked leftward asymmetry of the anterior lobe of the cerebellum, combined leftward asymmetry of the planum temporale and posterior ascending ramus of the sylvian fissure and large duplication of Heschl’s gyrus on the left.

Eckert et al. (2003), using the same method as that followed by Leonard et al. (2001), found significant morphological cerebral alterations in dyslexic children, such as smaller right anterior cerebellar lobes, pars triangularis of the inferior frontal gyrus bilaterally and overall brain volume. All these areas showed significant correlations with reading, spelling and language measures.

In a recent VBM study, Brambati et al. (2004) observed significant reductions of grey matter volume in areas of the brain associated with language and reading processing in people with a family history of dyslexia in comparison with controls who had no reading problems. Significant grey matter reductions were located bilaterally in the planum temporale, inferior temporal cortex, cerebellar nuclei, and in the left superior and inferior temporal regions.

Finally, there is one MRI morphometric study on the cerebellum: Rea et al. (2002) found that, although normal controls had a larger right hemispheric cerebellar cortical surface, the cerebellar hemispheres in the dyslexic subjects were symmetric.

Taken together, the MRI morphometric studies published so far on dyslexia reveal complex and variable anatomical patterns: these may be relevant for dyslexia as a neurological syndrome and the distribution of the underlying pathology but perhaps less informative on which morphometric abnormality is truly relevant for the core neuropsychological syndrome of dyslexia.

To date, no study has directly assessed the consistency between structural and functional imaging data collected during the performance of specific tasks in dyslexia.

In this paper we report a VBM study in the same groups of subjects that were previously described with a PET activation reading protocol. We performed a VBM analysis in subjects from three different cultural contexts: France, Italy and UK. For these subjects, PET activation data revealed a large area of shared reduced activation during reading tasks primarily in the left temporal and temporo-occipital region (Paulesu et al., 2001). The VBM analysis is an unbiased technique for characterizing regional cerebral volume and tissue concentration differences in structural magnetic resonance imaging. It has been widely used to evaluate grey and white matter densities in Alzheimer disease (Baron et al., 2001; Frisoni et al., 2002; Karas et al., 2003), mild cognitive impairment (Chetelat et al., 2002) and other degenerative disorders as semantic and frontal dementia (Mummery et al., 2000; Burton et al., 2002; Tisserand et al., 2002). VBM allowed us to assess whether altered activation is underpinned by a detectable morphological abnormality of grey and white matter densities of specific dysfunctional brain regions and to identify regions that correlate with poor performance in the reading task.

**Methods**

**Subjects**

The study comprises 3 groups of subjects with dyslexia: 10 Italian (mean age, 22 years; SD, 4), 11 French (mean age, 27 years; SD, 6) and 11 English (mean age, 24 years; SD, 5). They were matched with 9 Italian (mean age, 27 years; SD, 6), 12 French (mean age, 28 years; SD, 6) and 11 English control subjects (mean age, 24 years; SD, 3). All subjects had participated in a previous cross-cultural PET study of reading. Behavioural tests are briefly described in Table 1. No statistical differences in full IQ among controls and dyslexic subjects for each national group separately and as a whole were found (C versus D IT P = 0.13; C versus D FR P = 0.91; C versus D UK P = 0.33, C versus D overall P = 0.13; for a full description of the behavioural data, see Paulesu et al., 2001). Subjects also had a MPRAGE MRI scan for subsequent VBM analysis.
Mean and SD of the performance in behavioural tasks are reported for each of the six groups.

Table 1  Performance in intelligence, reading and phonological tasks

<table>
<thead>
<tr>
<th></th>
<th>Full IQ</th>
<th>Verbal IQ</th>
<th>Performance IQ</th>
<th>Non-word minus word reading (ms)</th>
<th>Digit naming</th>
<th>Spoonerism (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control subjects</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>French (n = 12)</td>
<td>119 ±12</td>
<td>123 ±9</td>
<td>110 ±13</td>
<td>128 ±91</td>
<td>14 ±2</td>
<td>115 ±31</td>
</tr>
<tr>
<td>Italian (n = 9)</td>
<td>123 ±10</td>
<td>123 ±9</td>
<td>119 ±12</td>
<td>56 ±38</td>
<td>25 ±6</td>
<td>45 ±22</td>
</tr>
<tr>
<td>UK (n = 11)</td>
<td>118 ±12</td>
<td>119 ±14</td>
<td>112 ±19</td>
<td>208 ±89</td>
<td>14 ±3</td>
<td>69 ±20</td>
</tr>
<tr>
<td><strong>Dyslexic subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>French (n = 11)</td>
<td>109 ±8</td>
<td>109 ±11</td>
<td>108 ±11</td>
<td>334 ±267</td>
<td>18 ±3</td>
<td>189 ±86</td>
</tr>
<tr>
<td>Italian (n = 10)</td>
<td>116 ±9</td>
<td>112 ±10</td>
<td>119 ±8</td>
<td>176 ±76</td>
<td>33 ±6</td>
<td>98 ±52</td>
</tr>
<tr>
<td>UK (n = 11)</td>
<td>105 ±10</td>
<td>104 ±9</td>
<td>103 ±14</td>
<td>583 ±189</td>
<td>19 ±3</td>
<td>173 ±71</td>
</tr>
</tbody>
</table>

Mean and SD of the performance in behavioural tasks are reported for each of the six groups.

**Structural MRI scanning protocol**

**Italian group**

MRI was performed on a 1.5 T whole body scanner (General Electric Medical Systems, Milwaukee, WI, USA), using a standard quadrature head-coil. A high resolution T1 weighted anatomical scan (3D, SPGR, IR PREPPED) was acquired for each subject using a MPRAGE sequence (flip angle 10°, TI = 700 ms, FOV = 200 mm × 200 mm, matrix 256 × 192) yielding 124 axial slices and a slice thickness of 1.5 mm with in-plane resolution of 1 mm × 1 mm.

**France and UK groups**

MRI was performed on a 2.0 T Siemens Vision Scanner. A 3D structural MRI was acquired on each subject using a T1 weighted MPRAGE sequence (TR/TE/TI/NEX 9.7/4/600/1, flip angle 12°, FOV = 256 mm × 192 mm, matrix 256 × 192) yielding 108 axial slices and a slice thickness of 1.5 mm with in-plane resolution of 1 mm × 1 mm.

**Data analysis**

Data were analysed on a Windows 2000-PC workstation using Matlab 6.5 (MatWorks, Natick, MA, USA) and statistical parametric mapping software (SPM 2, Wellcome Department of Imaging Neuroscience, London, UK, 2002).

**MRI data preprocessing**

MRI data were preprocessed on the optimized VBM protocol as described by Good et al. (2001).

In short, this procedure involves extraction of the brain from the native skull space to determine ideal stereotactic normalization parameters. Furthermore, the native MRI scans are stereotactically normalized and segmented into grey matter, white matter and CSF compartments. Finally, a Jacobian modulation was applied to the data to preserve the absolute regional amount of grey matter from distortions introduced by the stereotactic normalization (Ashburner and Friston, 2000). Each normalized, segmented and modulated image was finally smoothed with a 12 mm FWHM kernel.

**Statistical analyses**

**Between-group differences**

Anatomical differences between groups were computed with the t statistic on a voxel by voxel basis in the form of conjunction analysis of the independent between-group effects. The grey and white matter densities were compared as absolute units. Decreases and increases of densities were investigated. We also investigated the presence of culture-specific differences by computing the relevant interaction effect analyses: here each culture-specific difference between controls and dyslexics (e.g. French controls compared with French dyslexics) was compared with the same differences in the other two cultural contexts with appropriate linear contrasts.

**Correlation analyses**

The grey and white matter densities of dyslexic subjects of the three cultural contexts were also correlated with reading performance (reading speed) using linear regression. For each dyslexic subject of each nation and each normal control, voice onset time for single word reading was transformed into a Z-score with reference to the distribution of the reading times of the group of normal controls of the same nation. These Z-scores were then used for linear regression.

**Statistical thresholds**

For the grey matter brain regions identified as hypoactivated by the previous PET experiment (Paulesu et al., 2001), we applied small volume corrections (significance referred to as SVC in the following paragraphs) interrogating, in the VBM data, 10 mm wide spheres centred on the peaks identified by the PET data (Fig. 1).
For brain regions outside those identified by the PET data, a threshold of $P < 0.05$ corrected for the whole brain was used.

**Further analyses**

In order to test whether the present voxel-based morphometry data could support competing hypotheses on dyslexia, a similar small volume correction was performed on the brain areas called in question by the two best specified hypotheses: the cerebellum and the visual motion perception area MT/VS of the magnocellular system. For the cerebellar hypothesis, we selected the stereotactic coordinates of the reduced cerebellar activation as described by Nicolson et al. (1999): $x = -34; y = -40; z = -34$. For the visual magnocellular hypothesis, we selected the visual motion area MT/VS. As previous papers on dyslexia did not report the stereotactic coordinates for the altered response in MT/VS, we used the probabilistic stereotactic coordinates proposed by Mendola et al. (1999): left MT/VS, $x = -46; y = -70; z = 2$; right MT/VS, $x = 45; y = -67; z = -1$.

A similar small volume correction for spheres 10 mm wide was applied to the white matter within the arcuate fasciculus, as abnormalities here were expected by previous diffusion tensor imaging MRI data (Klingberg et al., 2000).

**Anatomical localization of the cerebral areas of altered grey and white matter density**

Anatomical localizations have been performed according to a revised definition of the stereotactic space (Montreal Neurological Institute) and the stereotactic templates released with SPM2 and MIRCro softwares (www.fil.ion.ucl.ac.uk/spm; www.mircro.com).

**Results**

**Between-group comparisons**

**Grey matter**

The comparison between dyslexic subjects and controls, in the form of conjunction analysis of the independent between-groups effects (France, Italy and UK), revealed a significant reduction of grey matter densities in the left middle temporal gyrus (BA 21) ($P = 0.02$ SVC; Table 2 and Fig. 2A).

A significant increase of grey matter densities was detected in a region of the left middle temporal gyrus posterior to that showing reduced grey matter (BA 37) ($P = 0.05$ SVC; Table 2 and Fig. 2A).

**Table 2** Grey matter density changes in dyslexics and correlation with reading performance

<table>
<thead>
<tr>
<th>Anatomical region</th>
<th>BA</th>
<th>Talairach coordinates</th>
<th>Z-score</th>
<th>Uncorrected P-value</th>
<th>SVC P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grey matter reductions in dyslexics</td>
<td>21 L</td>
<td>−56 −51</td>
<td>2.3</td>
<td>0.001</td>
<td>0.02</td>
</tr>
<tr>
<td>Grey matter augmentations in dyslexics</td>
<td>37 L</td>
<td>−60 −60</td>
<td>2.9</td>
<td>0.002</td>
<td>0.05</td>
</tr>
<tr>
<td>POSITIVE CORRELATION BETWEEN READING</td>
<td>37 L</td>
<td>−46 −50</td>
<td>2.6</td>
<td>0.004</td>
<td>0.04</td>
</tr>
</tbody>
</table>

No significant changes were detected at $P < 0.05$ corrected for whole brain outside the areas identified by the previous PET experiment and no significant change corrected for small volume of grey matter density was found around the cerebellar location described by Nicolson et al. (1999) or in area MT/VS.

Finally, the interaction effects analyses failed to show culture-specific differences in the VBM data.

**White matter**

Analysis of white matter density revealed a common reduction for the dyslexic subjects in the frontal and parietal portion of the arcuate fasciculus (Table 3 and Fig. 2B): in the depth of left Broca’s area (underneath BA 44; $P = 0.02$ SVC), in the left post-central gyrus (underneath BA 3; $P = 0.05$ SVC). Within the arcuate fasciculus there was also a trend for a significant white matter reduction in the depth of the supra-marginal gyrus ($P = 0.01$ uncorrected).

Just as for the grey matter, the interaction effect analyses did not show culture-specific differences in the white matter data.

**Linear regression analysis**

The same region showing an increment of grey matter density in the dyslexic population (the left inferior temporal gyrus; BA 37), also showed a negative correlation with reading $Z$-score ($P = 0.04$ SVC), in that the higher the ‘grey matter’ signal, the worse the performance (Table 2 and Fig. 3).

Exploration of the datapoints of the correlation analysis shows one outlier (Z-score 10 for its reading time). In order to test whether the significance of the regression analysis was heavily biased by this subject, we performed an additional regression analysis after excluding him; the result in BA 37 remained significant ($P = 0.05$ SVC).

**Discussion**

As discussed in the Introduction section, MRI morphometric studies in dyslexia have provided conflicting results. This may, in part, depend on the diffuse nature of the pathology...
underlying dyslexia as one can clearly appreciate from the diagrams of the distribution of cortical pathology described in post-mortem studies (Galaburda et al., 1985; Humphreys et al., 1990). The distribution of the pathology is somewhat variable from subject to subject, involving regions outside the boundaries of the brain areas that consistently have been found to be activated in reading tasks (Fiez and Petersen, 1998). Other causes of the discrepancies may be different inclusion criteria adopted to recruit the patients or the very different MRI techniques used over 20 years of such studies. Only recently the MRI morphometric techniques have reached a more standardized and user-independent approach with the VBM method (Ashburner and Friston, 2000; Good et al., 2001). Previous VBM studies in dyslexia

Fig. 2 (A) Decreases (in red) and increases (in blue) of grey matter in dyslexics versus controls, superimposed on a 3D rendering of the mean grey matter image of the dyslexic subjects. (B) Decreases (in red) of white matter in dyslexics versus controls, superimposed on a 3D rendering of the mean grey matter image of the dyslexic subjects. (C) The bar graphs represent the effect of interest in the middle temporal gyrus ($x = -56; y = -51; z = 2$) in the middle posterior temporal gyrus ($x = -60; y = -60; z = 5$) and deep in the inferior frontal gyrus ($x = -40; y = 3; z = 22$) in the three groups (control subjects in yellow and light blue, dyslexic subjects in orange and dark blue). The error bars indicate standard deviations.

Table 3 White matter density changes in dyslexics

<table>
<thead>
<tr>
<th>Anatomical region</th>
<th>BA</th>
<th>Talairach coordinates</th>
<th>Z-score</th>
<th>Uncorrected P-value</th>
<th>SVC P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inf frontal gyrus</td>
<td>44</td>
<td>L -40 3 22</td>
<td>3.1</td>
<td>0.001</td>
<td>0.02</td>
</tr>
<tr>
<td>Postcentral gyrus</td>
<td>3</td>
<td>L -42 -15 25</td>
<td>2.7</td>
<td>0.004</td>
<td>0.05</td>
</tr>
<tr>
<td>Supramarginal gyrus</td>
<td>40</td>
<td>L -55 -25 23</td>
<td>2.3</td>
<td>0.01</td>
<td>0.1</td>
</tr>
</tbody>
</table>
have shown either diffuse abnormalities (not only in the orbital portion of the left inferior frontal gyrus and superior temporal gyrus, but also outside the classical language regions) (Brown et al., 2001) or abnormalities in regions of the temporal lobes (Brambati et al., 2004). However, even though VBM was used in these studies, there are sufficient differences in the methodology with our study to make a comparison difficult. In the study by Brown et al. (2001), a low statistical threshold was used ($P < 0.05$ corrected only for spatial extent); in the study by Brambati et al. (2004), a small sample of familial dyslexics was studied with the concrete possibility that these cases represent a special subgroup when compared with our sample of sporadic dyslexics.

Taken together, all these considerations emphasize the importance of combining morphological and functional anatomical observations in order to ease the interpretation of the functional relevance of morphometric data and to identify abnormalities specific for the cognitive disorder under investigation. Our study represents the first such explicit attempt allowing us to address the question of whether the brain regions that proved to be dysfunctional in an activation protocol also show structural abnormalities. Notably, we were able to submit the same dyslexic subjects and their normal control subjects to both PET and MRI scanning. Our previous PET activation paper (Paulesu et al., 2001) demonstrated that well compensated adult developmental dyslexics from three different orthographic cultural contexts had reduced activation of the left middle and inferior temporal regions during reading tasks. An additional argument that supports the abnormal function of the left temporal lobe in dyslexia is a recent observation that the very same left inferior temporal region that shows reduced activation during reading tasks is also hypoactive during picture naming tasks (McCrorry et al., 2005).

We now present evidence that these brain regions are also anatomically abnormal.

The analysis of the grey and white matter data revealed that the VBM pattern in dyslexia cannot be described simply as a pattern of reduced grey and/or white matter, as in degenerative disorders. Instead, a cortical structural disorganization of the cortex with both reduction and increases of ‘grey matter’ was seen.

The left middle temporal region was the site of maximal difference in brain activation in normal and dyslexic subjects, and here an area of reduced grey matter density was observed which can be interpreted as a regional atrophy. However, this was surrounded by a more posterior region of relative augmentation of grey matter that spanned downwards into inferior temporal cortex. Notably, the higher the density of the grey matter in this latter region, the more impaired were the subjects in the reading tasks, as shown in the linear regression analysis between the $Z$-scores of reading performance and the regional grey matter density. We thus have two strands of evidence, one, a between-group difference when dyslexics are compared with normal controls, and the other, a within-group correlation in the dyslexics alone.

How can we interpret this finding? Since the late 1970s it has been known that microscopic analysis performed on a limited number of dyslexic brains found cortical abnormalities in the form of cortical dyslaminations, ectopias and scars (Galaburda and Kemper, 1979; Galaburda et al., 1985; Humphreys et al., 1990). We suggest that the automated brain tissue classification technique of VBM classifies ectopias and dislaminations as grey matter, with the consequence of a
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local augmentation of ‘grey matter’ volume. In other neurological disorders characterized by similar areas of cortical dysplasias, such as in partial epilepsy, the cortex appears microscopically thicker. A recent paper has reported augmented grey matter using VBM in such pathology (Merschhemke et al., 2003). We suggest that in dyslexics, the more severe the microscopic pathology, the more pervasive is the behavioural deficit.

One consequence of dysplasias and ectopias is reduced connectivity between neighbouring regions. This was shown to be the case in rats in which the pathology was experimentally induced (Rosen et al., 2000). This reduced connectivity may cause atrophic changes in neighbouring areas, such as the one seen in the left middle temporal gyrus.

Analysis of the white matter data also showed abnormalities. Significant reduction of white matter density in dyslexia was seen within the arcuate fasciculus. This finding replicates diffusion tensor imaging data of the Klingberg et al. (2000) and supports the idea that a part of the neurology underlying dyslexia could be attributed to a perturbed connectivity within the language network (Paulesu et al., 1996).

To what extent is a phonological interpretation of dyslexia supported by the previous PET activation experiment and present anatomical data on this sample of dyslexics from three European cultures? The same brain areas with reduced activation were found in dyslexics from three countries with different language background, different writing systems and different school systems (Paulesu et al., 2001). These areas included the left temporal lobe, primarily the middle temporal and the inferior temporal gyri. All have been previously implicated in tasks requiring phonological retrieval, such as naming or reading (Price et al., 1996; Vandenberghe et al., 1996; Poldrack et al., 1999; Jobard et al., 2003). On the other hand, no detectable abnormalities were found in the extrastriate visual cortex like area MT/V5 or in the cerebellum to support, in a simple way, alternative general interpretations of dyslexia (Stein et al., 2000; Nicolson et al., 2001a, b). For the cerebellum, however, it should be noted that a lack of grey or white matter differences in a voxel-based morphometry analysis may depend on a combination of smaller and larger lobules that could cancel out each other in the between-group analysis. To exclude this possibility, a detailed region of interest analysis of the individual lobules would be needed.

To summarize, the previous PET data on the same samples (Paulesu et al., 2001) and the present voxel-based morphometry data speak in favour of a disorder of the brain areas involved in phonology and in the decoding aspects of reading.

The phonological disorder observed in dyslexia, however, cannot be simply explained with the local malfunction of a single brain area associated with phonological processing. A more elaborated neurocognitive explanation has to be found to account for the complexity of the core phonological and reading disorder of dyslexia and the complexity of the imaging data available so far.

First, as documented in other disorders acquired in childhood, the developing brain shows a great deal of plasticity that allows the child to recover after local damage (Ewing-Cobbs et al., 2003). However, as dyslexia may remain severe across the entire life, a focal lesion model seems quite unlikely.

In addition, local grey matter pathology may also translate into white matter alterations to explain, at least in part, the diffuse nature of the functional cortical abnormalities documented in dyslexia with functional imaging. The disconnection hypothesis (Paulesu et al., 1996) is now supported by animal models of dyslexia (Rosen et al., 2000) and by two observations that the arcuate fasciculus is abnormal in dyslexia: a diffusion tensor imaging study (Klingberg et al., 2000) and the present VBM study. The coexistence of local cortical changes together with abnormality of cortico-cortical connectivity within the language neural network offers a more realistic description of the neurology of dyslexia at a systems level and may explain why tasks like reading or naming, which require the integration of multiple visual, phonological and articulatory codes, are sensitive in revealing a dyslexic brain to teachers and parents.

In the future it might be possible to integrate evidence from anatomical, physiological and behavioural investigations and to link them with genetic and cultural influences. This should lead to a full causal explanation of dyslexia.

References


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