Brain and Language 185 (2018) 54–66

Contents lists available at ScienceDirect

Brain and Language

journal homepage: www.elsevier.com/locate/b&l

Global gray matter morphometry differences between children with reading disability, ADHD, and comorbid reading disability/ADHD

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ARTICLE INFO

Keywords:
Reading disability
ADHD
Reading disability/ADHD
Structural neuroimaging
Thalamus
Frontal lobes
Striatum
VBM
Morphometry
Occipital lobes

ABSTRACT

Extensive, yet disparate, research exists elucidating structural anomalies in individuals with Reading Disability (RD) or ADHD. Despite ADHD and RD being highly comorbid, minimal research has attempted to determine shared patterns of morphometry between these disorders. In addition, there is no published research examining the morphometry of comorbid RD and ADHD (RD/ADHD). Hence, we conducted voxel-based morphometry on the MRI scans of 106 children, ages 8–12 years, with RD, ADHD, or RD/ADHD, and typically developing controls. We found right caudate and superior frontal regions in both RD and ADHD, along with areas specific to RD and to ADHD that are consistent with current theories on these disorders. Perhaps most importantly, we found a potential neurobiological substrate for RD/ADHD. Further, our findings illustrate both shared and specific contributors to RD/ADHD, supporting two current theories on the comorbidity of RD and ADHD, thereby facilitating future work on potential etiologies of RD/ADHD.

1. Introduction

Reading Disability (RD) and Attention Deficit Hyperactivity Disorder (ADHD) are two neurodevelopmental disorders that have a comorbidity greater than expected based upon the base rate of either disorder alone, about 25–40% (Boada, Willcutt, & Pennington, 2012; Shaywitz & Shaywitz, 2005). Despite the high comorbidity between these two disorders, the literature is disparate on whether comorbid RD/ADHD is a unique disorder or merely a summation of both RD and ADHD etiologies. As the literature deliberates, any contributions to understanding the neurobiological correlates of comorbid RD/ADHD may have wide-reaching implications in the field. Therefore, the primary purpose of this study was to discover whether there are distinct patterns of gray matter morphometry in children with comorbid RD/ADHD as compared to controls using VBM and if these patterns differ from having either disorder alone. Our secondary purpose was to determine if there are shared neurobiological correlates of RD and ADHD.

1.1. Reading disability

Reading Disability (RD) is often defined as poor word identification and decoding skills (basic reading) despite intact cognitive ability (IQ or other cognitive functions; Pennington, Peterson, & Mcgrath, 2010). There is substantial heterogeneity between theories on the etiology of reading disability. This heterogeneity is likely due to the diversity of symptoms across individuals with the disorder (Tamboer, Scholte, and Vorst, 2015) and to the different operational definitions of reading disability used throughout the literature. In terms of the latter, some researchers used the poor reader definition of reading disability which requires reading ability to be below average despite the child not being intellectually disabled; no IQ-achievement discrepancy is required (Siegel, 1992). Others used the discrepancy definition of reading disability which requires reading ability to be significantly below the child's measured intellect, following the DSM-IV as well as the USA's IDEA requirements prior to 2004. The World Health Organization defined development dyslexia as poor word recognition and spelling abilities despite adequate instruction, intelligence and sensory abilities (see Peterson & Pennington, 2012), so dyslexia could be considered a subset of reading disability given the additional spelling requirement. The RD literature utilizes all three definitions. Irrespective of how RD is defined, three theories of reading disability have been utilized more often than the rest in neuroimaging studies: double deficit, dual route, and visual attention.

The double-deficit theory postulates that dyslexia is due to poor phonological awareness, rapid automatized naming, or both (Jednoróg, Gawron, Marchewka, Heim, & Grabowska, 2013; Pugh et al., 2013). The dual route theory suggests that there are two routes to reading: phonological and orthographic, and reading problems can occur due to

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https://doi.org/10.1016/j.bandl.2018.08.004
Received 4 October 2017; Received in revised form 4 April 2018; Accepted 18 August 2018

damage or faulty development in either route (Coltheart, Rastle, Perry, Langdon, & Ziegler, 2001). The visual attention hypothesis states that reading problems are due to poor phonological processing, visual attention, both, or neither (Bosse, Tainturier, & Valdois, 2007). Hence, while there is heterogeneity between theories on the etiology of RD, one commonality across all theories is poor phonological processing, which is the most common deficit found in RD (Ramus et al., 2003). Further support for these theories is found in the morphometry literature.

Previous RD research using VBM analysis has found gray matter abnormalities in the occipital cortex, inferior and lateral temporal cortices, parietal cortex, frontal cortex and the cerebellum. Using the double deficit hypothesis of reading disability as a paradigm, Jednoróg et al. (2013) found children with poor phonological awareness had smaller gray matter volume clusters in the right precentral and left parietal lobe but larger gray matter volume clusters in the left cerebellum and right putamen. Children with poor rapid autonomic naming had the same brain regions implicated but in an opposite volumetric pattern to the poor phonological awareness group. For children with both poor phonological processing and rapid autonomic naming (double deficit), the VBM analysis found decreased gray matter in the right supramarginal gyrus and increased gray matter in the left cerebellum. In a study testing the visual attention theory of reading disability (Stein & Walsh, 1997), the authors found that left posterior STG and middle temporal deviations were associated with poor phonological processing/verbal working memory, and right lateral occipital/superior parietal deviations were related to visual attention deficits based on correlational analyses.

In contrast to these two theories, a considerable amount of research, both structural and functional, has been published related to the dual route model. Two studies proposed a similar model of dyslexia based on fMRI methodology (Pugh et al., 2000; Shaywitz, Lyon, & Shaywitz, 2006). They suggested three circuits are involved with dyslexia: ventral, dorsal and anterior. The ventral circuit includes the left lateral extrastriate and inferior occipital-temporal regions and is involved with rapid recognition of familiar words and letter strings (orthographic route to reading). The dorsal circuit includes left superior temporal and inferior parietal structures and is involved with the decoding of novel words (phonological route). The anterior route is used by individuals with RD to compensate for deficits in posterior functioning and includes the inferior frontal gyrus. Areas homologous to the dorsal and ventral routes in the right hemisphere may be used to compensate as well. All of the areas involved in the dorsal, ventral, and anterior circuits have been implicated in various VBM studies on RD (Black et al., 2012; Hoeft et al., 2007; Im, Raschle, Smith, Ellen Grant, & Gaab, 2015; Linkersdorfer, Lonnemann, Lindberg, Hasselhorn, & Fiebach, 2012; Raschle, Chang, & Gaab, 2012; Richardson and Price, 2009; Richlan, Kronbichler, & Wimmer, 2013; Tamboer et al., 2015; Xia, Hoeft, Zhang, & Shu, 2016). Nonetheless, results are variable regarding whether these clusters are equal to, larger, or smaller than controls across studies (Jednoróg et al., 2013; Pernet, Andersson, Paulesu, & Demonet, 2009). This variability may be related to the heterogeneity of the disorder and/or variations in the operational definitions of RD used, language spoken by the various samples, and ages included in the various samples. Two recent review articles recapitulate this point. Xia, Hancock, and Hoeft (2017) and Ramus, Altarelli, Jednoróg, Zhao, and Scotto di Covella (2017) both cite language, as well as small sample size, as limitations in RD studies that use imaging methodology. Other potential causes of heterogeneity in RD studies include variability in VBM methodology (Ramus, et al., 2017) and not considering RD's comorbidity with other neurodevelopmental disorders or RD subtypes (Xia et al., 2017). Therefore, future studies (including the current study) should address these methodological shortcomings.

1.2. Attention-Deficit/Hyperactivity Disorder

Attention-Deficit/Hyperactivity Disorder (ADHD) describes children who have heightened levels of inattention and/or hyperactivity/impulsivity for their age (APA, 2013). The most commonly cited theory on the etiology of ADHD is the frontal-striatal theory, which suggests that the prefrontal cortex and basal ganglia are not functioning optimally in ADHD (Barkley, 1997; Castellanos & Proal, 2012; Castellanos et al., 1996). Barkley (1997) found that the worst deficits in ADHD are within the areas of inhibition, working memory, self-regulation, sustained attention, other executive functions, and motor control. Many of these deficits are associated with the prefrontal-striatal circuit (in particular the dorsolateral prefrontal cortex and caudate), especially the cognitive aspects of executive functioning such as working memory, planning, and problem-solving (Castellanos & Proal, 2012; Castellanos et al., 1996; Dang et al., 2014; Monchi, Petrides, Mejia-Constain, and Strafella, 2007). Other structural research has identified additional frontal circuits that may be invovled in executive functions, including the inferior frontal-striatal-cerebellar (Carmona et al., 2005; Makris et al., 2015; Rubia, 2011), prefrontal-posterior parietal (Carmona et al., 2005; Seidman et al., 2006), and orbitofrontal-limbic (Carmona et al., 2005; Makris et al., 2007; Seidman et al., 2011) circuits. These circuits play a role in motor/behavioral inhibition, emotional regulation, selective attention, and visual regulation of attention (Castellanos & Proal, 2012), potentially for both bottom-up and top-down processes, depending upon the region and circuit (Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008). Moreover, the orbitofrontal-limbic circuit may serve an additional purpose of aiding in delay aversion processing – a behavior often compromised in those with ADHD (Sonuga-Barke et al., 2008). In general, the literature strongly implicates prefrontal (DLPFC, inferior frontal and orbitofrontal), striatal, limbic (cingulate and medial temporal lobe), and cerebellar abnormalities that may give rise to the various ADHD symptoms presented in the literature.

Corresponding with the different theories proposed, gray matter morphometry studies have found reduced volume in various parts of the prefrontal, parietal, temporal and cingulate cortices, the striatum, the cerebellum, and in total brain volume (Carmona et al., 2005; de Mello et al., 2013; Seidman et al., 2011; Yang et al., 2008). More specifically, multiple experiments have found that children with ADHD have smaller total gray and white matter volume compared to children without it (Carmona et al., 2005; Castellanos et al., 2002; Lim et al., 2013; Seidman et al., 2006; Yang et al., 2008), which persists from childhood into at least adolescence (Castellanos et al., 2002). When examining the frontal-striatal circuit, participants with ADHD have smaller gray matter clusters compared to controls in the dorsolateral prefrontal cortex, inferior frontal cortex, caudate, putamen, and anterior cingulate (Carmona et al., 2005; de Mello et al., 2013; Makris et al., 2015; Seidman et al., 2011; Tremols et al., 2008; see Krain and Castellanos, 2006 or Seidman, Valera, & Makris, 2005 for a review). In addition, reduced gray matter has been found in individuals with ADHD in the cerebellum and temporal-parietal regions (Carmona et al., 2005; Depue, Burgess, Bidwell, Willcutt, and Banich, 2011a; Lim et al., 2013; Pironti et al., 2014; van 't Ent et al., 2007; Villemonteix et al., 2015) and in orbitofrontal and limbic structures (Carmona et al., 2005; Frodl & Skokauskas, 2012; Krain & Castellanos, 2006; Seidman et al., 2006; van 't Ent et al., 2007). Nonetheless, not all studies find reduced gray matter volume in these structures. For example, some researchers have found that people with ADHD have larger clusters in the dorsolateral prefrontal cortex, orbitofrontal cortex, caudate, putamen, inferior parietal cortex and/or temporal cortex (Makris et al., 2015; Moreno-Alcázar et al., 2016; Seidman et al., 2011, 2005), while others have found the basal ganglia, amygdala, and hippocampus to be commensurate in size to controls (Pironti et al., 2014). This variability may be related to the heterogeneity of symptomology and behavioral deficits found in ADHD (e.g., about 20% of individuals with ADHD do not present with an executive function deficit, and some have other deficits