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Dyscalculia and dyslexia in adults: Cognitive bases of comorbidity

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ABSTRACT

The developmental learning disabilities dyscalculia and dyslexia have a combined prevalence of 10% or more, and a co-occurrence (comorbidity) rate of around 40%. The causes and consequences of this comorbidity are poorly understood, despite implications for identification and remediation. We examined the cognitive bases of MDRD comorbidity in four groups of 85 adults (dyscalculia only, dyslexia only, comorbid and control), controlling for IQ and attentional difficulties. We used a computerized testing battery including core components of mathematics and reading, plus domain general capacities. Our results provide one of the first descriptions of dyscalculia symptoms in adults, showing that impairment on core numerical tasks continues into adulthood. Dyscalculia and dyslexia showed independent domain specific deficits, however we also found evidence for domain general symptoms associated with both disorders. We argue that the presence of multiple underlying and additive impairments supports complex multifactorial models of comorbidity.

1. Introduction

At least 10% of children experience mathematical learning disabilities (dyscalculia; MD) and/or reading learning disabilities (dyslexia; RD), even though they have at least average intelligence and an adequate learning environment (Butterworth, 2010; Peterson & Pennington, 2012). These developmental disorders continue into adulthood and are associated with poor socio-economic outcomes (Parsons & Bynner, 1997; Rivera-Batiz, 1992).

Dyscalculia and dyslexia show a high rate of co-occurrence or comorbidity (average 40%; Wilson & Waldie, in preparation), far greater than would be expected by chance, however the cause of this is unknown. Subtypes of both disorders have been proposed, which may be linked to comorbidity. Describing the phenotype of comorbidity in childhood and adulthood thus has important implications for identification, remediation, and our conception of learning disabilities.

The primary goal of this study was to establish a cognitive phenotype of comorbidity in adults, measuring core cognitive components involved in numerical cognition and reading. A secondary goal was to provide a description of adult dyscalculia.

1.1. Dyscalculia

Dyscalculia is considerably under-researched compared to dyslexia (Gersten, Clarke, & Mazzocco, 2007). There is a broad consensus that dyscalculia is characterized by unexpected severe impairment in mathematics, in the absence of intellectual disabilities, with prevalence estimates averaging 6% (Badian, 1999; Barbaresi, Katusic, Colligan, Weaver, & Jacobsen, 2005; Gross-Tsur, Manor, & Shalev, 1996; Landerl & Moll, 2010; Lewis, Hitch, & Walker, 1994; Räsänen & Ahonen, 1995; Reigosa-Crespo et al., 2011). This definition is very similar to those used by the DSM-IV (*mathematics disability*) and ICD-10 (*specific disorder of arithmetical skills*), thus here we use the terms dyscalculia and mathematical disabilities interchangeably.[1]

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[1] Recently some authors have proposed the existence of two populations; severe difficulties ("dyscalculia" or "mathematical learning disabilities"), and a broader "low achieving" group (Geary, 2011), also termed "arithmetical dysfluency" (Reigosa-Crespo et al., 2011), "mathematical difficulties" (Mazzocco, 2007), or in one instance "mathematical learning disabilities" (Rubinsten & Henik, 2009). Some authors have proposed that the mathematics difficulties in this broader group are the result of domain general impairments (Reigosa-Crespo et al., 2011; Rubinsten & Henik, 2009). However thus far studies have not found a clear difference in domain general capacities between these two groups (Murphy, Mazzocco, Hanich, & Early, 2007), or have found more severe deficits only in the dyscalculia group (for a review see Geary, 2011).

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1.2. Dyscalculia over the developmental trajectory

The first signs of dyscalculia are seen in early schooling as delays learning counting and using efficient arithmetic strategies, followed by a persistent difficulty in recalling arithmetical facts (for a review see Geary, 2011). Recent research has focused on difficulties with number (Butterworth & Reigosa, 2007; Geary, 2011). Eight to ten year old children with dyscalculia are slower to subitize (i.e. rapidly enumerate groups of 1–4 objects) and to count (Koontz & Berch, 1996; Schleifer & Landerl, 2011; van der Sluis, de Jong, & van der Leij, 2004). Their placement of numbers on a blank number line is less accurate and linear (Geary, Hoard, Nugent, & Byrd-Craven, 2008), they have a poorer understanding of place value (Chan & Ho, 2010), they are less efficient at approximate calculation (Rousselle & Noël, 2008), and they process numbers less automatically (Ashkenazi, Rubinsten, & Henik, 2009; Landerl & Kölle, 2009; Rousselle & Noël, 2007). Not only are they slower to select the larger of two one-digit numbers (Landerl, Bevan, & Butterworth, 2004), but they show a larger distance effect when doing so (i.e. a steeper in RT or accuracy as a function of numerical distance, a pattern typical of less precise number representation; Ashkenazi, Mark-Zigdon, & Henik, 2009; Rousselle & Noël, 2007; although for a conflicting result see Landerl & Kölle, 2009).

Difficulties with arithmetic in dyscalculia continue into high school (Calhoon, Emerson, Flores, & Houchins, 2007; Cawley & Miller, 1989; Shalev, Manor, & Gross-Tsur, 2005), and number difficulties are present for fractions and decimals (Mazzocco & Devlin, 2008) at age 11–13 years. In adults, dyscalculia has not been systematically studied, with studies focusing on isolated tasks (e.g. number/size interference; Rubinsten & Henik, 2005, 2006; number bisection; Ashkenazi & Henik, 2010b; Mussolin, Martin, & Schiltz, 2011). Ashkenazi and Henik (2010a) found that adults with dyscalculia (excluding RD or ADHD) were slower to compare and calculate with multidigit numbers, fractions and decimals. However it is not clear whether this is due to the same core number difficulties found in children, or to difficulties with higher order processes such as comprehension of algebra, fractions/decimals or procedural memory. Ideally, one would seek to answer such developmental questions with longitudinal data (Ansari & Karmiloff-Smith, 2002); however converging evidence from cross-sectional data also provides important information. In the current cross-sectional study, we sought to examine whether lower-order number impairment in dyscalculia is also seen in adults.

1.3. Disentangling the core deficits in dyscalculia

A numerosity representation (or "number sense") deficit has been proposed as a core deficit in dyscalculia (for reviews see Butterworth, 2005; Wilson & Dehaene, 2007). Approximate representation of numerosity, or the number of objects in a set, is a prelinguistic competency possessed by animals and pre-verbal humans (Dehaene, 2001). This capacity is localized to at least one particular brain area (the intra-parietal sulcus; IPS) and is proposed to represent the semantics of number (Piazza & Izard, 2009). Number sense can be measured directly using nonsymbolic tasks (e.g. using groups of dots rather than digits or number words), including comparison, estimation, addition and subtraction of sets of objects (Cantlon, Brannon, Carter, & Pelphrey, 2006; Izard & Dehaene, 2008; Piazza, Pinel, Le Bihan, & Dehaene, 2007). Number sense is also accessed in symbolic tasks which rely heavily on understanding quantity, e.g. subtraction or approximation, as opposed to those relying on verbal memory, e.g. recall of multiplication or addition facts (Dehaene, Piazza, Pinel, & Cohen, 2003). Numerosity representation increases in acuity over childhood (Halberda & Feigenson, 2008; Piazza et al., 2010), probably as a result of "tuning" due to experience with symbolic numbers (Pica, Lemer, Izard, & Dehaene, 2004), and is predictive of mathematical achievement (Desoete, Ceulemans, De Weerdt, & Pieters, 2012; Gilmore, McCarthy, & Spelke, 2010; Halberda, Mazzocco, & Feigenson, 2008).

This core deficit hypothesis is supported by findings that children with dyscalculia represent numerosity with less acuity (Landerl, Fussenegger, Moll, & Willburger, 2009; Mazzocco, Feigenson, & Halberda, 2011; Mussolin, Mejias, & Noël, 2010; Piazza et al., 2010). Brain imaging studies in children have shown both functional and structural IPS impairments in dyscalculia (Ashkenazi, Rosenberg-Lee, Tenison, & Menon, 2012; Kaufmann, Wood, Rubinsten, & Henik, 2011; Kaufmann et al., 2009; Kucian et al., 2006; Mussolin et al., 2010; Rotzer et al., 2008; Rykhlevskaia, Uddin, Kondos, & Menon, 2009). In the only study measuring nonsymbolic number representation in adults with dyscalculia, lower precision was found for both perception and estimation , and precision was correlated with arithmetical skills (Mejias, Grégoire, & Noël, in press).

An alternative (but not mutually exclusive) hypothesis is that there is an access deficit in linking symbolic and nonsymbolic number (Mundy & Gilmore, 2009; Rousselle & Noël, 2007) and that this deficit disrupts the normal tuning of numerosity representation over childhood (Noël & Rousselle, 2011). This is supported by the failure of several studies to find numerosity impairment in younger children with dyscalculia (De Smedt & Gilmore, 2011; Holloway & Ansari, 2009; Iuculano, Tang, Hall, & Butterworth, 2008; Landerl & Kölle, 2009; Price, Holloway, Räsänen, Vesterinen, & Ansari, 2007; Rousselle & Noël, 2007). However an alternative explanation is that these studies did not calculate numerosity acuity precisely enough, or used less stringent selection criteria for dyscalculia (see Mazzocco et al., 2011 for a discussion). More evidence is thus needed to distinguish between the number sense and access deficit hypotheses.

1.4. Dyslexia

Developmental dyslexia, or reading disability (RD), is defined similarly as an unexpected and severe difficulty reading, unexplained by general cognitive abilities (IQ), or inadequate teaching (American Psychiatric Association, 2000). There is strong evidence that dyslexia is hereditary, including the identification of several risk genes (Fisher & Francks, 2006; McGrath, Smith, & Pennington, 2006). Its prevalence is also similar; around 5–11% (Flannery, Liderman, Daly, & Schultz, 2000; Katusic, Colligan, Barbaresi, Schaid, & Jacobsen, 2001; Shaywitz, Shaywitz, Fletcher, & Escobar, 1990). Here we briefly review the cognitive and neural symptoms of dyslexia (for a comprehensive review see Peterson & Pennington, 2012).

Individuals with dyslexia are slow to match letter combinations to sounds using grapheme–phoneme correspondence rules (Coltheart, Rastle, Perry, Langdon, & Ziegler, 2001; Joubert et al., 2004). It is generally agreed that this is due to an underlying deficit in *phonological processing* (Démonet, Taylor, & Chaix, 2004; Snowling, 2000; Vellutino, Fletcher, Snowling, & Scanlon, 2004; Wagner et al., 1997). Phonological awareness or awareness of the individual phonemes in words (Høien, Lundberg, Stanovich, & Bjaalid, 1995) is one of the strongest predictors of reading ability (Castles & Coltheart, 2004; Wagner et al., 1997; Ziegler et al., 2010), and is clearly impaired in dyslexia (Boada & Pennington, 2006; Pennington, Van Orden, Smith, Green, & Haith, 1990; Torppa, Lyytinen, Erskine, Eklund, & Lyytinen, 2010). Some researchers have also proposed a deficit in *lexical access* (retrieval of words from memory; Vukovic & Siegel, 2006; Wolf & Bowers, 1999), exemplified by impaired rapid naming (RAN) of letters, digits, shapes, and objects.

Phonological and lexical deficits are both consistent with brain imaging studies showing impairment in perisylvian areas, including Broca's area (Georgiewa et al., 1999), the temporo-parietal region (Hoeft et al., 2006; Maisog, Einbinder, Flowers, Turkeltaub, & Eden, 2008; Meyler et al., 2007; Niogi & McCandliss, 2006; Pugh et al., 2001; Schlaggar & McCandliss, 2007; Shaywitz et al., 1998, 2002; Temple et al., 2001), and the superior temporal sulcus (Blau, van Atteveldt, Ekkebus, Goebel, & Blomert, 2009; Blau et al., 2010; Blomert, 2011). These areas are involved in phonological and lexical processing in normal readers (Turkeltaub,