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Reading deficits in schizophrenia and their relationship to developmental dyslexia: A review

Veronica Whitford ^{a,b,*}, Gillian A. O'Driscoll ^{c,d,e,f}, Debra Titone ^{c,g}

^a Department of Brain and Cognitive Sciences, McGovern Institute for Brain Research, Massachusetts Institute of Technology, 43 Vassar Street, Cambridge, MA 02139, United States

^b Graduate School of Education, Harvard University, 13 Appian Way, Cambridge, MA 02138, United States
^c Department of Psychology, McGill University, 1205 Doctor Penfield Avenue, Montreal, QC H3A 1B1, Canada

^d Department of Psychiatry, McGill University, 1033 Pine Avenue West, Montreal, QC H3A 1A1, Canada

c Douglas Mental Health University Institute, McGill University, 6875 LaSalle Boulevard, Verdun, QC H4H 1R3, Canada

^f Montreal Neurological Institute and Hospital, McGill University, 3801 University Street, Montreal, QC H3A 2B4, Canada

^g Centre for Research on Brain, Language and Music, McGill University, 3640 de la Montagne Street, Montreal, QC H3G 2A8, Canada

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ABSTRACT

Although schizophrenia and developmental dyslexia are considered distinct disorders in terms of clinical presentation and functional outcome, they both involve disruption in the processes that support skilled reading, including language, auditory perception, visual perception, oculomotor control, and executive function. Further, recent work has proposed a common neurodevelopmental basis for the two disorders, as suggested by genetic and pathophysiological overlap. Thus, these lines of research suggest that reading may be similarly impacted in schizophrenia and dyslexia. In this review, we survey research on reading abilities in individuals with schizophrenia, and review the potential mechanisms underlying reading deficits in schizophrenia that may be shared with those implicated in dyslexia. Elucidating the relationship between reading impairment in schizophrenia and dyslexia could allow for a better understanding of the pathophysiological underpinnings of schizophrenia, and could facilitate remediation of cognitive deficits that impact day-to-day functioning.

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1. Introduction

Schizophrenia is a psychiatric disorder characterized by disturbances in neurocognitive processes that impact nearly all areas of functioning, including language (Andreasen, 1979; Kuperberg, 2010a, 2010b; Kuperberg et al., 2005; Levy et al., 2010; Li et al., 2009; Spitzer, 1997). Indeed, an extensive body of research has reported schizophrenia-related deficits in both receptive and expressive language, at multiple levels (e.g., sub-lexical, lexical, sentence, discourse) and components (e.g., speech, semantics, syntax) of processing (e.g., Chen et al., 1994; Ditman and Kuperberg, 2007, 2010; Gouzoulis-Mayfrank et al., 2003; Kiang et al., 2008; Kuperberg, 2010a, 2010b; Kuperberg et al., 2006; Lelekov et al., 2000; Mathalon et al., 2010; Morice and McNicol, 1985; Passerieux et al., 1995; Ruchsow et al., 2003; Spitzer et al., 1994; Titone and Levy, 2004; Titone et al., 2000, 2002).

Although many of the above studies (and countless others) have used printed text to study language in schizophrenia, skilled reading

http://dx.doi.org/10.1016/j.schres.2017.06.049 0920-9964/© 2017 Elsevier B.V. All rights reserved. has surprisingly received relatively little attention until recently. The upsurge of interest in this area is driven by two key factors. First, the ability to read is essential for effective functioning in modern societies. Indeed, it is strongly linked to economic, occupational, and social success in both healthy individuals (e.g., Green and Riddell, 2007; Kirsch et al., 2002; Sticht, 1988) and individuals with mental illness, including schizophrenia (e.g., Gold et al., 2002; McGurk and Meltzer, 2000; Revheim et al., 2014). Understanding reading problems in schizophrenia has particular relevance for understanding challenges to patient functioning, and for pathways to remediation. For example, following prescription labels, medical appointment information, consent forms, transportation timetables, and so forth, all rely on intact reading abilities (see Carpenter et al., 2000; Christopher et al., 2007).

Second, there is increasing evidence of a common neurodevelopmental basis between schizophrenia and developmental dyslexia, also referred to as reading disorder, which is a specific and significant difficulty with reading that is not attributable to any major developmental delay or inadequate reading instruction (e.g., Ferrer et al., 2010; Snowling, 2000). A common neurodevelopmental basis between the two conditions is suggested by genetic and pathophysiological overlap (e.g., Becker et al., 2012; Condray, 2005; Horrobin et al., 1995; Jamadar et al., 2011; Leonard et al., 2008; Stefansson et al., 2014; Trulioff et al., 2017). For example, Leonard et al. (2008) found that

^{*} Corresponding author at: Department of Brain and Cognitive Sciences, McGovern Institute for Brain Research, Massachusetts Institute of Technology, 46-5081, 43 Vassar Street, Cambridge, MA 02139, United States.

E-mail addresses: vwhitfor@mit.edu (V. Whitford), gillian@psych.mcgill.ca (G.A. O'Driscoll), dtitone@psych.mcgill.ca (D. Titone).

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structural brain abnormalities implicated in dyslexia (e.g., reduced cerebral and temporal lobe volumes) were predictive of cognitive processing in schizophrenia, including reduced reading comprehension. Extending this work, Jamadar et al. (2011) found that a risk gene for dyslexia, namely DCDC2, accounted for important brain-volume variation in schizophrenia, particularly in regions implicated in reading (e.g., Broca's area, lingual gyrus, Wernicke's area). Further, Stefansson et al. (2014) conducted a population-wide study (N = 101,655) of rare copy number variants linked to schizophrenia and found that microdeletion at 15q11.2(BP1-BP2) was associated with poorer reading performance in the general population, even after controlling for I.Q. Using structural magnetic resonance imaging (MRI) in 15 non-clinical carriers of the deletion versus 201 healthy non-carriers, the authors also found significant reductions in the volumes of brain structures implicated in both schizophrenia and dyslexia (e.g., reduced gray matter in the anterior cingulate cortex and left insula, reduced white matter in both temporal lobes). Additionally, there is a growing body of work suggesting that alterations in left-right brain asymmetries may lead to neurodevelopmental disorders more generally, including dyslexia and schizophrenia more specifically (reviewed in Duboc et al., 2015; Paracchini et al., 2016; Trulioff et al., 2017).

Evidence of a common neurodevelopmental basis between schizophrenia and dyslexia is also suggested by a higher prevalence of reading impairment in the clinically well, first-degree relatives of patients with schizophrenia (e.g., Edmonstone, 1992; Erlenmeyer-Kimling et al., 1984; Fish, 1987; Marcus, 1974; Roberts et al., 2013), including increased rates of dyslexia in their unaffected offspring (Erlenmeyer-Kimling et al., 1984; Fish, 1987; Horrobin et al., 1995; Marcus, 1974). Further, there is some evidence of elevated schizotypal symptoms in dyslexia (e.g., Richardson, 1994; Richardson and Stein, 1993); these are attenuated or subclinical symptoms (i.e., formes frustes) of schizophrenia, such as perceptual aberrations (e.g., perceiving one's own voice as coming from a distance, having thoughts so loud that they can almost be heard, etc.).

A better understanding of reading deficits in schizophrenia is important for a number of reasons, and may be helped by leveraging what is known already about reading disorder. Specifically, a better understanding of this area could elucidate neurodevelopmental processes related to schizophrenia vulnerability, could be used to better identify the onset of a cognitive prodrome (as reading skills are routinely tracked years before the typical age of onset in schizophrenia), and, to the extent that schizophrenia and dyslexia have common bases, could lead to remediation strategies for reading in schizophrenia that improve patient functioning.

In sum, there is evidence of impaired reading in schizophrenia, and some evidence of shared pathophysiology between schizophrenia and dyslexia. In this review, we provide some background information on skilled reading, and then review the literature on reading impairment in schizophrenia. Finally, we consider neural and cognitive mechanisms of dyslexia to evaluate potential mechanisms that may contribute to the reading impairments in schizophrenia.

2. Background information on skilled reading

Many separate, yet interdependent perceptual, oculomotor, linguistic, and cognitive processes underlie skilled reading. At the most basic level, reading involves a series of rapid and ballistic eye movements, called *saccades*, which position printed material on the *fovea*, that is, the area of the retina with maximal visual acuity. Between saccades are brief pauses called *fixations*, during which visual information is extracted. Readers also extract some low-level information (e.g., word length, word shape, word spacing) beyond the fovea, that is, in the parafoveal region. This area of effective vision, called the *attentional* or *perceptual span*, generally extends 3–4 characters to the left and 14– 15 characters to the right of fixation in skilled readers of left-to-right orthographies, such as English (reviewed in Schotter et al., 2012). The perceptual span influences where people move their eyes during reading, and is modulated by text difficulty: more visual attention is allocated to the right of fixation when text is easier to process, and vice versa. Once readers perceive printed stimuli, they must decode their physical features and map them onto a particular orthographic structure. For example, a vertical line transected by a horizontal line represents the letter "t" in the English word "*teacher*". This orthographic information subsequently interacts with readers' lexical knowledge, including spellingto-sound correspondences (e.g., *teacher* = $ti/\xi_{\partial r}$), to allow for word recognition (reviewed in Liversedge et al., 2011; Radach and Kennedy, 2013; Rayner, 1997, 1998, 2009; Rayner et al., 2012; Whitford et al., 2016).

Leading models of word reading, including the Dual Route Cascaded model (e.g., Coltheart et al., 2001) and various triangle models (e.g., Harm and Seidenberg, 1999, 2004; Plaut et al., 1996; Seidenberg and McClelland, 1989) differ on the processes that guide word recognition (reviewed in Rayner and Reichle, 2010; Spivey et al., 2012). In particular, the Dual Route Cascaded model posits that word recognition is driven by grapheme-to-phoneme conversion rules, which involve mapping printed letters (i.e., graphemes) onto their corresponding speech sounds (i.e., phonemes), and by direct retrieval, which involves directly accessing a word's meaning from memory. The relative contribution of these two "routes" to word recognition varies as a function of wordlevel properties (e.g., whether a word is regularly- or irregularlyspelled: apple vs. yacht), and readers' familiarity with a word. In contrast, triangle models posit that word recognition is driven by spreading activation from orthographic input processing units to phonological output processing units within a multi-layer network, following a connectionist learning algorithm. Although these models differ in detail, both agree that lexical processing is hierarchical in nature: bottom-up orthographic processing precedes top-down semantic processing.

While the aforementioned models are centered on word recognition, readers rarely read words in isolation, but rather read words embedded in larger contexts, such as sentences and paragraphs. As such, readers typically engage in memory-dependent, post-lexical processing following word recognition, which includes construing meaning within the larger sentence or discourse context and revising understanding as a function of subsequent linguistic information (reviewed in Liversedge et al., 2011; Radach and Kennedy, 2013; Rayner, 1997, 1998, 2009; Rayner et al., 2012; Whitford et al., 2016).

3. Reading ability in schizophrenia

The literature on reading ability in schizophrenia can be divided into studies examining single-word reading in patients, studies examining connected text reading (i.e., sentences, paragraphs) in patients, and studies examining premorbid reading ability, that is, reading ability prior to illness onset.

3.1. Studies of single-word reading in schizophrenia

The most commonly used assessments of single-word reading in schizophrenia are the National Adult Reading Test (NART; Blair and Spreen, 1989; Nelson, 1982; Nelson and Wilson, 1991) and the Wide Range Achievement Test (WRAT; Jastak and Jastak, 1978; Jastak and Wilkinson, 1984; Wilkinson, 1993; Wilkinson and Robertson, 2006), which assess direct retrieval and word decoding abilities. Under the long-held assumption that reading is unaffected by brain dysfunction in schizophrenia, most studies that have used these measures have done so to match schizophrenia patients and controls on premorbid I.Q. Thus, by design, there were no between-group differences on these measures.

For example, in the one of the earliest studies, Dalby and Williams (1986) found that WRAT performance was comparable in schizophrenia patients and matched controls. A number of subsequent studies have similarly reported intact single-word reading (e.g., Crawford et al., 1992; Kremen et al., 1996; O'Carroll et al., 1992; Revheim et al.,

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