Schizotypy and false memory

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**Abstract**

Using the Deese–Roediger–McDermott (DRM) paradigm the present study examined the relationship between schizotypy and recognition memory. Participants scoring in the upper and lower quartile ranges for schizotypy (Schizotypal Personality Questionnaire brief version; SPQ-B) and on each of the SPQ-B subscales (cognitive-perceptual, interpersonal and disorganized) were compared on true and false memory performance. Participants scoring in the lower quartile range on the cognitive-perceptual subscale recognised a higher proportion of both true and false memories than those scoring in the higher quartile range. Participants scoring in the upper quartile on the interpersonal factor recognised fewer true items than those in the lower quartile range. No differences were found for overall schizotypy or on the disorganized subscale.

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1. Introduction

Factor analytical studies have identified a trisyndromic model of schizophrenia comprising disorganized, negative, and positive syndromes (Andreasen, Arndt, Alliger, Miller, & Flaum, 1995; Cuesta & Peralta, 1995; Daban et al., 2003, Peralta, De Leon, & Cuesta, 1992). Each of these syndromes has been linked to specific cognitive deficits (Fisher, Heller, & Miller, 2007; Gopal & Variend, 2005; Kuperberg & Heckers, 2000). Disorganized symptoms have been found to correlate with decreased attention-span, intelligence, and sensory-motor function (Basso, Nasrallah, Olson, & Bornstein, 1998). Negative symptoms have been associated with deficits in executive function, visual and verbal memory, and
working memory (Basso et al., 1998; Brown & White, 1992; Cuesta & Peralta, 1995). Whilst, deficits in semantic processing (Fisher et al., 2007), verbal memory (McDermid Vaz & Heinrichs, 2002), and context maintenance (Epstein, Stern, & Silbersweig, 1999; Fisher et al., 2007, Servan-Schreiber, Cohen, & Steingard, 1996) have been found to be related to positive symptoms.

Collectively, these findings indicate that schizophrenia significantly impairs cognitive function. Of particular interest to the current paper is the observation that schizophrenia has been found to disrupt memory processes (Aleman, Hijman, De Haan, & Kahn, 1999; Goldberg & Gold, 1995; McKenna, Clare, & Baddeley, 1995; Moritz, Woodward, Jelinek, & Kilnge, 2008; Stip, 1996). Generally, deficits in short-term memory, working memory, and declarative memory (episodic and semantic memory) have been reported among individuals with schizophrenia (Heinrichs & Zakzanis, 1998; Weiss & Heckers, 2001) and schizophrenic patients have demonstrated compromised recall and recognition relative to healthy participants (Moritz, Woodward, Cuttler, Whitman, & Watson, 2004). More specifically, studies have reported schizophrenia to be associated with diminished memory accuracy (Brébion, Amador, Smith, Sharif, & Gorman, 2000), reduced vividness of recall (Huron et al. 1995), and poor confidence judgments (Moritz & Woodward, 2006).

Unfortunately, the relationship between schizophrenia and memory is less certain than these findings suggest because of confounding variables, such as medication, treatment, and symptom severity (Mitropoulou et al., 2005). For this reason, researchers have frequently studied schizotypy (Cadenhead, Perry, Shafer, & Braff, 1999; Gooding & Braun, 2004; Raine & Lencz, 1995). Schizotypy is defined by the presence of a number of traits or symptoms, which resemble an attenuated form of schizophrenia (Raine et al., 1994). The continuum model (Kendler et al., 1991) proposes that schizotypy is non-clinical and a form of normal individual variation (Buchy, Woodward, & Liotti, 2007; Claridge & Beech, 1995). This model suggests that individuals in the general population exhibit schizotypal traits on a continuum ranging from normality (or health) (Sellen, Oaksford, & Gray, 2005) to schizophrenia at the extreme (Meehl, 1962, 1990).

The similar pathology of schizotypy and schizophrenia forms the basis of the full dimensional view of schizophrenia spectrum (Buchy et al., 2007; Claridge & Beech, 1995), where schizophrenia and schizotypy are considered to possess common dimensions; cognitive-perceptual aberrations, interpersonal deficits, and cognitive disorganisation (Buchy et al., 2007; Siever et al., 2002; Stefanis et al., 2002). Support for the full dimensional view is provided by the observation that many cognitive biases (e.g., knowledge corruption and attributional bias) associated with schizophrenia have been demonstrated in healthy individuals displaying schizotypy (Buchy et al., 2007). These findings suggest schizotypy research is likely to inform understanding of mnemonic deficits arising from schizophrenia without encountering confounds inherent within clinical populations.

Particularly, the current study will make use of the overlap between schizotypy and schizophrenia to explore the relationship between negative, positive, and disorganized symptoms and false memory. There are a number of reasons why research in this area is required. Firstly, studies using schizophrenic patients are typified by poor overall recall, a factor that is likely to reduce false memory, and potentially obscure the relationship between symptomology and false recall (Laws & Bhatt, 2005). Secondly, relatively little attention has been directed to studying the relationship between schizotypy and false memory (false positive memories) (Laws & Bhatt, 2005). Finally, the current study intends to extend the work of Laws and Bhatt (2005) and Dehon, Bastin, and Laroi (2008), who found that normal individuals scoring high in delusional ideation demonstrated poorer mnemonic performance in comparison to participants low in delusional ideation.

Laws and Bhatt (2005) investigated false memory using the Deese–Roediger–McDermott (DRM) false memory paradigm (Deese, 1959; Roediger & McDermott, 1995). The DRM involves participants being presented with lists of words. Each list contains a series of words (e.g., thread, pin, eye, sewing, sharp, point, prick, thimble, haystack, thorn, hurt, injection, syringe, cloth, knitting) that are strong associates of a non-studied item (e.g., ‘needle’). Typically, the DRM elicits false recall or recognition of non-presented associated items (Deese, 1959; Roediger & McDermott, 1995).

The production of non-presented associated items in the DRM may be explained by presented words activating non-presented associates via spreading associative activation (Fisher et al., 2007; Roediger & McDermott, 1995). This notion is supported by findings from several studies. For example, Roediger, Watson, McDermott, and Gallo (2001) found that the best predictor of both false recall and
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