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Review

Evidence of a dimensional relationship between schizotypy and schizophrenia: A systematic review

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ABSTRACT

The personality dimension of schizotypy is well established, and schizotypal traits can be taken to represent a proneness towards developing psychosis. Yet, there are competing theories about the latent structure of schizotypy. More specifically, there is controversy over the extent to which this propensity towards psychosis is present only in a small proportion of the population, or whether it is spread dimensionally throughout the general community. On the basis of accumulating research findings the present article argues for a fully dimensional model of schizotypy. It describes recent neurobiological, neuropsychological, social and environmental evidence supporting the idea that schizotypy in healthy populations, and disorders on the schizophrenia spectrum are fundamentally linked. Directions for further research are also considered.

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1. Introduction

Schizophrenia is a psychiatric disorder characterised by experiences such as hallucinations, delusions, personality changes, thought disorder, bizarre behaviour, impaired social interaction, and difficulties in carrying out daily activities (Compton and Broussard, 2009). These symptoms are usually subsumed under three main categories. They are; positive symptoms including hallucinations and delusions, disorganised symptoms including thought disorder and bizarre behaviour; and negative symptoms including alogia, apathy, and amotivation. Although these psychotic symptoms are most commonly associated with schizophrenia, they are also characteristic of other psychotic disorders like schizophreniform disorder, schizoaffective disorder, and delusional disorder. Symptoms of psychosis can also be seen in

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neurological disorders such as dementia, and in psychiatric disorders that are not generally considered under the rubric of psychotic disorders, for example severe depression.

Categorical diagnostic categories for mental health difficulties like schizophrenia are described in current nosological systems, such as the *Diagnostic System of Mental Disorders* (DSM-IV-TR; APA, 2000). They are useful in that they help to ensure disorders are easily identifiable; they serve as a basis for treatment decision-making; and they aid communication between clinicians (Livesley and Jackson, 1992; Kraemer et al., 2004).

However, there are also a number of criticisms of the assumption that psychotic disorders are truly categorical phenomena. For example, there is considerable hetereogenity within diagnostic categories (Wing and Agrawal, 2003; Beck et al., 2009), and homogeneity between them. Furthermore, diagnoses do not necessarily remain stable across individuals' lifetimes, and it is often not until a person has experienced multiple episodes and chronic disability that a psychotic diagnosis becomes stable and clear-cut (McGorry et al., 2009). Also, despite numerous studies into the physiological correlates of schizophrenia and related disorders, no biological markers, or endophenotypes have yet been discovered (Beck et al., 2009). There is no test (biological or otherwise) that will unequivocally distinguish someone with psychosis, from someone who is psychologically healthy (Wing and Agrawal, 2003; Wong and van Tol, 2003; Beck et al., 2009). This is reflected in Heinrichs' (2005) review of biological studies, which describes a substantial overlap between samples of people with schizophrenia, and samples of people who are psychologically healthy.

This overlap between clinical and non-clinical samples indicates that categorical diagnoses such as that of schizophrenia, decided upon by expert committee consensus rather than by nature, may obscure the true psychosis phenotype. This could reflect a misrepresentation of latent constructs, and may lead to erroneous diagnoses, inappropriate treatment, and conflicting research findings. Indeed, there is a growing consensus that dimensional views of schizophrenia and other psychotic disorders may be a more valid representation of the population distribution (van Os et al., 2009; Neuvo et al., 2012).

For similar reasons, traditional (categorical) understandings of schizotypy have also met with theoretical criticism. Schizotypy describes a cluster of personality traits that include odd or bizarre behaviour, strange speech, magical thinking, unusual perceptual experiences, and social anhedonia. There is some disagreement regarding the underlying factor structure of schizotypy (Stefanis et al., 2004; Mason and Claridge, 2006; Fonseca-Pedrero et al., 2011). However, the prevailing understanding is that it is comprised of three identifiable factors, which broadly correspond to the positive, negative and disorganised dimensions of schizophrenia (Wuthrich and Bates, 2006; Fonseca-Pedrero et al., 2011). The first factor is the 'cognitive-perceptual factor', which includes magical thinking, unusual perceptual experiences, ideas of reference and paranoia (Raine, 1991, 2006). The second is the 'interpersonal factor', which includes constricted affect, social anxiety, lack of close personal relationships, and suspiciousness (Raine, 1991, 2006). The final 'disorganised factor' includes odd behaviour and odd speech (Raine, 1991, 2006).

Recent reviews summarising research into schizotypy have examined a range of topics associated with the construct. Examples include unusual speech associated with schizotypy (Kiang, 2010); the assessment of schizotypy (Fonseca-Pedrero et al., 2008); affective traits in schizophrenia and schizotypy (Horan et al., 2008); schizotypal personality disorder and schizophrenia (Siever and Davis, 2004); as well as cannabis use, psychosis and schizotypy (Compton et al., 2007). Much of this research is based on theoretical assumptions arising from two competing models of the distribution of schizotypy in the general population–call the *quasi-dimensional* and *fully dimensional* approaches respectively. However, these models are often taken at face value, and evidence for them has not recently been critically scrutinised. The present review sought to resolve this by examining recent empirical taxometric, neuropsychological, environmental and biological evidence pertaining to the potential dimensional relationship between schizophrenia and schizotypy in otherwise psychologically healthy populations. Some methodological issues prevalent in the area will also be considered, as well as directions for future research.

For the sake of brevity, the present review includes findings pertaining to schizophrenia and schizotypal personality traits. However reflecting the aforementioned difficulties, controversies and complexities inherent in conducting research into categorical psychiatric constructs, we do acknowledge that the findings herein can be also be common to other clinical phenomena; such as schizoaffective disorder (e.g. Cheniaux et al., 2008), first-episode psychosis (e.g. Mesholm-Gately et al., 2009), bipolar disorder and mania (e.g. Berrettini, 2000; Bramon and Sham, 2001); and other personality constructs such as psychoticism (e.g. Mason and Claridge, 2006). It is for this reason, we prefer to use the terms 'schizotypy' and 'schizophrenia' in a relatively broad sense.

2. The quasi-dimensional approach

The *quasi-dimensional approach* to schizotypy is based on a disease model of mental illness. It posits that schizotypy is a personality organisation specific to a small group of individuals within the population (approximately 10%), who are labelled 'schizotypes' (Rado, 1953; Meehl, 1990; Lenzenweger, 1994; Beauchaine et al., 2008). They are compared to a larger group (approximately 90%) who are not at risk. As reviewed by Lenzenweger (2006), the model can be attributed to Meehl's (1962) theory; which described a specific genetic vulnerability towards developing psychosis. This vulnerability was said to exist in the form of a genetic predisposition, manifesting as a neurointegrative defect called schizotaxia. According to Meehl (1962), schizotaxia is necessary but not sufficient to cause schizophrenia. Rather than causing schizophrenia directly, it interacts with environmental influences throughout a person's lifetime to determine the degree of decompensation they experience (Lenzenweger, 2006). From this perspective, genetic vulnerability towards developing psychotic symptoms is considered to be 'taxonic' (Korfine and Lenzenweger, 1995; Waller et al., 2006). The approach is quasi-dimensional only because it refers to levels of expression of a proposed disease process. Otherwise it is a discontinuous, categorical theory wherein any one individual is considered to either possess a genetic vulnerability, or they do not.

Support for the quasi-dimensional approach can be found in studies that employ taxometric analyses (Waller and Meehl, 1998; Rawlings et al., 2008b). Taxometric analyses are a group of statistical procedures that are used to determine the underlying structure of latent constructs. They are able to determine whether a given construct is categorical (taxonic), or dimensional. In 2008, a review of 19 published taxometric studies pertaining to schizotypy reported that fifteen supported a categorical model (Rawlings et al., 2008b). Furthermore the taxonic base rate estimates in these studies, ranging between 0.03 and 0.13, appeared to support Meehl's (1990) postulation that 10% of the population are schizotypes (Rawlings et al., 2008b).

Yet there are criticisms of the quasi-dimensional model, and some of these focus particularly on the sampling methods used when conducting taxometric analyses (Rawlings et al., 2008b). For instance, taxometric studies have often used either clinical samples with insufficient power for the purposes of taxometrics, or they have used large student samples, which may not be representative of the general population (Rawlings et al., 2008b). Additionally,

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