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Original Article

Autistic-like and schizotypal traits in a life history perspective: diametrical associations with impulsivity, sensation seeking, and sociosexual behavior

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ABSTRACT

According to recent theoretical models, autistic-like and schizotypal traits can be regarded as opposite sides of a single continuum of variation in personality and cognition, and may be diametrically associated with individual differences in life history strategies. In this view, schizotypy is a psychological phenotype oriented toward high mating effort and reduced parenting, consistent with a fast life history strategy, whereas autistic-like traits contribute to a slow strategy characterized by reduced mating effort and high parental investment. In this study, we tested the hypothesis that autistic-like and schizotypal traits would be diametrically associated with unrestricted sociosexuality, impulsivity, and sensation seeking (three key behavioral correlates of fast life history strategies in humans) in a sample of 152 young adults (18–38 years). The results were consistent with a diametrical autism–schizotypy axis of individual variation. In line with our hypotheses, autism–schizotypy scores were uniquely associated with individual differences in impulsivity, sensation seeking, and sociosexual behavior, even after controlling for variation in Big Five personality traits. However, we found no significant associations with sociosexual attitude in the present sample. Our findings provide additional support for a life history model of autistic-like and schizotypal traits and demonstrate the heuristic value of this approach in the study of personality and psychopathology.

1. Introduction

Initially described as milder manifestations of psychopathology (Meehl, 1962; Wing, 1988), autistic-like and schizotypal traits stand at the boundary between normal and disordered variation. Autistic-like traits – also known as the "broader autistic phenotype" – comprise reduced social/communicative skills, narrow interests and repetitive behaviors, and heightened attention to patterns and details; they are elevated in patients with autism spectrum disorders (ASDs) as well as in their relatives (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001). Mirroring the distinction between positive and negative symptoms in schizophrenia, schizotypal traits include both *positive schizotypy* – a tendency to experience unusual cognitive and perceptual phenomena, magical ideation, and reference/paranoid thoughts – and *negative schizotypy* – traits of social anxiety, social withdrawal, and constricted affect. Measures of schizotypy usually include a third dimension labeled *disorganization* that reflects odd or eccentric patterns of speech and behavior. Schizotypal traits are elevated in relatives of psychotic patients and constitute a risk factor for schizophrenia spectrum disorders (SSDs; Claridge, 1997; van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009). Both autistic-like and schizotypal features show moderate to substantial heritability, with estimates in the .50–.70 range (Ericson, Tuvblad, Raine, Youjng-Wolff, & Baker, 2011; Hoekstra, Bartels, Verweij, & Boomsma, 2007; Ronald, Larsson, Anckarsäter, & Lichtenstein, 2011).

While autistic-like and schizotypal traits can be associated with pathological outcomes, they are also found outside the clinical spectra of ASDs and SSDs, and are increasingly recognized as important dimensions of normal personality variation. Indeed, accumulating empirical findings show that moderate amounts of autistic and schizotypal features may confer desirable and potentially adaptive traits such as creativity, enhanced perceptual and spatial skills, and even artistic and scientific talent (e.g., Baron-Cohen, Ashwin, Aswin, Tavassoli, & Chakrabarti, 2009; Fletcher-Watson et al., 2012; Happé & Vital, 2009; Kyaga et al., 2011; Nettle & Clegg, 2006; Stevenson & Gernsbacher, 2013). Intriguingly, there is evidence that autistic-like and schizotypal traits may be understood as functionally opposite sides of a single overarching continuum, as postulated in the *diametrical model* developed by Crespi and Badcock (2008).

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1.1. The diametrical model of autism and psychosis

The relation between autism spectrum disorders (ASDs) and schizophrenia spectrum disorders (SSDs) has been debated for the better part of a century (see Crespi, 2011; Crespi & Badcock, 2008). At the descriptive level, the two clusters of disorders show a number of overlapping features, including social discomfort, reduced social skills, and impaired or dysfunctional mindreading. In addition, several genes and chromosome regions have been implicated in the etiology of both ASDs and SSDs (e.g., Carroll & Owen, 2009; see Crespi, Stead, & Elliot, 2010). The phenotypic overlap with autism is stronger for negative symptoms of schizophrenia such as blunted affect, poverty of speech, and anhedonia than for positive symptoms such as delusions, hallucinations, and thought disorganization.

According to the diametrical model of autism and psychosis (Crespi & Badcock, 2008; see Crespi et al., 2010; Dinsdale, Hurd, Wakabayashi, Elliot, & Crespi, 2013), the commonalities between ASDs and SSDs are mostly superficial; the apparent phenotypic similarities between autistic features and negative psychotic symptoms actually reflect the action of largely *opposite* causal factors. On this view, ASDs and SSDs represent opposite pathological extremes in the development of the human social brain. The autistic extreme of the autism-psychosis continuum is characterized by high levels of *mechanistic cognition* (visuospatial abilities, cause–effect inference) and low levels of *mentalistic cognition* (communication deficits, reduced empathy and social understanding). In contrast, psychosis is characterized by high mentalistic and low mechanistic abilities; in SSDs, hyper-mentalizing – expressed in traits such as paranoid ideation, exaggerated sensitivity to nonverbal cues, and over-responsiveness to gaze – is characteristically associated with poor visuospatial abilities and failures in logical reasoning (Crespi & Badcock, 2008; Zhai et al., 2011). For different reasons, both profiles may result in a pattern of reduced social skills, dysfunctional mindreading, and high levels of social anxiety. In addition, the negative features of premorbid schizophrenia are liable to be misdiagnosed as autistic disorders in younger patients who later go on to develop SSDs, thus contributing to inflate the apparent diagnostic overlap between the two spectra (Crespi, 2011).

Further support for a diametrical relation between ASDs and SSDs comes from the divergent patterns of brain and body development associated with the two spectra. While the autism spectrum is marked by early overgrowth (e.g., high birth weight and length, large brain volume, fast childhood growth), psychosis correlates with reduced growth, especially during prenatal and early postnatal development (Crespi & Badcock, 2008). Finally, when the same genes are implicated in both ASDs and SSDs, the relevant genetic and/or epigenetic effects often show opposite functional profiles in the two spectra. For example, different mutations of the same gene may determine up-regulation of a molecular pathway in ASDs and down-regulation in SSDs; in other cases, ASDs and SSDs are associated with opposite patterns of copy number variation (more versus fewer copies of a genetic region), opposite methylation patterns (hyper- versus hypo-methylation), and so forth (see Crespi & Badcock, 2008; Crespi et al., 2010; Gilman et al., 2012; see also Kalkman, 2012). In particular, Crespi and Badcock (2008) reviewed evidence that ASDs are associated with over-expression of paternally expressed imprinted genes (i.e., genes that are differentially expressed depending on a chromosome's parent of origin) and/or under-expression of maternally expressed genes, while SSDs tend to show the opposite expression pattern.

It is important to stress that, in this model, not *all* etiological factors are assumed to operate in a diametrical fashion. For example, deleterious mutations that affect neural integrity and developmental insults such as infections and nutritional deficits are likely to act as non-specific risk factors for both kinds of disorders (Crespi, 2011; Crespi et al., 2010; see also Keller & Miller, 2006).

1.2. The diametrical model and the structure of trait variation

The logic of the diametrical model is not restricted to diagnosable disorders, but extends to normative individual variation in autistic-like and schizotypal traits. According to the model, autistic-like traits and schizotypy represent opposite sides of a mechanistic–mentalistic continuum, with ASDs and SSDs as pathological extremes (Crespi & Badcock, 2008; Dinsdale et al., 2013). For example, Brosnan, Daggar, and Collomosse (2010) found that a profile of high empathizing and low systemizing specifically predicted the occurrence of positive symptoms in a female sample. Russell-Smith, Maybery, and Bayliss (2010) showed that autistic-like and positive schizotypal traits had diametrical associations with performance on a perceptual task involving local visual processing. In another study, Russell-Smith, Bayliss, Maybery, and Tomkinson (2013) tested the association between autistic-like and schizotypal traits and various measures of mechanistic and mentalistic cognition, with mostly null results. However, the analytic strategy employed by these authors (comparing matched groups of $N = 20$ extracted from a larger sample) suffers from low statistical power and a high likelihood of Type II errors.

In non-clinical samples, questionnaire measures of autistic-like and schizotypal traits show moderate positive correlations with one another. This pattern is explained by the large statistical overlap that exists between negative schizotypy and the interpersonal facet of autistic-like traits (Del Giudice, Angeleri, Brizio, & Elena, 2010; Dinsdale et al., 2013; see also Russell-Smith, Maybery, & Bayliss, 2011). However, such overlap is likely to be at least in part spurious, reflecting vague formulation of questionnaire items rather than true phenotypic similarity (discussed in Del Giudice et al., 2010).

When the overlap with negative schizotypy is statistically controlled for, measures of positive schizotypy and autistic-like traits become approximately orthogonal (reviewed in Del Giudice et al., 2010; Dinsdale et al., 2013), suggesting a two-dimensional structure rather than a single bipolar continuum.[1] This, however, is only one possible interpretation of the data. In a recent study, Dinsdale et al. (2013) applied principal component analysis (PCA) to a mixture of scales assessing autistic-like and schizotypal traits. The first unrotated component captured the common variance attributable to general manifestations of social isolation, impairment, and/or anxiety (as well as shared method variance due to item similarity). After the first component was extracted, the second component showed a clear-cut diametrical structure, with opposite loadings from scales measuring positive schizotypy and autistic-like traits (Fig. 1c). As can be seen in Fig. 1c, negative schizotypy and disorganization have smaller loadings on the bipolar factor, thus falling in the middle of the autistic-schizotypal continuum. This finding was replicated in a previously published dataset by Wakabayashi, Baron-Cohen, and Ashwin (2012); see Dinsdale et al., 2013 and Fig. 1d.

In fact, questionnaire data are equally consistent with two mathematically equivalent psychometric structures: (a) a bipolar autism–schizotypy factor coupled with an orthogonal unipolar factor of social difficulty; and (b) two orthogonal unipolar factors of autistic-like traits (plus negative schizotypy) and positive schizotypy (plus disorganization). As shown in Fig. 1, the latter is readily obtained as an orthogonal rotation of the former. Whereas unrotated solutions tend to recover a general factor followed by a number of smaller bipolar factors, standard rotation algorithms (e.g., Varimax) are designed to break down general and bipolar factors to approximate a "simple structure", whereby each of the variables tends to load highly on some of the factors and have small loadings on the other factors (see Darton, 1980; Russell, 2002). Although the simple structure criterion can be a

[1] In this context, the term "bipolar" is used to describe a factor or component characterized by sets of opposite-sign loadings, as is customary in the psychometric literature; it does not imply any connection with symptoms or disorders in the bipolar spectrum.