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Original Article

Developmental Instability and Markers of Schizotypy in University Students

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Abstract: Fluctuating asymmetries (FA) and minor physical anomalies (MPAs) are markers of developmental instability (DI), an index of the degree to which an organism was subject to genomic or environmental stress during development. Measures of DI are characteristic of schizophrenia and are thought to reflect an underlying genetic liability for schizophrenia spectrum disorders. Whereas MPAs reflect developmental stress relatively early in the first trimester in utero, skeletal FAs reflect developmental stress throughout the lifespan. Both measures were collected to provide some indication of the associated developmental time course. In addition to DI measures, several psychometric measures of schizotypy were administered in a sample of university students (n = 81). It was hypothesized that increased DI may relate to schizotypal symptoms in a group of healthy undergraduate students. Schizotypy scores were positively correlated with FA, but not MPAs. This finding suggests that DI, as indexed by FA, is important for normal range variation in schizotypal characteristics, just as it is important for normal range variation in intelligence. Second, considered in the context of studies demonstrating that schizophrenia is associated with elevated MPAs, these results suggest that developmental stress likely occurs earlier in development for schizophrenia than schizotypy.
Introduction

Yeo, Gangestad, and Thoma (2007) recently proposed a two-factor theory of the etiology of schizophrenia spectrum disorders based upon an evolution-based model of developmental instability. The authors proposed that a possible mediator and moderator of genetic and environmental influences on schizophrenia spectrum disorders is developmental instability (DI). DI concerns the accuracy with which genotypes are translated into phenotypes (Møller and Swaddle, 1997). It has been defined as propensity to experience “developmental noise” (Adams and Niswander, 1967; Waddington, 1957), or the effects of genetic or environmental stresses (e.g., mutations, pathogens, toxins) during development to which an organism is not fully adapted. This results in the imprecise expression of developmental design (Lerner, 1954; Ludwig, 1932; Parsons, 1990). Two commonly measured markers of DI in humans include fluctuating asymmetry (FA), defined as deviation from bilateral symmetry on traits that are typically symmetric at a population level (Van Valen, 1962), and phenodeviant physical traits, or minor physical anomalies (MPAs).

In the Yeo et al. (2007) model, developmental instability represents a general vulnerability factor that is shared across developmental disorders. Each disorder also has a set of unique genetic (and perhaps environmental) causes. For example, multiple studies have shown increased MPAs in patients with schizophrenia when compared to normal control subjects (for a review, see Murphy and Owen, 1996). In addition, dermatoglyphic fluctuating FA appears to be increased in twins with schizophrenia compared to the unaffected co-twin (e.g., Markow and Gottesman, 1989), suggesting that early events in gestational development interact with other genetic and environmental sources of influence to predispose the disorder. Other studies have linked the presence of schizophrenia to prenatal exposure to influenza virus and cold temperatures (Brown et al., 2004; Venables, 1996), suggesting a neurodevelopmental role for a specific physiological stressor in the development of schizophrenia.

“Schizotypy” is a term commonly used to describe a continuum of correlated personality traits that include a tendency to report unusual sensory experiences, hold odd or sometimes paranoid beliefs, and show less interest in and enjoyment from social interactions. Mild cognitive difficulties are often reported in schizotypic individuals, representing in mild form some of the attention deficits encountered in schizophrenia. Meehl’s influential formulation (1990) specifically drew attention to the relationship between schizotypy and schizophrenia. He suggested that schizotypy or “schizoid taxon” denote individuals who carry the predisposing gene pattern for schizophrenia, but this genotype is not fully expressed as schizophrenia. This genetic predisposition is phenotypically expressed as specific personality traits, cognitive slippage, soft neurological signs, and “soft psychometric signs” (Meehl, 1990).
The aim of the current study was to investigate associations between normal variation in DI and expression of these soft psychometric signs.

Several recent studies have investigated the role of markers of DI in the etiology of schizotypy. One recent study, using a single dermatoglyphic FA measure, found it to be positively correlated with scores on the Social Anhedonia Scale in males, but not in females (Rosa et al., 2000). Other recent studies demonstrated that groups of college students identified as schizotypic exhibit more dermatoglyphic anomalies than control groups (Chok, Kwapi, and Scheuermann, 2005; Chok and Kwapi, 2005). As no previous studies have comprehensively investigated a relationship between measures of skeletal fluctuating asymmetry and schizotypy, we chose to investigate the role of developmental instability in the etiology of schizotypy via a comprehensive measurement of DI indicators. In an unselected sample of university students, we investigated whether individuals scoring relatively higher on schizotypy scales would have increased FA and MPAs. We predicted positive correlations between markers of DI and schizotypy.

Materials and Methods

Participants

Sixteen males and forty-four females ranging in age from 17 to 49 years were recruited from the University of New Mexico (UNM) Psychology Department subject pool and were given credits toward psychology classes for participation in the experiment. Twenty-one males, ranging in age from 19 to 28, were recruited by posters on the University of Utah (U of U) campus (total n = 81). University of Utah participants were paid $16 per hour for their participation in several lab projects, including the current project. All potential participants were screened for history of neurological disorder or disease, serious medical illness or disease, learning disability, or head injury. Using a subset of questions derived from the SCID-I for DSM-III, potential participants were also screened for history of significant mood disorders, psychotic disorders and anxiety disorder. Before all testing procedures, participants were informed of potential risks related to participation and signed a consent form describing procedures and advising them of their right to leave the study at any time.

Procedures

Measurement of FA and MPAs

We measured the right and left sides of seven bilateral traits: ear length, ear width, elbow width, hand width, wrist width, ankle width, and foot width. FA for a given structure was calculated by taking the absolute value of the difference between the left and right sides, divided by one-half the sum of left plus right sides (individual $FA = |R - L| / [0.5 \times (R + L)]$; Yeo, Gangestad, Thoma, Shaw, and Repa, 1997; Yeo, Gangestad, Edgar, and Thoma, 1999). These traits generally show very little directional asymmetry (Furlow, Armijo-Prewitt, Gangestad, and Thornhill, 1997). In our sample, only hand width deviated from symmetry. Directional asymmetry (DA) exists when a trait is consistently larger on one side of the body than another across a population. The most straightforward means of accounting for DA is simply to subtract mean DA from individual asymmetry values (Palmer and Stroebeck, 1986). Correction for directional bias was implemented by first
calculating deviation from the population average hand asymmetry for each individual and then subtracting that average from each individual’s directional asymmetry (Palmer, 1994). FA measures for the seven individual traits were summed into a total composite measure, which we simply refer to as FA.

To assess MPAs, we used the procedures outlined in the 1989 revision of the Waldrop scale (Waldrop, Halverson, and Shetterly, 1989; see also Waldrop, Pedersen, and Bell, 1968). The measures consisted of observed head circumference, fine electric hair, hair whorls, inter-ocular distance, height of ears, structure of the ear lobes, steeped palate, tongue furrows, curvature of the fifth finger, and abnormalities of the toes. A score for MPAs was then derived from a point system based upon the presence and degree of each of these body features.

Schizotypy test battery

Several measures of schizotypy were combined into a single questionnaire. The Schizoid Taxon Scale (STS, Golden and Meehl, 1979) is a 7-item measure whose items were selected using a taxometric procedure. The items concern a variety of schizotypic symptoms, including cognitive slippage, social anhedonia, and ambivalence. The Magical Ideation Scale (MIS, Eckblad and Chapman, 1983) is a measure of “a belief...of the possibility that events which, according to the causal concepts of this culture, cannot have a causal relation with each other” (p. 217). Examples of item content include belief that others can read one’s mind, belief that certain numbers have special powers, and sensing an evil presence. The Social Anhedonia Scale (SAS; Mishlove and Chapman, 1985) is a measure of shut-in, schizoid, and avoidant behavior and of lack of pleasure derived from social relationships. Both the MIS and SAS have been shown to predict later onset of schizophrenia (Chapman, Chapman, Kwapil, and Eckblad, 1994).

Analysis

A principal components analysis was conducted to construct a measure associated with variance shared by all three measures. The first principal component accounted for 51% of the total variance in the individual measures. Individual variables had loadings of .86 (STS), .71 (MIS), and .53 (SAS). A first principal component of schizotypy has been proposed as a general schizotypy measure (Williams and Irwin, 1991). In our analyses, we refer to this measure as Schizotypy. Sex and height were partialled out of all correlations, since body size may be associated with measured FA (Palmer, 1994).

Results

There were no significant correlations between MPAs and measures of Schizotypy. However, FA correlated significantly and positively with Schizotypy ($r = .23, p = .03$), the Magical Ideation Scale ($r = .26, p = .02$), and with Golden and Meehl’s STS ($r = .19, p = .05$). No significant relationship was found for the Social Anhedonia Scale ($r = .08, p = .26$; see Table 1). There were no significant interactions between sex or height and DI in predicting measures of Schizotypy.
Table 1. Partial Correlations between Schizotypy and DI Measures*

| Variables                              | FA    | MPAs  |
|----------------------------------------|-------|-------|
| Schizotypy                             | .23   | -.03  |
|                                        | *p=.03| *p=.42|
| Golden and Meehl Schizoid              | .19   | .05   |
| Taxon Scale                            |       |       |
|                                        | *p=.05| *p=.35|
| Magical Ideation Scale                 | .24   | -.08  |
|                                        | *p=.02| *p=.24|
| Social Anhedonia Scale                 | .10   | -.04  |
|                                        | *p=.20| *p=.39|

*Controlling for the effect of sex and height.  
Note: FA = Fluctuating Asymmetry and MPA = Minor Physical Anomalies.

It should be noted that although the obtained correlations represent relatively small effect sizes, but they may substantially underestimate the magnitude of the relationship between schizotypy and the underlying trait of FA. Gangestad and Thornhill (1999) have estimated that the FA composite used in the current study correlates about .5 with underlying DI and, hence, correlations with FA underestimate correlations with DI by a factor of about two. Table 2 shows a summary of inter-scale correlations.

Table 2. Correlations between the Schizotypy Measures

| Variables                              | STS   | MIS   | SAS   |
|----------------------------------------|-------|-------|-------|
| Schizotypy (1st PC)                    | .86   | .71   | .53   |
| Schizoid Taxon Scale (STS)             |       |       |       |
| Magical Ideation Scale (MIS)           |       |       | .30   |
| Soc Anhedonia Scale (SAS)              |       |       |       |

Note. n = 81. For all correlations, p < .01 with the exception of MIS and SAS correlation for which p < .05. STS = Schizoid Taxon Scale, MIS = Magical Ideation Scale, and SAS = Social Anhedonia Scale.

Since the Chapman scales (i.e., Magical Ideation and Social Anhedonia Scales) were designed as taxon scales, a further analysis was done to investigate how many of the
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Subjects in this sample may have met the suggested criteria for inclusion in the “schizotypy taxon.” Three of the 81 subjects reached criteria for schizotypy as assessed with the Magical Ideation Scale, and four reached criteria for schizotypy using the Social Anhedonia Scale. There were no significant differences between these subjects and the rest of the sample on DI measures or on any demographic variables.

To investigate the possibility that motivational differences may exist between samples drawn from UNM (compensated with class credits) and U of U (compensated at $16 per hour), group differences on Schizotypy scale scores were investigated. Of the three Schizotypy taxon scales, there was a group difference on one; the U of U group had lower scores on the Magical Ideation Scale ($p = .002$). No other comparisons approached the level of significance. It seems unlikely that this difference was secondary to motivational issues because it is doubtful that motivation would affect one schizotypy taxon scale and not others. To test whether the DI-schizotypy relationships generalized across samples, group differences in Pearson correlations were investigated (Cohen, 1988). None of the group comparisons between correlations approached the level of significance, indicating no correlational differences between the UNM and U of U groups.

Discussion

Fluctuating asymmetry (FA) correlated positively with test scores on instruments assessing schizophrenia proneness in a sample of normal, undergraduate university students. There are two important implications of this association between schizotypy scores and FA. The first is that DI, as indexed by FA, is important for normal range variation in schizotypy, just as it is important for normal range variation in intelligence (Furlow et al., 1997; Prokosch, Yeo, and Miller, 2005; Thoma, Yeo, Gangestad, Halgren, Sanchez and Lewine, 2005). Hence, the genetic factors linked with DI, including mutation load and epistatic interactions among genes (Leamy and Klingenberg, 2005; Yeo et al., 1999), might be relevant for normal variation in schizotypy scores. Second, the current results are relevant for our understanding of the role of DI in schizophrenia, suggesting that increased MPAs and FA in schizophrenia do not result from some feature of the disorder per se (e.g., lifestyle influences or medication effects) and likely index a broad underlying vulnerability to schizophreniform disorders.

In contrast, MPAs, another marker of DI, were not related to schizotypy in the current study, although MPAs have long been associated with schizophrenia. Whereas MPAs reflect slowed or disrupted development in the first or second trimester, skeletal FA reflects less of a “time-locked” developmental abnormality and actually increases during adulthood (Gangestad and Thornhill, 1999). The current data suggest that schizotypy is associated with developmental perturbations later in development and not necessarily early prenatal perturbations. Some studies have found increased MPAs in schizotypal adolescents (Weinstein, Diforio, Schifffman, Walker, and Bonsall, 1999) and in unaffected relatives of schizophrenics (Ismail, Cantor-Graae, and McNeil, 2000) suggesting that further research is needed to investigate the temporal specificity of each category of DI measure and possibly to delineate the timing of DI associated events in each disorder. Of possible relevance, a recent study did not show a schizophrenia group to have increased skeletal FA (Edgar et al., 2006). Hence, though both schizophrenia and schizotypy are
disorders characterized by increased DI, the current findings suggest that the impact of DI on the developing brain occurs somewhat later in those with schizotypal characteristics.

These findings thus suggest a novel interpretation of the distinction between schizophrenia and schizotypy. The timing, rather than the nature of neurodevelopmental stresses, may determine whether an individual’s genotype is expressed as schizophrenia or schizotypy. Both schizophrenia and schizotypy appear to be characterized by an increased vulnerability factor, indexed by DI, as well as an increased dose of unknown specific and unique etiologic factors for schizophreniform disorders. Theoretically, schizophrenia might entail a greater loading of the disorder-specific factor, the general vulnerability factor, or both. Direct comparison in a single study of FA levels in schizotypal personality disorder versus schizophrenia would be especially informative, but we are aware of no such studies.

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