



## Developmental instability in social anhedonia: An examination of minor physical anomalies and clinical characteristics

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### ABSTRACT

Developmental instability (DI) refers to the inability of the developing brain to buffer the effects of genetic and environmental insults. This concept has been invoked to better understand how fetal brain development goes awry in schizophrenia and related spectrum disorders. This study examined one marker of DI, minor physical anomalies (MPAs), and its association with a putative indicator of schizotypy, the trait of social anhedonia. MPAs and clinical symptoms were assessed within a community sample of psychometrically identified individuals high in social anhedonia and a matched group of healthy controls. Results indicated that, compared to the controls, MPAs were elevated in the social anhedonia group. Additionally, within the social anhedonia group, MPAs were significantly correlated with clinical ratings of schizoid personality disorder characteristics and also showed strong associations with schizotypal personality disorder ratings. These findings indicate a relationship between developmental anomalies and negative schizotypy and suggest that, when combined with psychometrically identified risk, the presence of MPAs may further elevate the probability of clinical manifestations of schizophrenia-spectrum characteristics.

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### 1. Introduction

Schizophrenia and related spectrum disorders such as schizotypy have been viewed from a neurodevelopmental perspective (e.g., Cannon, 1998; Weinberger, 1987; Walker, 1994). This developmental conceptualization proposes that genetic and environmental factors interact to determine fetal brain development and later risk for schizophrenia. In attempting to understand how development may go awry in schizophrenia and other disorders, developmental instability has been invoked as an important concept (Yeo et al., 2007). Developmental instability (DI) refers to an individual's

inability to buffer the effects of genetic and environmental stressors on development. The DI model is rooted in the assumption that the ability of an organism to precisely carry out its genetic “design” is an imprecise, epigenetic process. Increased developmental stability acts to “buffer” development, allowing the organism to express its genotype precisely even in the face of adverse environmental conditions (Waddington, 1957; Jantz and Webb, 1980a,b). On the other hand, DI is thought to result in increased developmental imprecision and “noise” in developmental pathways (Yeo et al., 2007). One result of DI is that environmental stressors may have more of an impact on the individual (Yeo et al., 2007).

DI is measured through the assessment of proxy measures or markers of this developmental disturbance (Yeo et al., 2007). Putative markers of DI include minor physical anomalies (MPAs) as well as dermatoglyphic abnormalities. MPAs are morphologic anomalies that are evident in the craniofacial region (e.g., flat or prominent forehead, wide-

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spaced eyes, asymmetrical ears, narrow or steeped palate) and limbs (e.g., webbed toes). Dermatoglyphic abnormalities can include deviations from symmetry on bilateral traits (e.g., palmar ridge counts) that are symmetric in the general population (Yeo et al., 2007). Deviation from symmetry is thought to represent developmental error either resulting from genetic or environmental disruption of growth (Yeo and Gangestad, 1998).

There is accumulating evidence that, while certainly not unique to schizophrenia, markers of DI are present at higher levels in schizophrenia than in nonpsychiatric control groups (e.g., Compton et al., 2007; Edgar et al., 2006; Green et al., 1989a,b; Lane et al., 1997). With regard to MPAs, in a recent review Compton and Walker (2009) concluded that an accumulation of studies has found MPAs in schizophrenia and that these results are consistent with the neurodevelopmental model of schizophrenia (see also meta-analyses on MPAs by Weinberg et al., 2007). Similarly, in a review of studies on dermatoglyphic anomalies Chok et al. (2005) found that such anomalies were more prevalent in schizophrenia than in controls (with some exceptions: Cantor-Graae et al., 1998; Rosa et al., 2000).

Given the above findings in schizophrenia, researchers have begun to extend research on neurodevelopmental anomalies to the study of schizotypal traits. Schizotypal traits may represent phenotypic manifestations of genetic liability for schizophrenia (Meehl, 1962). The use of these traits in psychometric high-risk paradigms may afford opportunities to study correlates of schizotypy (Blanchard et al., *in press*; Chapman et al., 1994; Gooding et al., 2005; Kwapil, 1998) prior to illness onset and free of complicating factors including medication exposure.

A small number of studies have now demonstrated an association between MPAs, dermatoglyphic anomalies and psychometrically identified positive (as indexed by traits such as perceptual aberrations and magical ideation) and negative (relating to physical and social anhedonia) schizotypy traits in nonclinical samples (Barrantes-Vidal et al., 2002; Chok et al., 2005; Chok and Kwapil, 2005; Daly et al., 2008; Rosa et al., 2000; Thoma et al., 2008). However, there are important limitations to the existing literature. First, although some studies suggest that indices of developmental deviance are related to anhedonia (Daly et al., 2008) and that negative schizotypy is more strongly related to such anomalies than positive schizotypy (Chok and Kwapil, 2005; Rosa et al., 2000) these findings are not consistent across methods and studies. Results vary across measures of DI (e.g., Daly et al., 2008) and there is some evidence that associations between developmental deviance and negative schizotypal traits are most pronounced in males (Daly et al., 2008; Rosa et al., 2000). Further, these developmental deviance findings are not consistent across studies as one study failed to show an association between anhedonia and DI (Thoma et al., 2008). Thus, replication focusing on anhedonia is clearly required.

A second limitation with the existing research is that, although MPAs are important indicators of developmental deviance and are present in schizophrenia (Compton and Walker, 2009), only one published study has examined MPAs in psychometrically assessed schizotypy traits (Thoma et al., 2008). Other studies have focused on various dermatoglyphic anomalies and it is important to determine if these findings generalize to MPAs.

A third concern is that none of the published studies on DI and psychometrically identified schizotypy has directly assessed clinical symptoms of schizophrenia-spectrum personality disorders. This is important as not all putative schizotypes will develop clinical symptoms and it would be informative to determine what factors may account for this heterogeneity in outcomes. It may be that greater DI contributes to greater clinical severity within psychometrically identified high-risk samples (i.e., enhancing risk stratification, Compton and Walker, 2009). Relatedly, since DI is not specific to schizophrenia-spectrum disorders (Compton and Walker, 2009; Lloyd et al., 2008; Yeo et al., 2007) it would be informative to demonstrate that obtained group differences in developmental deviance are actually associated with clinically rated characteristics of personality pathology associated with schizophrenia-spectrum disorders.

A final limitation with the existing literature is that the majority of studies on developmental deviance and schizotypal traits have been conducted in college samples (Chok et al., 2005; Chok and Kwapil, 2005; Daly et al., 2008; Thoma et al., 2008). Given the potential lack of representativeness of high functioning college students, it would be informative to determine if findings of DI and schizotypy can be replicated in community samples.

To address the above issues, this study sought to examine the incidence of MPAs in a community sample of individuals high in social anhedonia and matched controls. It was hypothesized that MPAs would be more prevalent in social anhedonics compared to controls. We also examined the association between MPAs and clinical ratings of schizophrenia-spectrum personality disorder characteristics. We hypothesized that within the social anhedonia group, MPAs would be related to greater clinical severity as measured by dimensional personality disorder characteristics of schizotypal, schizoid, and paranoid personality disorder. Finally, based on prior findings that DI is more associated with negative schizotypal characteristics in males (Daly et al., 2008; Rosa et al., 2000) we explored gender effects within the analyses.

## 2. Methods

### 2.1. Sample

The present study recruited community participants from the larger ongoing Maryland Longitudinal Study of Schizotypy (MLSS; Blanchard et al., *in press*). The MLSS is a study focusing on social anhedonia as a putative indicator of schizotypy (Meehl, 1962) and the risk for development of schizophrenia and related spectrum disorders. Prior publications on this sample have examined clinical (Blanchard et al., *in press*), cognitive (Cohen et al., 2006) and behavioral (Collins et al., 2005) features in probands as well as parental characteristics of participants within the MLSS (Cohen et al., *in press*; Emmerson et al., 2009).

Full details of the MLSS recruitment methodology can be found in Blanchard et al. (*in press*). Briefly, the MLSS participants were drawn from a larger community sample of 2434 18- to 19-year-olds recruited by the UMCP Survey Research Center using random digit dial methods. The participants were mailed a consent form and a screening

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