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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.

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 steepled 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

questionnaire that included the Revised Social Anhedonia Scale (SocAnh Scale; Eckblad et al., 1982), Perceptual Aberrations Scale (PerAb; Chapman et al., 1976), and Magical Ideation Scale (MagicID; Eckblad and Chapman, 1983). A validity scale (The Infrequency Scale; Chapman and Chapman, 1976) was embedded within the screening questionnaire, and individuals who endorsed three or more items in the unexpected direction were excluded from the study (as in Chapman and Chapman, 1976).

A subset of the full MLSS social anhedonia participants ($n=36$) was recruited to participate in this study focusing on additional assessments of minor physical anomalies. In the full MLSS study, two methods were used to identify participants with extreme scores on social anhedonia. The first method involved identifying individuals at least 1.9 standard deviations above SocAnh Scale mean. The second selection method involved using the taxometric method of maximum covariate analysis (MAXCOV-HITMAX; Waller and Meehl, 1998). Individuals with Bayesian probabilities greater than or equal to .50 were assigned to the social anhedonia taxon group (see Blanchard et al., 2000; Horan et al., 2004 for a review of this methodology). There were no statistically significant differences between subjects identified through these two procedures for any of the dependent variables examined in the overall MLSS study (Blanchard et al., in press). In either selection method, scores on the PerAb and MagicID scales were not considered in forming the social anhedonia group.

A subset of the full MLSS control participants ($N=41$) was recruited to participate in this study. The control group consisted of individuals without elevated scores on the SocAnh Scale, operationalized as a SocAnh score .50 standard deviations below the gender and race group derived mean and a Bayesian probability of taxon membership below .50. An additional inclusion criterion specified that control participants not score higher than .50 standard deviations above the mean on the PerAb or MagicID scales of psychosis proneness.

2.2. Measures

2.2.1. Minor physical anomalies

Minor physical anomalies (MPAs) were assessed using the Manual for Assessing Minor Physical Anomalies (Waldrop et al., 1989), which sets forth guidelines for determining the presence and degree of a variety of MPAs. These include wide-spaced eyes, low-seated ears, single transverse palmer crease, and anomalies of the fingers and toes. This manual is the most popular assessment tool for MPAs (Yeo et al., 1993). It has been used in many other studies of MPAs, including both those of individuals at genetic risk for schizophrenia, for whom it may be associated with clinical history and outcome (e.g., Schiffman et al., 2002), and those of normal college-age individuals (e.g., Yeo et al., 1997). MPAs as measured by Waldrop and colleagues' manual have been shown to remain stable from birth (Waldrop, 1975). Inter-rater reliability for the overall MPA score has been found to be high (e.g., .70; Waldrop et al., 1968). Raters were graduate students trained using the Waldrop Manual. All MPA assessments were conducted in the same laboratory room providing consistent lighting for purposes of physical assessment. Discussion of the scoring criteria and group ratings on practice subjects were carried out prior to rating actual subjects in order to ensure agreement about the criteria. In addition, regular discussions about any difficulties in ratings took place during supervision, and periodic checks were conducted in order to ensure ongoing agreement. MPA ratings were conducted blind to group status and clinical ratings.

2.2.2. Schizophrenia-spectrum symptom ratings

The International Personality Disorders Examination (IPDE; Loranger et al., 1995) is a semi-structured interview which yields both categorical and dimensional ratings of Axis II disorders (each symptom item is rated on a 3-point scale: 0 = not present; 1 = subthreshold; 2 = threshold). The IPDE was administered to assess schizoid, schizotypal, and paranoid personality disorders, consisting of items related to unusual thinking or beliefs, unusual perceptual experiences, suspicious or paranoid ideation, inappropriate or constricted affect, odd or eccentric behavior or appearance, impaired social relationships, and social anxiety. A number of studies have used the IPDE for the assessment of schizophrenia-spectrum disorders in putatively psychosis-prone individuals (e.g., Chapman et al., 1994; Kwapil et al., 2002; Horan et al., 2007). All diagnostic interviewers were Masters-level doctoral students under the supervision of a licensed clinical psychologist with extensive training and experience in clinical assessment (JJB). A consensus meeting was used to discuss the diagnostic material (ratings and notes), issues raised in an independent video review, and to consult the video tape for further clarification. Final symptom severity ratings and diagnostic decisions were determined within the consensus meeting. IPDE ratings were not available for one control participant ($N=40$).

3. Results

Demographic characteristics are presented in Table 1. The groups did not differ in sex X^2 (1, 77) = .019, $p>.05$, or race, X^2 (4, 77) = 7.76, $p>.05$.

Group ratings of MPAs are presented in Table 2. A group × sex ANOVA was conducted to examine group differences. The main effect of group was significant, $F(1, 73)=4.34$, $p=.04$, $d=.45$. However, the main effect of sex $F(1, 73)=.55$, and the group by sex interaction $F(1, 73)=.001$, was not

Table 1
Demographic characteristics in control ($n=41$) and social anhedonia ($n=36$) groups.

	Control	Social anhedonia
	n (%)	*n* (%)
Sex		
Male	20 (48.8%)	18 (48.7%)
Female	21 (51.2%)	19 (51.4%)
Race		
White	26 (63.4%)	19 (51.4%)
Black	10 (24.4%)	16 (43.2%)
Asian	1 (2.4%)	1 (2.7%)
Hispanic	3 (7.3%)	1 (2.7%)
Other	1 (2.4%)	0 (.0%)

Table 2
Descriptive statistics for minor physical anomalies and personality disorder dimensional ratings.

	Control	Social anhedonia
	Mean (SD)	Mean (SD)
Minor physical anomalies	1.83 (1.75)	2.68 (1.99) *
Schizoid personality disorder	.24 (.62)	1.49 (2.09) ***
Schizotypal personality disorder	.32 (.69)	1.03 (1.42) **
Paranoid personality disorder	.34 (.88)	1.08 (1.75) *

* $p<.05$.
** $p<.001$.
*** $p<.0001$.

significant, $ps>.05$. The social anhedonia group had significantly higher MPAs than did the control group.

Group differences in clinical characteristics within the full MLSS sample have been reported elsewhere (Blanchard et al., in press). Personality disorder characteristics were evaluated within the current sample (see Table 2) with separate group × sex ANOVAs conducted on each IPDE spectrum dimensional score. A significant main effect of group for schizoid dimensional scores was obtained, $F(1, 73)=13.75$, $p=.0001$, $d=.81$. However, the main effect of sex and the sex × group interaction was not significant, $Fs(1, 73)=.55$, .001, respectively, $ps>.05$. For schizotypal dimensional scores, there was a main effect for group $F(1, 73)=12.10$, $p=.001$, $d=.64$. The main effect for sex and the sex × group interaction was not significant, $Fs(1, 73)=1.75$, .002, respectively, $ps>.05$. Finally, a similar pattern emerged for paranoid dimensional scores, with a significant main effect for group, $F(1, 73)=4.60$, $p=.04$, $d=.53$, with social anhedonia participants having higher scores than control participants. However, the main effect of sex and the sex × group interaction for paranoid scores was not significant, $Fs(76)=$ .07, .18, respectively, $ps>.05$. In summary, consistent with findings from the full MLSS sample, group differences in personality characteristics were found across the IPDE dimensional scores with the social anhedonia group obtaining higher pathological ratings compared to controls.

Correlations between MPAs and IPDE dimensional scores within the Social Anhedonia group are presented in Table 3. MPAs were significantly correlated with schizoid dimensional ratings ($r=.39$, $p=.02$). The correlation between MPAs and schizotypal dimensional ratings was not significant ($r=.29$, $p=.09$) but the magnitude of the correlation approximated a medium effect size (Cohen, 1992). Paranoid dimensional scores were not correlated with MPAs ($r=.09$, $p=.58$). In order to examine if sex of participants contributed to these relationships, partial correlations were conducted. After controlling for sex, correlations between MPAs and personality dimensional scores were essentially unchanged for schizoid ($pr=.39$, $p=.03$), schizotypal ($pr=.30$, $p=.08$), and paranoid ($pr=.10$, $p=.57$) dimensional scores.

Table 3
Correlations between minor physical anomalies (MPAs) and personality disorder dimensional ratings in the social anhedonia group ($N=36$).

Dimensional scores	MPAs
Schizoid	.39 [a]
Schizotypal	.29 [b]
Paranoid	.09

[a] $p<.05$.
[b] $p=.09$.

4. Discussion

The present study sought to examine whether developmental instability was associated with social anhedonia, a putative indicator of risk for schizophrenia and related spectrum disorders. Results indicated that MPAs were higher in the social anhedonia group compared to the control group. This finding is consistent with the proposal that neurodevelopmental aberrations may contribute to the spectrum of schizophrenia liability (Compton and Walker, 2009; Yeo et al., 2007). These results are also consistent with prior findings that other markers of developmental instability are associated with negative schizotypy (Chok et al., 2005; Daly et al., 2008; Rosa et al., 2000) and extend these findings to MPAs.

The association between MPAs and clinical characteristics was examined within the social anhedonia group. As predicted, greater MPAs within this group were significantly correlated with schizoid personality dimensionality ratings. The correlation with schizotypal ratings was not significant, but a medium effect size was obtained. No relationship was found between MPAs and paranoid personality dimensional ratings. These results suggest that schizotypal and schizoid characteristics may have stronger associations with DI than paranoid characteristics. This latter finding was not predicted and caution is warranted in the interpretation of this unexpected result. Overall, these findings are interesting in that they suggest that in association with psychometrically identified risk, the presence of MPAs may further elevate the probability of clinical manifestations of spectrum disorders. This would appear to be consistent with Compton and Walker's (2009) suggestion that DI may enhance risk stratification. These findings require further replication but suggest that the joint use of psychometric measures of schizotypy and indicators of developmental deviance may yield an especially vulnerable high-risk group. Ultimately, longitudinal designs would be required to test this conjecture, but initial validation could come from cross-sectional studies assessing symptom and functional correlates of DI in psychometrically identified individuals.

Prior research has yielded inconsistent findings with regard to the role of sex in the association between schizotypy and measures of DI. Some studies have found that this association is particularly pronounced in males (Daly et al., 2008; Rosa et al., 2000); however, the present study found no main effects or interactions for sex in MPAs or in personality dimensional scores. Further, sex made no contribution to the correlational findings between MPAs and personality scores within the social anhedonia group. The discrepancy in findings may relate to sample differences in prior studies which used college samples compared to the community sample in the current study. Additionally, most prior studies of psychometrically identified schizotypy have not included MPAs but have focused on other indices of developmental deviance such as dermatoglyphics. Caution is warranted in interpreting the current results given the modest sample size and limited power to detect small gender differences. However, the current null findings indicate the need to further assess the role of sex in DI and schizotypy.

A limitation to the present findings is that only MPAs were addressed. There are other indicators of DI including fluctuating

asymmetries that are found in dermatoglyphics features. Although these various indicators of developmental disturbance are presumed to reflect the same underlying process, findings would suggest that these indicators may be differentially related to schizotypy (e.g., Daly et al., 2008). Thus, caution is warranted in extending the present results with MPAs to other markers of DI. Additionally, different markers of DI may represent anomalies occurring at different periods of development (Yeo et al., 2007) and thus may provide information regarding what period appears to be most related to schizotypy. Finally, neurological soft signs have been used as broad indicators of developmental deviance (e.g., Barrantes-Vidal et al., 2002; Bollini et al., 2007) and were not evaluated in this study. Future studies should include broader assessments of developmental abnormalities to further examine which markers may be most informative in understanding heightened risk in putative schizotypes.

The present findings are limited in that they are cross-sectional. Although there is an accumulation of evidence suggesting that social anhedonia identifies individuals who are at risk for schizophrenia-spectrum personality disorders (Gooding et al., 2005; Kwapil, 1998), it is unclear to what extent participants in this cross-sectional study will ultimately develop schizophrenia-spectrum disorders. Further, the current cross-sectional results with young adults cannot address the conjecture that elevated MPAs will ultimately contribute to longitudinal clinical outcomes during adulthood.

A final interpretive caution concerns the specificity of the current findings. The two-factor DI model of Yeo et al. (2007) proposes that DI is related to a variety of neurodevelopmental disorders other than schizophrenia (e.g., ADHD and dyslexia). Compton et al. (2009) have also noted that, although the study of MPAs in other disorders has been limited, elevated MPAs have been found in psychiatric disorders other than schizophrenia. For example, MPAs have been found to be elevated in affective psychosis (Lloyd et al., 2008). Thus, the mere elevation of DI within any psychometrically identified group does not allow conclusions regarding specific diagnostic outcomes. It would be informative to examine other symptom domains and risk groups to determine the specificity of findings obtained with psychometric measures of schizotypy.

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Contributors

Jack Blanchard and Minu Aghevli designed the study, wrote the protocol, and ran the study. Jack Blanchard and Minu Aghevli conducted statistical analyses. All authors contributed to literature reviews. Jack Blanchard and Minu Aghevli wrote initial drafts of the manuscript. Marsha Sargeant and Amy Wilson contributed to subsequent drafts with writing, editing, and final formatting. All authors contributed and have approved the final manuscript.

Conflict of interest

All authors declare that there are no conflicts of interest to disclose.

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References

Barrantes-Vidal, N., Fanana, L., Rosa, A., Caparros, B., Riba, M.D., Obiols, J.E., 2002. Neurocognitive, behavioral and neurodevelopmental correlates of schizotypy clusters in adolescents from the general population. Schizophr. Res. 61 (2–3), 293–302.

Blanchard, J.J., Collins, L.M., Aghevli, M., Leung, W.W., Cohen, A.S., in press. Social anhedonia and schizotypy in a community sample: The Maryland Longitudinal Study of Schizotypy. Schizophr. Bull. doi: 10.1093/schbul/sbp107.

Blanchard, J.J., Gangestead, S., Brown, S., Horan, W., 2000. Hedonic capacity revisited: a taxometric analysis of social anhedonia. J. Abnorm. Psychol. 109 (1), 87–95.

Bollini, A.M., Compton, M.T., Esterberg, M.L., Rutland, J., Chien, V.H., Walker, E.F., 2007. Associations between schizotypal features and indicators of neurological and morphological abnormalities. Schizophr. Res. 92, 32–40.

Cannon, T.D., 1998. Neurodevelopmental influences in the genesis and epigenesist of schizophrenia: an overview. Appl. Prev. Psychol. 7, 47–62.

Cantor-Graae, E., Ismali, B., McNeil, T.F., 1998. Neonatal head circumference and related indices of fetal development in schizophrenic patients. Schizophr. Res. 32 (3), 191–199.

Chapman, L. J., Chapman, J. P., 1976. The Infrequency Scale: Unpublished test.

Chapman, L., Chapman, J., Kwapil, T., Eckblad, M., Zinser, M., 1994. Putatively psychosis-prone subjects 10 years later. J. Abnorm. Psychol. 103 (2), 171–183.

Chapman, L., Chapman, J., Raulin, M., 1976. Scales for physical and social anhedonia. J. Abnorm. Psychol. 85 (4), 374–382.

Chok, J.T., Kwapil, T.R., 2005. Extralimital triradii as a putative marker of schizotypy. Schizophr. Res. 76 (2–3), 239–245.

Chok, J.T., Kwapil, T.R., Scheuermann, A., 2005. Dermatoglyphic anomalies in psychometrically identified schizotypic young adults. Schizophr. Res. 72 (2–3), 205–214.

Cohen, J., 1992. A power primer. Psychol. Bull. 112, 155–159.

Cohen, A.S., Emmerson, L.C., Mann, M.C., Forbes, C.B., Blanchard, J.J., in press. Schizotypal, schizoid and paranoid characteristics in the biological parents of social anhedonics. Psychiatry Res.

Cohen, A.S., Leung, W.W., Saperstein, A.M., Blanchard, J.J., 2006. Neuropsychological functioning and social anhedonia: results from a community high-risk study. Schizophr. Res. 85 (1–3), 132–141.

Collins, L.M., Blanchard, J.J., Biondo, K.M., 2005. Behavioral signs of schizodia and schizotypy in social anhedonics. Schizophr. Res. 78 (2–3), 309–322.

Compton, M.T., Bollini, A.M., Mack, L.M., Kryda, A.D., Rutland, J., Weiss, P.S., Cercu, Z., Esterberg, M.L., Walker, E.F., 2007. Neurological soft signs and minor physical anomalies in patients with schizophrenia and related disorders, their first-degree biological relatives, and non-psychiatric controls. Schizophr. Res. 94 (1), 64–73.

Compton, M.T., Walker, E.F., 2009. Physical manifestations of neurodevelopmental disruption: are minor physical anomalies part of the syndrome of schizophrenia? Schizophr. Bull. 35 (2), 425–436.

Daly, M.P., Gooding, D.C., Jessen, H.M., Auger, A.P., 2008. Indicators of developmental deviance in individuals at risk for schizophrenia. Schizophr. Res. 101 (1–3), 152–160.

Eckblad, M., Chapman, L., 1983. Magical ideation as an indicator of schizotypy. J. Consult. Clin. Psychol. 51 (2), 215–225.

Eckblad, M., Chapman, L., Chapman, J., Mishlove, M., 1982. The Revised Social Anhedonia Scale. Unpublished test, University of Wisconsin, Madison, WI.

Edgar, J.C., Yao, R.A., Gangestad, S.W., Davis, J.T., Cañive, J.M., Lewine, J.D., Blake, M.B., 2006. Reduced auditor M100 asymmetry in schizophrenia and dyslexia: applying a developmental instability approach to assess atypical brain asymmetry. Neuropsychologica 44 (2), 289–299.

Emmerson, L.C., Miller, S.L., Blanchard, J.J., 2009. Behavioral signs of schizoidia and schizotypy in the biological parents of social anhedonics. Behav. Modif. 33 (4), 452–473.

Gooding, D.C., Tallent, K.A., Matts, C.W., 2005. Clinical status of at-risk individuals five years later: further validation of the psychometric high-risk strategy. J. Abnorm. Psychol. 114 (1), 170–175.

Green, M., Satz, P., Gaier, D., Ganzell, S., Kharabi, F., 1989a. Minor physical anomalies in schizophrenia. Schizophr. Bull. 15 (1), 91–99.

Green, M., Satz, P., Smith, C., Nelson, L., 1989b. Is there atypical handedness in schizophrenia? J. Abnorm. Psychol. 98 (1), 57–65.

Horan, W.P., Brown, S.A., Blanchard, J.J., 2007. Social anhedonia and schizotypy: the contribution of individual differences in affective traits, stress, and coping. Psychiatry Res. 149 (1–3), 147–156.

Horan, W.P., Blanchard, J.J., Gangestad, S.W., Kwapil, T.R., 2004. The psychometric detection of schizotypy: do putative indicators identify the same latent class? J. Abnorm. Psycho. 113 (3), 339–357.

Jantz, R., Webb, R., 1980a. Dermatoglyphic asymmetry as a measure of canalization. Ann. Hum. Biol. 7 (5), 489–493.

Kwapil, T., 1998. Social anhedonia as a predictor of the development of schizophrenia-spectrum disorders. J. Abnorm. Psychol. 107 (4), 558–565.

Kwapil, T.R., Mann, M.C., Raulin, M.L., 2002. Psychometric properties and concurrent validity of the Schizotypal Ambivalence Scale. J. of Nerv. Ment. Dis. 190 (5), 290–295.
Jantz, R., Webb, R., 1980b. Dermatoglyphic asymmetry as a measure of canalization. Ann. Hum. Biol. 7 (5), 489–493.
Lane, A., Kinsella, A., Murphy, P., Byrne, M., Keenan, J., Colgan, K., Cassidy, B., Sheppard, N., Horgan, R., Waddington, J.L., Larkin, C., O'Callaghan, E., 1997. The anthropometric assessment of dysmorphic features in schizophrenia as an index of its development origins. Psychol. Med. 27 (5), 1155–1164.
Lloyd, T., Dazzan, P., Dean, K., Park, S., Fearon, P., Doody, G., Tarrant, J., Morgan, K., Morgan, C., Hutchinson, G., Leff, J., Harrision, Gl, Murray, R., Jones, P., 2008. Minor physical anomalies in patients with first-episode psychosis: their frequency and diagnostic specificity. Psychol. Med. 38, 71–77.
Loranger, A., Andreoli, A., Berger, P., Buchheim, P., Channabasavanna, S., Coid, B., Dahl, A., Diekstra, R., Ferguson, B., Jacobsberg, L., Janca, A., Mombour, W., Pull, C., Ono, Y., Regier, D., Sartorius, N., Sumba, R., 1995. The International Personality Disorder Examination (IPDE) Manual. World Health Organization, New York.
Meehl, P., 1962. Schizotaxia, schizotypy, schizophrenia. Am. Psychol. 17 (12), 827–838.
Rosa, A., van Os, J., Fananas, L., Barrantes, N., Caparros, B., Guiterrez, B., Obiols, J., 2000. Developmental instability and schizotypy. Schizophr. Res. 43 (2–3), 124–134.
Schiffman, J., Ekstrom, M., LaBrie, J., 2002. Minor physical anomalies and schizophrenia-spectrum disorders: a prospective investigation. Am. J. Psychiatry 159 (2), 238–243.
Thoma, R.J., Gangestad, S.W., Euler, M.J., Lysne, P.A., Monnig, M., Yeo, R.A., 2008. Developmental instability and markers of schizotypy in university students. Evol. Psychol. 6 (4), 586–594.
Waddington, C., 1957. The strategy of the genes. MacMillan, New York.
Walker, E.F., 1994. Neurodevelopmental precursors of schizophrenia. In: David, A.S., Cutting, J.C. (Eds.), The Neuropsychology of Schizophrenia. The Neuropsychology of Schizophrenia. Lawrence Erlbaum Associates, Inc, Hillsdale, NJ, England, pp. 119–129.
Waldrop, M., 1975. "Manual for assessing minor physical anomalies." Unpublished manuscript, Laboratory of Developmental Psychology, National Institute of Mental Health, Bethesda, MD.
Waldrop, M., Halverson, C., Shetterly, H., 1989. Manual for the Assessment of Minor Physical Anomalies. (Rev. Ed.). Unpublished manuscript, University of Georgia (Athens).
Waldrop, M., Pederson, F., Bell, R., 1968. Minor physical anomalies and behavior in preschool children. Child Dev. 39 (2), 391–400.
Waller, N.G., Meehl, P.E., 1998. Multivariate taxometrics procedures. Sage, Thousand Oaks, California.
Weinberg, S.M., Jenkins, E.A., Marazita, M.L., Maher, B.S., 2007. Minor physical anomalies in schizophrenia: a meta-analysis. Schizophr. Res. 89 (1–3), 72–85.
Weinberger, D.R., 1987. Implications of normal brain development for the pathogenesis of schizophrenia. Arch. Gen. Psychiatry 44, 660–669.
Yeo, R., Gangestad, S., 1998. Developmental instability and phenotypic variation in neural organization. In: Raz, N. (Ed.), The other side of the error term: aging and development as model systems in cognitive neuroscience. St. Elsevier Science, New York, pp. 1–51.
Yeo, R., Gangestad, S., Thoma, R., Shaw, P., Repa, K., 1997. Developmental Instability and cerebral lateralization. Neuropsychology 11 (4), 552–561.
Yeo, R., Gangestad, S., Daniel, W., 1993. Hand preference and developmental instability. Psychobiology 21 (2), 161–168.
Yeo, R., Gangestad, S., Edgar, C., Thoma, R., 2007. Developmental instability and individual variation in brain development: implications for the origin of neurodevelopmental disorders. Curr. Dir. Psychol. Sci. 16 (5), 245–249.