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Fluctuating Dermatoglyphic Asymmetries in Youth at Ultrahigh-risk for Psychotic Disorders

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Abstract

Fluctuating dermatoglyphic asymmetry represents one specific class of minor physical anomaly that has been proposed to reflect prenatal insult and vulnerability to psychosis. However, very little is known about fluctuating dermatoglyphic asymmetry in youth showing symptoms of ultrahigh risk (UHR) for psychosis. Using high-resolution photographs of fingerprints and clinical interviews, the UHR group in this study showed greater fluctuating dermatoglyphic asymmetry compared to controls; however, this was not further linked to symptomatology. The results of this study provide an important perspective on potential biomarkers and support neurodevelopmental conceptions of psychosis.

Keywords

psychosis; ultrahigh-risk; dermatoglyphics; fluctuating asymmetry; neurodevelopment

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Conflict of Interest

None.

Contributors

Authors O.D.F.R., V.A.M. and D.J.D. developed the study concept. V.A.M. obtained funding for the study. All authors contributed to the study design. Testing, data collection as well as data analysis and interpretation were performed by O.D.F.R. and L.I. under the supervision of V.A.M. and D.J.D. O.D.F.R. drafted the paper under the supervision of D.J.D. L.I. and V.A.M. provided the critical revisions. All authors approved the final version of the paper for submission.

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1. Introduction

A large body of research indicates that genetics and prenatal insult are both significant contributors to vulnerability for psychosis (Mittal et al., 2008a). One proposed measure that taps into both processes is dermatoglyphics; the patterns of ridge folds on the fingers, palms and soles of feet that form during the second trimester (Bramon et al., 2005). As finger and handprints are formed from the same germinal layer as the brain, asymmetries in the fingers may also reflect insult to early prenatal brain development (Compton and Walker, 2009).

Fluctuating dermatoglyphic asymmetry, characterized by different ridge patterns or counts between homologous fingers, may be linked to heritable factors (Golembo-Smith et al., 2012). Cross sectional studies of family members with a relative or twin with schizophrenia and young adults with schizotypal personality disorder provide substantial evidence for fluctuating asymmetry in those at risk for psychosis (Gabalda and Compton, 2010; Markow and Gottesman, 1989; Mittal et al., 2012; Rosa et al., 2005). Given that dermatoglyphics represent an intersection between the intrauterine environment and genetic factors, these features hold significant promise for biomarker development. Indeed, one notable prospective investigation observed that high-risk family members of persons with schizophrenia, who also go on to develop a psychotic disorder, have simpler fingerprint patterns when compared to healthy persons and high risk persons who do not develop psychosis (Langsley et al., 2005).

Despite a large body of research pointing to fluctuating dermatoglyphic asymmetry as an etiological marker of risk for psychosis, very little is known about whether fluctuating asymmetry is present in individuals at imminent risk for psychosis such as those with ultrahigh-risk (UHR) syndromes. Finding reliable risk factors for this population is particularly important as a significant proportion will go on to develop psychosis in a short period (Cannon et al., 2008). Indeed, phenotypic markers that allow for early identification of individuals at UHR can help to reduce the duration of untreated psychosis and inform decisions about targeted intervention implementation. While altered dermatoglyphics do not pinpoint a specific prenatal event or genetic contribution; this line of research holds several advantages that may be useful in risk-calculator development (Cannon, 2010). Specifically, dermatoglyphics are readily quantifiable in non-invasive and inexpensive fashion, they provide continuous data, and they also are not affected by rater bias or retrospective bias in comparison to methods of self-report for evaluating the prenatal environment (McIntosh et al., 2002). Further, innovations in print scanning provide a way to clearly examine dermatoglyphics without the complications of ink or colorless ink methods.

In this study, clinical symptoms and dermatoglyphic asymmetries were evaluated in UHR participants and healthy controls. We predicted that the UHR group would exhibit greater fluctuating dermatoglyphic asymmetry when compared with controls and that these asymmetries would be associated with elevated symptoms.

2. Materials and Methods

2.1 Participants

Participants (n=51 UHR, n=45 Control) between the ages of 12 and 21 (Mean age=18.20, SD=2.39) were recruited by Internet advertising, email postings, newspaper ads, and community professional referrals. Exclusion criteria included history of head injury, the presence of a neurological disorder, and lifetime substance dependence. The presence of an Axis I psychotic disorder was an exclusion criterion for UHR participants. The presence of a psychotic disorder in a first-degree relative or meeting for an Axis I disorder was an exclusionary criterion for controls. The Institutional Review Board approved the protocol and informed consent procedures.

2.2 Procedure: Clinical interviews

UHR individuals met criteria for a prodromal syndrome based on the Structured Interview for Prodromal Syndromes (SIPS) (Miller et al., 2003). A total sum score for the positive and negative symptom domain was calculated. The Structured Clinical Interview for Axis-I DSM-IV Disorders (SCID) (First et al., 1995) was also administered to rule out a psychotic disorder diagnosis.

2.3 Dermatoglyphics

Fingerprints were obtained by utilizing digital scans of the actual hand from a high-definition photo scanner (Epson Perfection V550 Photo Scanner). Coders rated each finger dermatoglyphic by using Adobe Photoshop (CS3), which allowed high-level zoom/enlargement and demarcation. Because of this technology, the prints were of very high quality, and consequently it was not necessary to code partial prints. Employing a widely adopted procedure described by Holt (Holt, 1958), the number of dermal ridges crossing a line drawn between the center of the pattern (core) and the triradius of each fingerprint was counted for all fingers on both hands. The number of ridges on each finger of the right hand was subtracted from the number of ridges on the homologous finger of the left hand. The total ridge count asymmetry score was calculated by summing the absolute values of the differences observed for each homologous finger pair. Participants were excluded from the analysis if more than one finger pair lacked a valid ridge count. Seven raters trained by practicing on sets of handprints for a one-month period. Intra-class correlation coefficients (ICCs) were based on 10 fingerprint patterns from one individual. For single and average measures the ICCs .80. Quality was periodically assessed by a gold standard rater (DJD).

3. Results

3.1 Participants

No significant differences were found for demographic variables including age, sex, education or parental education. Men and women in this sample did not differ in terms of fluctuating dermatoglyphic asymmetries, $z=-1.837$, $p=.1$. As expected, UHR participants were rated significantly higher than controls on positive and negative symptom domains (see Table 1).

3.2 Group Differences in fluctuating dermatoglyphic asymmetries and relationship to symptoms

A Kolmogorov-Smirnoff test revealed that the distribution of total asymmetry scores were not normal, $D=.13$, $p=.001$, with skewness of 1.004 ($SE=.25$) and kurtosis of 1.09 ($SE=.49$). A Mann-Whitney U test was used to examine group differences. The UHR group showed significantly elevated total dermatoglyphic asymmetries compared to healthy controls $z=-2.22$, $p=.05$, $r=.23$ (see Figure 1). The control group showed a limited range in symptom scores; Spearman correlations run within the UHR group revealed that there was not a significant relationship between dermatoglyphic asymmetries and elevated positive, $r(49)=-.07$, $p=.6$ or negative symptoms, $r(49)=.19$, $p=.17$.

4. Discussion

A neural diathesis-stress model of psychosis suggests that early vulnerability may, in part, reflect genetic or epigenetic alterations and prenatal insult (Walker et al., 2008). Evidence suggests that objective signs of prenatal insult such as minor physical anomalies and fluctuating dermatoglyphic asymmetry may be related to an underlying vulnerability that later interacts with neurodevelopmental and environmental factors, ultimately contributing to psychosis (Golembo-Smith et al., 2012; Mittal et al., 2012; Mittal et al., 2007; Mittal et al., 2008b; Weinstein et al., 1999). Importantly, this is the first study in an UHR sample to find that these individuals show a greater number of dermatoglyphic asymmetries compared to healthy controls.

Fluctuating dermatoglyphic asymmetries are a part of the myriad minor physical anomalies (MPAs) linked to abnormal neurodevelopment in psychosis (Gabalda and Compton, 2010; Weinstein et al., 1999). In contrast to our hypothesis, we did not find an association between symptoms and fluctuating dermatoglyphic asymmetries. The present results are consistent with cross sectional investigations in psychosis patients, their healthy relatives and schizotypal individuals that have noted null results in terms of the relationship between fluctuating dermatoglyphic asymmetries and symptoms (Chok et al., 2005; Compton and Walker, 2009; Gabalda and Compton, 2010; Mittal and Walker, 2011). However, other cross sectional studies have found associations between palmar dermatoglyphic asymmetries and negative symptoms in schizophrenia patients as well as negative schizotypy in university students (Páez et al., 2001; Rosa et al., 2005). One explanation is that previous work in schizophrenia patients notes a large amount of heterogeneity in examining dermatoglyphic abnormalities (Golembo-Smith et al., 2012). Future work should assess whether separate measures of dermatoglyphics (i.e., palmer ridge counts, ATD angle, 2D4D ratio) are associated with specific symptom domains. Another explanation is that dermatoglyphics are closely associated with altered neurodevelopment, which may be a part of the diathesis for psychopathology but not specific to symptoms of risk for psychosis. Certainly, findings linking MPAs to bipolar disorder support this idea (Trixler et al., 2001). It is important to consider that UHR syndromes are defined in large part due to the presence of positive symptoms; findings of group differences in dermatoglyphic features support a link with this particular domain. However, the findings do not support a link between asymmetries and further variability of positive symptoms, or any association with negative symptoms.

The major limitations to this study relate to lack of corroborative markers (e.g., serological or birth record data, family handprints) as well as a follow-up time point. Previous work in larger samples of psychosis patients has noted that it may be important to examine dermatoglyphics separately between men and women because of sex differences in intrauterine environment (Saha et al., 2003). We did not find a significant difference between men and women in terms of dermatoglyphic asymmetry, but larger samples may be needed to examine sex differences during the UHR period. While this series of results does lay the groundwork for biomarker development, indicating for the first time that abnormal morphological features are present in UHR populations, large-scale studies utilizing multiple time points are necessary for examining links between dermatoglyphic asymmetries with eventual conversion to psychosis.

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Figure 1.

Asymmetrical dermatoglyphic patterns. Figure taken from Mittal and colleagues (Mittal et al., 2012). In this example of an asymmetrical print pair, taken from a non-participant model, the ridge count between the triradius (meeting of patterns on the periphery of the print) and core (center of print) for the left finger L3 (A) is markedly lower than that of the corresponding homologous right finger R3 (B). The UHR group showed a greater total asymmetry score with all five-finger pairs combined.

Table 1

UHR and healthy controls did not differ in terms of age, education, sex, and parental education. UHR participants were rated significantly higher on both positive and negative symptom domains. UHR individuals showed significantly increased fluctuating dermatoglyphic total scores compared to healthy controls. NS indicates not significant.

	UHR	Control	Statistic	p
Age				
Mean (SD)	18.55 (1.72)	17.80 (2.94)	$t(69.22) = 1.49$	NS
Sex				
Male	31	22		
Female	20	23		
Total	51	45	$\chi^2(1, N = 96) = 1.37$	NS
Race				
American Indian or Alaskan Native	2	0		
East Asian	2	5		
Southeast Asian	0	2		
Black	1	1		
Central/South American	10	9		
West/Central Asia and Middle East	1	2		
White	34	26		
Interracial	1	0	$\chi^2(7, N = 96) = 7.39$	NS
Ethnicity				
Hispanic	11	9		
Not Hispanic	40	36	$\chi^2(1, N = 96) = .04$	NS
Education (years)				
Mean (SD)	12.28 (1.84)	11.86 (2.96)	$t(72.73) = .87$	NS
Parent Education (years)				
Mean (SD)	15.81 (1.97)	15.66 (3.06)	$t(94) = .28$	NS
UHR Symptoms				
Positive Mean (SD)	12.08 (4.54)	.56 (1.25)	$t(58.51) = 17.38$	.001
Negative Mean (SD)	9.71 (6.70)	.80 (.80)	$t(51.62) = 9.88$	.001
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	UHR	Control	Statistic	p
Mean Rank	54.42	41.79	U = 845.5	.05
Mean (SD)	15.00 (7.29)	11.98 (6.29)	-	-