Dermatoglyphic anomalies in psychometrically identified schizotypic young adults

By: James T. Chok, Thomas R. Kwapil, Angela Scheuermann

Chok, J.T., Kwapil, T.R., & Scheuermann, A. (2005). Dermatoglyphic anomalies in psychometrically identified schizotypic young adults. *Schizophrenia Research, 72*, 205-214.

Made available courtesy of Elsevier: http://dx.doi.org/10.1016/j.schres.2004.03.012

Abstract:

Dermatoglyphic anomalies are hypothesized to indicate disruptions in the second trimester of prenatal development, a time period that appears to be critical in the etiology of schizophrenia. The present study examined the presence of dermatoglyphic anomalies in psychometrically identified schizotypic young adults (n=51) and control participants (n=63) selected based upon their scores on the Perceptual Aberration [J. Abnorm. Psychology 87 (1978) 399] and Magical Ideation Scales [J. Consult. Clin. Psychol. 51 (1983) 215]. It was hypothesized that schizotypic participants would exhibit higher rates of dermatoglyphic anomalies than control participants. The Perceptual Aberration–Magical Ideation group exhibited lower total and absolute finger ridge counts and less complex pattern types than control participants—findings consistent with anomalies reported in patients with schizophrenia. These findings encourage future examination of these anomalies in individuals at-risk for schizophrenia and related disorders.

Keywords: Dermatoglyphic; Schizotypy; Schizophrenia; Neurodevelopment

Article:

The reliable identification of individuals who are at-risk for schizophrenia and related disorders should increase our understanding of the etiology of such conditions, and may foster the development of preventative treatment interventions. Current etiological models of schizophrenia (e.g., Meehl, 1990, Gottesman, 1991 and Andreasen, 1986) assume that the disorder arises from an interaction or accumulation of multiple risk factors, including genetic inheritance, gene expression, pre- and peri-natal insults, and other biopsychosocial stressors. These models explicitly or implicitly assume that there is a dynamic continuum of schizotypic adjustment with full-blown schizophrenia at the severe end of the continuum. Furthermore, these models suggest the existence of schizotypic individuals who have a vulnerability for schizophrenia and related disorders. However, it is hypothesized that the majority of these individuals will not develop schizophrenia, although they may experience attenuated or transient symptoms of the disorder and may genetically transmit this risk to their offspring (e.g., Gottesman and Bertelsen, 1989). These symptoms are presumed to fall on a continuum from relative health to subclinical deviance to schizophrenia-spectrum personality disorders to full-blown clinical psychosis. Thus, schizotypy appears to be expressed across a dynamic continuum of adjustment with severity modulated by the interaction of biopsychosocial factors (Gooding and Iacono, 1995).

Schizotypy (and consequently the vulnerability for schizophrenia) is presumed to arise from processes of neural dysmaturation beginning during the second trimester of gestation (e.g., Andreasen, 1986, Keshavan, 1997 and Weinberger, 1987). While genetic factors are likely the largest contributors to prenatal neural dysmaturation, a variety of other adverse prenatal events can disrupt neurodevelopment and likely contribute to the risk for schizophrenia and related disorders, such as exposure to the influenza virus Mednick et al., 1988, Barr et al., 1990 and O'Callaghan et al., 1991b, increased psychological distress, and famine Huttunen and Niskanen, 1978 and Susser et al., 1996.

The proposed mechanism for increased risk for schizophrenia due to prenatal insults is a disruption of cell migration that leads to brain formation, which largely occurs during the second trimester of pregnancy. Neurohistological studies involving posthumous examination of the brains of patients with schizophrenia and control participants provide evidence of cell disorientation and misplacement in schizophrenia Conrad et al., 1991, Conrad and Scheibel, 1987, Arnold et al., 1991 and Selemon et al., 1995. Prenatal disruptions in brain formation could lead to further neural dysmaturation during childhood and adolescence (e.g., disruptions in synaptic pruning during adolescence). Thus, it appears that prenatal disruptions set in motion a pattern of neural dysmaturation that can manifest in adolescence and adulthood as schizophrenia (Andreason, 1999).

Dermatoglyphic anomalies and schizophrenia

Prenatal neural development during the second trimester of gestation is accompanied by formation of the much of the ultimate ectoderm. In fact, these two processes are inextricably intertwined. For example, the neural tube develops from the elementary ectoderm, and the lumen of the neural tube goes on to develop into the ventricular system of the brain. Thus, if risk for schizophrenia were heightened by disruptions affecting neurodevelopment, one would surmise that these disruptions might also be noted on the ultimate ectoderm (O'Connell et al., 1997). Consistent with these hypotheses, there is extensive evidence that disruptions in skin formation on the fingers and palms are more common in patients with schizophrenia than in samples of individuals without schizophrenia. These differences in ectoderm development are generically referred to as “dermatoglyphic anomalies” and can be categorized into two distinct groups: (1) qualitative and/or quantitative differences in finger and palmar ridge formation characteristics (e.g., people with schizophrenia may have higher or lower overall finger ridge counts than control participants), and (2) fluctuating dermatoglyphic asymmetries (FDA), which are defined as random differences between homologous finger and palmer ridge characteristics (e.g., individuals with schizophrenia are reported to have a higher degree of asymmetry between finger pairs than control participants).

Patients with schizophrenia have been found to differ from controls on a variety of dermatoglyphic measures. Table 1 provides a synopsis of the empirical literature investigating the role of dermatoglyphic anomalies in individuals with psychotic disorders. While it is clear that the majority of investigations have yielded significant findings, some studies have indicated no significant differences Cantor-Graae et al., 1998 and Rosa et al., 2000b and others have yielded very small differences. For example, Mellor (1992) reported higher rates of fluctuating

dermatoglyphic asymmetry (FDA) among patients with schizophrenia for finger ridge counts, fingerprint patterns, palmar angles, and palmar ridge counts in comparison to control participants. However, the observed differences in the study were modest (e.g., a group of 482 patients with schizophrenia had 23.1% of their finger pairs discordant compared to a control group of 166 participants, who had 18% of their finger pairs discordant).

TABLE 1 OMITTED FROM THIS FORMATED DOCUMENT

Furthermore, few investigations have examined dermatoglyphic anomalies in those at-risk for schizophrenia. In order to be a valid marker, dermatoglyphic anomalies should be present in the prodromal, active, and residual phases of schizophrenia (consistent with the recommendations of Garver, 1987), and should be found at higher rates in schizotypic individuals and in patients with schizophrenia-spectrum disorders (i.e., disorders such as schizotypal personality disorder that are thought to be etiologically related to schizophrenia). Weinstein et al. (1999) have provided evidence that individuals with schizotypal personality disorder have higher absolute finger ridge count asymmetry than control participants; however, this was the only dermatoglyphic index they investigated. Furthermore, Rosa et al. (2000c) found that degree of FDA was positively correlated with the negative dimension of schizotypy among a sample of adolescent males, although no such relation existed among positive dimensions of schizotypy.

Dermatoglyphic anomalies and other forms of pathology

In addition to schizophrenia, dermatoglyphic abnormalities have been associated with epilepsy, Down's syndrome, mental retardation, neurofibromatosis, spina bifida, polydactyly (Cummins and Midlo, 1943), congenital heart disease (Hale et al., 1961), borderline personality disorder (Jelovac et al., 1995), and autism (Walker, 1977). Although dermatoglyphic anomalies are not unique to schizophrenia, it is hypothesized that examining these characteristics in a population of schizotypic individuals may further enhance our ability to identify individuals at risk for schizophrenia and related disorders.

Goals and hypotheses of the present study

The present study examined the presence of dermatoglyphic anomalies in schizotypic college students and control participants identified by scores on the Perceptual Aberration (Chapman et al., 1978) and Magical Ideation Scales (Eckblad and Chapman, 1983). Consistent with Garver's guidelines for robust markers of psychosis (1987), we hypothesized that dermatoglyphic anomalies would be found at a higher rate in schizotypic young adults than in a control sample. It should be noted that dermatoglyphic anomalies are thought to be a “gross marker” of a contributory risk factor (nonspecific disruption of prenatal development during the second trimester of gestation). Thus, our current knowledge about dermatoglyphic anomalies does not allow us to comment on the exact nature or severity of prenatal developmental disruptions (events which are neither necessary, nor sufficient, causes of schizotypy). Therefore, we do not offer hypotheses regarding specific types of anomalies or about the overall frequency of anomalies in our schizotypic sample. However, the presence of dermatoglyphic anomalies in psychometrically identified schizotypic samples may facilitate the identification of individuals at

risk for schizophrenia and move us closer to understanding their potential association with the etiology and development of schizotypy.

Methods

Participants

The Perceptual Aberration and Magical Ideation Scales were administered to approximately 1200 undergraduate students enrolled in general psychology courses at the University of North Carolina at Greensboro over the course of three semesters. Fifty-one schizotypic participants, identified by standard scores of at least +1.96 on either of the scales, and 63 control participants who received standard scores of less than +0.5 on both scales participated in the dermatoglyphic assessment. The selection procedures followed the recommendations of Chapman et al. (1995). Control participants were selected from among a larger pool of candidate participants using a semi-random procedure. When a Perceptual Aberration–Magical Ideation participant was identified, the next comparison participant on a sequential list was selected. Ninety-two percent of the students contacted agreed to participate in the dermatoglyphic assessment. The participants were limited to Caucasian and African American students because normative data on the schizotypy scales were not available for other ethnic groups. Table 2 provides demographic information for the participants. The groups did not differ on any of these characteristics.

Table 2.
Demographic characteristics of the participants by group

	Group			
	Perceptual Aberration/Magical Ideation (*n*=51)		Control (*n*=63)	
	Mean	S.D.	Mean	S.D.
Age	19.5	2.2	19.0	0.9
Years of education	12.3	1.3	12.3	0.6
Percent female	80%		70%	
Percent African American	20%		25%	

Materials

Screening questionnaires

The Perceptual Aberration and Magical Ideation Scales were intermixed with a 13-item infrequency scale (Chapman and Chapman, 1983) that was designed to screen out random or "fake-bad" response styles. Participants who endorsed three or more items on the infrequency scale were omitted from participation in the dermatoglyphic assessment. The Perceptual Aberration Scale consists of 35 items that tap schizophrenic-like perceptual experiences and bodily distortions. Sample items include, "I sometimes have the feeling that some parts of my body are not attached to the rest of me" [keyed true], and "My hands and feet have never seemed

far away" [keyed false]. The Magical Ideation Scale is made up of 30 items that measure belief in implausible or invalid causality. Sample items include: "I have occasionally had the silly feeling that a TV or radio broadcaster knew I was listening to him" [keyed true], and "Numbers like 13 and 7 have no special powers" [keyed false]. The scales were constructed using Jackson's (1970) method for rational scale development. The coefficient alpha internal consistency reliability was 0.88 for the Perceptual Aberration Scale and 0.84 for the Magical Ideation Scale in the screening sample. The Perceptual Aberration and Magical Ideation Scales have been used widely in cross-sectional and longitudinal studies of schizotypy. Groups identified as at-risk by these measures show psychological and physiological deficits similar to those seen in patients with schizophrenia and are at heightened risk for developing psychotic disorders (e.g., Chapman et al., 1994 and Chapman et al., 1995).

Assessment of dermatoglyphic anomalies

Twelve different finger and palmar measures of dermatoglyphic anomalies were completed for each participant. A triradius is present when three disparate ridges come together at approximately 120° angles (Holt, 1968). Finger ridge counts were determined by counting the number of ridges that intersected a straight line connecting the triradial point (the point of ridge intersection) to the point of the core (the ridge in the center of the pattern). If more than one triradius was present on a finger, multiple ridge counts were made (e.g., two triradii yielded two ridge counts). The ridges containing the point of the core and the triradial point were not included in this computation. Total finger ridge count (TFRC) was computed by summing the ridge counts of all 10 fingers, in which only the largest count was used on those digits with more than one ridge count. Absolute finger ridge count (AFRC) was determined by adding all of the ridge counts from each of the 10 fingers (including multiple ridge counts for fingers with two or more triradii). Palmar a–b ridge count was determined by counting the number of ridges that crossed a straight line drawn between the "a" and "b" triradii of the palm. The "a" area is the part of the palm just below the index finger and the "b" area is the part of the palm just below the middle finger. Parts "c" and "d" of the palm (located beneath the ring and last fingers, respectively) were not used for counting ridges because the ridges in these areas are often parallel to the line that is drawn to connect the triradii. Palmar atd angle was determined by measuring the angle formed when drawing a line from the palmar "a" triradius to the most distal "t" triradius at the base of the palm and then from that triradius to the palmar "d" triradius.

The frequency of pattern type on the fingers was also computed. Finger patterns were classified into three categories: (1) arches, which do not typically contain a triradius; (2) loops, which are characterized by a triradius that opens towards a side of the hand, and a core; (3) and whorls, closed patterns characterized by having at least two triradii and a core(s). In rare cases, an arch contains a triradius, but it always lacks a core, which precludes making a ridge count (and distinguishes them from loops and whorls). We also examined prints for signs of ridge dissociation, although none of the participants in the present study exhibited this severe anomaly.

Four FDA measures were computed for the participants by examining discrepancies between measures on the right and left hands or between homologous fingers. Because our hypotheses involved the degree of asymmetry, not the direction of lateralization, we used the absolute value of the discrepancies to compute FDA measures. Fluctuating asymmetry was determined for

TFRC and AFRC by comparing the relevant ridge counts of homologous fingers. The absolute differences for each of the five-finger pairs were summed to compute the asymmetry score. Fluctuating asymmetry of the palmar a–b ridge count was measured by taking the absolute difference between the right hand a–b ridge count and the left hand a–b ridge count. Likewise, palmar atd angle asymmetry was measured by taking the absolute difference between atd angles from the right and left hands.

Finger and palmar prints were made by one graduate and four undergraduate researchers using Perfect Ink printing apparatus (provided by Identicator, www.identicatorinc.com). Prints were scanned using a Visioneer 8900 USB scanner at 600 dpi and images were enlarged to over 600% using the "zoom" tool on Adobe Photoshop to provide a clear and accurate image of the prints for scoring. The "line-drawing" tool was also used to make a precise connection between the triradial point and the core. This tool also helped to minimize visual drifting, which can occur when trying to count ridges as the experimenter moves from the triradial point to the core. Varying density of ridges and other dermatoglyphic features that are often very minute in nature, such as interestial lines and islands, can easily be erroneously included in a ridge count if a precise line is not in place. Interrater reliability was assessed based upon prints from 72 participants that were scored independently by two trained raters. Disagreements were resolved by consensus for the purpose of subsequent analyses. The schedulers, examiners, and scorers were unaware of participants' group membership throughout the study.

Results

Table 3 presents the interrater reliability (intraclass correlations) for each of the dermatoglyphic measures. Table 4 presents the comparisons of the Perceptual Aberration–Magical Ideation and control groups on the dermatoglyphic measures. The schizotypy group had a significantly lower TFRC, AFRC, and number of whorls than the control group, and significantly more loops. The groups did not differ on either of the palmar measures. Likewise, the groups did not differ on any of the FDA comparisons. The effect sizes, as estimated by Cohen's d, were relatively small for all of the comparisons. While the groups did not differ on sex or ethnic composition, the analyses were re-run as stepwise regression analyses with these demographic characteristics entered at step one and group membership entered at step two. Significant effects were found for the demographic characteristics on only two of the 11 measures (number of loops and FDA TFRC). In both cases the effect resulted from Caucasian participants exceeding African American participants. The effects for group membership were substantially unchanged, although the difference on number of loops was only at the trend level ($p=0.06$).

Table 3.
Interrater reliability for dermatoglyphic anomaly measures (n=72)

	Intraclass correlation
Quantitative measures	
Total finger ridge count	0.97
Absolute finger ridge count	0.98
Palmar a–b ridge count	0.61
Palmar atd angle	0.70
Number of arches	0.97
Number of loops	0.86
Number of whorls	0.99
Fluctuating dermatoglyphic asymmetries	
Total finger ridge count	0.83
Absolute finger ridge count	0.90
Palmar a–b ridge count	0.70
Palmar atd angle	0.69

All correlations $p<0.001$.

Table 4.
Comparison of psychosis-prone and control groups on dermatoglyphic measures

	Group						
	Perceptual Aberration/Magical Ideation (*n*=51)		Control (*n*=63)				
	Mean	S.D.	Mean	S.D.	*t*-value	*p*-value	Cohen's *d*
Quantitative measures							
Total finger ridge count	108.3	53.0	129.1	56.9	2.00	0.048	0.38
Absolute finger ridge count	129.6	77.9	168.8	99.3	2.28	0.024	0.44
Palmar a–b ridge count	84.4	10.0	84.4	10.0	0.01	ns	0.00
Palmar atd angle	87.4	12.1	86.4	11.3	0.41	ns	0.08
Number of arches	1.1	2.1	0.8	1.8	0.85	ns	0.16
Number of loops	7.1	2.4	6.1	3.0	1.98	0.047	0.37
Number of whorls	1.7	2.2	3.1	3.2	2.61	0.010	0.48
Fluctuating dermatoglyphic asymmetry measures							
Total finger ridge count	16.4	7.6	14.8	7.9	1.04	ns	0.20
Absolute finger ridge count	21.5	12.5	24.5	13.8	1.17	ns	0.22
Palmar a–b ridge count	3.1	2.7	3.3	2.7	0.32	ns	0.06
Palmar atd angle	2.9	3.7	2.9	2.8	0.09	ns	0.01

Discussion

The present study investigated the presence of dermatoglyphic anomalies in psychometrically identified schizotypic college students. It represents one of the first systematic assessments of dermatoglyphic characteristics in a nonclinical sample presumed to be at risk for schizophrenia and related conditions. The finding of such characteristics would support the utility of dermatoglyphic anomalies as a marker of risk for schizotypy and potentially facilitate the identification of individuals at risk for schizophrenia and related disorders. Consistent with primary hypothesis of the study, students identified by the Perceptual Aberration and Magical Ideation Scales differed from control participants on measures of finger ridge counts and displayed fewer complex fingerprint patterns. However, the groups did not differ on the palmar measures or on the FDA measures.

While the findings of an increased prevalence of dermatoglyphic anomalies in a schizotypic sample are concordant with Rosa et al.'s (2000c) demonstration of greater FDA in adolescents with higher schizotypy scores, the specific anomalies that were present differ across these studies. For example, both the current investigation and Rosa et al. found no relation between FDA and positive schizotypy. However, the current study did find reduced TFRC and AFRC among positive schizotypes in comparison to control participants. This suggests that while symmetry may be intact among those with positive schizotypy, other anomalies, such as reduced finger ridge count, may be evident and representative of similar prenatal disturbances. FDA on the other hand, which was found to be related to negative schizotypy in the Rosa et al. study, may be more germane to prenatal disruptions in negative schizotypes, which were not examined in the current investigation.

One possible explanation for the mixed findings involves the nature of the present sample. The current investigation employed a sample of college students at-risk for schizophrenia. While a number of longitudinal studies (e.g., Chapman et al., 1994 and Kwapil, 1998) have demonstrated that psychometrically identified college students are at heightened risk for psychotic and spectrum disorders, they provide a conservative sample and likely underestimate the utility of this research method. Specifically, college students represent the healthiest members of their age cohort in terms of risk for schizophrenia. Presumably, many schizotypic individuals (especially those who will ultimately develop schizophrenia) will experience impaired functioning and limited resources in late adolescence that will prevent them from enrolling in college and participating in such studies. The Epidemiological Catchment Area Study (Robins et al., 1984) indicated that college-educated individuals have a lower risk of developing schizophrenia than their peers without a college education. It may be that the presence of dermatoglyphic anomalies is associated with significant neurocognitive deficits and psychosocial stressors that might lessen the likelihood of enrollment in college. Therefore, the schizotypic sample employed in the current study may exhibit fewer dermatoglyphic anomalies than schizotypic samples selected from the general population (this notion is consistent with a multiple-hit model of risk). Indeed, some researchers have suggested that the rate of dermatoglyphic anomalies is associated with symptom severity. For example, Markow and Wandler (1986) found that more severe cases of schizophrenia, as indicated by early age of onset and declining course of illness, were correlated with the degree of fluctuating dermatoglyphic asymmetry. It should also be noted that the palmar and FDA measures, which did not differ between the groups, tended to have lower reliability than the other measures.

Consistent with previous studies of schizophrenic patients, the values of Cohen's d reported in the current study suggest that the effect sizes for differences between schizotypic and control groups are relatively modest in college student samples. While the sample size was sufficient to detect medium effect sizes (as in the case of AFRC and whorls), the sample was underpowered for detecting small effect sizes reported for the FDA measures. While dermatoglyphic anomalies may be less frequent in college samples of schizotypes, it does not necessarily rule out their utility for identifying individuals at risk for schizophrenia and related disorders (even in college student samples). We are currently examining the degree to which dermatoglyphic anomalies increment the cross-sectional identification and longitudinal prediction of symptoms and neurocognitive deficits in psychometrically identified schizotypes.

The theory behind the co-occurrence of dermatoglyphic anomalies and schizophrenia is based on the idea that the lumen of the neural tube, which later becomes the ventricular system and substance that makes up the brain, develops from the rudimentary ectoderm. Therefore, if schizophrenia is the result of a developmental brain disorder, we would expect to see anomalies in the formations of the skin in a subset of patients. Taken together with the empirical literature that has linked fluctuating asymmetry with weakened developmental stability, dermatoglyphic anomalies appear to be a promising marker for a developmental disturbance in the prenatal environment that may increase the risk for schizophrenia. While the base rate of dermatoglyphic anomalies may be relatively low in the present sample, they may identify a subset of individuals at especially heightened risk for developing schizophrenia and related disorders. The present study provides the first step in examining dermatoglyphic anomalies in a nonclinical sample and should provide the foundation for examining the relationship between these characteristics and schizotypic symptoms.

Acknowledgements

This research was conducted in partial fulfillment of the requirements for the degree of Master of Arts at the University of North Carolina at Greensboro for the first author. This study was supported by a research grant from the University of North Carolina at Greensboro.

The authors are indebted to Kate Durkin, Jason Nifong, Julie Sands, and Casey Smith for their assistance with subject recruitment and data collection.

References

Andreasen, 1986.N.C. Andreasen (Ed.), Can Schizophrenia Be Localized in the Brain?, American Psychiatric Press, Washington, DC (1986), pp. 1–87

Andreason, 1999. N.C. Andreason A unitary model of schizophrenia: Bleuler's fragmented phrene as schizencephaly Arch. Gen. Psychiatry, 56 (1999), pp. 781–787

Arnold et al., 1991. S.E. Arnold, B.T. Hyman, G.W. Van Hoesen, A.R. Damasio Some cytoarchitectural abnormalities of the entorhinal cortex in schizophrenia Arch. Gen. Psychiatry, 48 (1991), pp. 625–632

Barr et al., 1990. C.E. Barr, S.A. Mednick, P. Munck-Jorgenson. Maternal influenza and schizophrenic births. Arch. Gen. Psychiatry, 47 (1990), pp. 869–874

Bracha et al., 1992. H.S. Bracha, E.F. Torrey, I.I. Gottesman, L.B. Bigelow, C. Cunniff Second-trimester markers of fetal size in schizophrenia: a study of monozygotic twins Am. J. Psychiatry, 149 (10) (1992), pp. 1355–1361

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

Chapman and Chapman, 1983. Chapman, L.J., Chapman, J.P. 1983. Infrequency Scale for Personality Measures. Unpublished scale available from T.R. Kwapil, Department of Psychology, University of North Carolina at Greensboro, Greensboro, NC 27402.

Chapman et al., 1978. L.J. Chapman, J.P. Chapman, M.L. Raulin Body image aberration in schizophrenia. J. Abnorm. Psychology, 87 (1978), pp. 399–407

Chapman et al., 1994. L.J. Chapman, J.P. Chapman, T.R. Kwapil, M. Eckblad, M.C. Zinser

Putatively psychosis-prone subjects 10 years later. J. Abnorm. Psychology, 103 (1994), pp. 171–183
Chapman et al., 1995. J.P. Chapman, L.J. Chapman, T.R. Kwapil. Scales for the measurement of schizotypy. A. Raine, T. Lencz, S. Mednick (Eds.), Schizotypal Personality Disorder, Cambridge Univ. Press, Cambridge, England (1995), pp. 79–100
Conrad and Scheibel, 1987. A.J. Conrad, A.B. Scheibel. Schizophrenia and the hippocampus: the embryological hypothesis extended. Schizophr. Bull, 13 (1987), pp. 577–587
Conrad et al., 1991. A.J. Conrad, T. Adebe, R. Austin, S. Forsythe, A. Scheibel. Hippocampal pyramidal cell disarray in schizophrenia as a bilateral phenomenon. Arch. Gen. Psychiatry, 48 (1991), pp. 413–417
Cummins and Midlo, 1943. H. Cummins, C. Midlo. Finger Prints, Palms and Soles. Dover, New York (1943)
Davis and Bracha, 1996. J.O. Davis, H.S. Bracha. Prenatal growth markers in schizophrenia: a monozygotic co-twin control study. Am. J. Psychiatry, 153 (1996), pp. 1166–1172
Eckblad and Chapman, 1983. M.L. Eckblad, L.J. Chapman. Magical ideation as an indicator of schizotypy. J. Consult. Clin. Psychol, 51 (1983), pp. 215–225
Fananas et al., 1990. L. Fananas, P. Moral, J. Bertranpett. Quantitative dermatoglyphics in schizophrenia: study of family history subgroups. Hum. Biol, 62 (1990), pp. 421–427
Fananas et al., 1996. L. Fananas, J. Van Os, C. Hoyos, J. McGrath, C.S. Mellor, R. Murray. Dermatoglyphic a–b ridge count as a possible marker for developmental disturbance in schizophrenia: replication in two samples. Schizophr. Res, 20 (1996), pp. 307–314
Fearon et al., 2001. P. Fearon, A. Lane, M. Airie, J. Scannell, A. McGowan, M. Byrne, M. Cannon, D. Cotter, P. Murphy, B. Cassidy, J. Waddington, C. Larkin, E. O'Callaghan. Is reduced dermatoglyphic a–b ridge count a reliable marker of developmental impairment in schizophrenia? Schizophr. Res, 50 (3) (2001), pp. 151–157
Garver, 1987. D.L. Garver. Methodological issues facing the interpretation of high-risk studies: biological heterogeneity. Schizophr. Bull, 13 (3) (1987), pp. 525–529
Gooding and Iacono, 1995. D.C. Gooding, W.G. Iacono. Schizophrenia Through the Lens of a Developmental Psychopathology Perspective. Developmental Psychopathology, Wiley, New York (1995), pp. 535–580
Gottesman, 1991. I.I. Gottesman. Schizophrenia Genesis Freeman, New York (1991)
Gottesman and Bertelsen, 1989. I.I. Gottesman, A. Bertelsen. Confirming unexpressed genotypes for schizophrenia: risks in the offspring of Fischer's Danish identical and fraternal discordant twins. Arch. Gen. Psychiatry, 46 (1989), pp. 867–872
Hale et al., 1961. A.R. Hale, J.H. Phillips, G.E. Burch. Features of palmar dermatoglyphics in congenital heart disease. J. Am. Med. Assoc, 25 (1961), pp. 461–468
Holt, 1968. S.H. Holt. The Genetics of Dermal Ridges. Charles C. Thomas, USA (1968)
Huttunen and Niskanen, 1978. M.O. Huttunen, P. Niskanen. Prenatal loss of father and psychiatric disorders. Arch. Gen. Psychiatry, 35 (1978), pp. 429–431
Jackson, 1970. D.N. Jackson. A sequential system for personality scale development. C.N. Spielberger (Ed.), Current Topics in Clinical and Community Psychology, vol. 2, Academic Press, New York (1970), pp. 61–96
Jelovac et al., 1995. N. Jelovac, J. Milicic, P. Rudan, M. Milas. Dermatoglyphic analysis in borderline personality disorder. Psychiatr. Danub, 7 (1995), pp. 139–145

Keshavan, 1997. M.S. Keshavan. Neurodevelopment and schizophrenia: quo vadis? M.S. Keshavan, R.M. Murray (Eds.), Neurodevelopment and Adult Psychopathology, Cambridge Uneversity Press, United Kingdom (1997), pp. 267–277

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

Markow and Wandler, 1986. T.A. Markow, K. Wandler. Fluctuating dermatoglyphic asymmetry and the genetics of liability to schizophrenia. Psychiatry Res, 19 (1986), pp. 323–328

Mednick et al., 1988. S.A. Mednick, R.A. Machon, M.O. Huttunen, D. Bonett. Adult schizophrenia following prenatal exposure to an influenza epidemic. Arch. Gen. Psychiatry, 45 (1988), pp. 189–192

Meehl, 1990. P.E. Meehl Toward an integrated theory of schizotaxia, schizotypy, and schizophrenia. J. Pers. Disord, 4 (1990), pp. 1–99

Mellor, 1968. C.S. Mellor. Dermatoglyphics in schizophrenia. Br. J. Psychiatry, 114 (1968), pp. 1387–1397

Mellor, 1992. C.S. Mellor. Dermatoglyphic evidence of fluctuating asymmetry in schizophrenia Br. J. Psychiatry, 160 (1992), pp. 467–472

O'Callaghan et al., 1991b. E. O'Callaghan, P. Sham, N. Takei, G. Glover, R.M. Murray. Schizophrenia after prenatal exposure to 1952 A2 influenza epidemic. Lancet, 337 (1991), pp. 1248–1250

O'Connell et al., 1997. P. O'Connell, P.W.R Woodruff, I. Wright, P. Jones, R.M. Murray. Developmental insanity or dementia praecox: was the wrong concept adopted? Schizophr. Res, 23 (1997), pp. 97–106

Reilly et al., 2001. J.L. Reilly, P.T. Murphy, M. Byrne, C. Larkin, M. Gill, E. O'Callaghan, A. Lane. Dermatoglyphic fluctuating asymmetry and atypical handedness in schizophrenia Schizophr. Res, 50 (3) (2001), pp. 159–168

Robins et al., 1984. L.N. Robins, J.E. Helzer, M.W. Myrna, H. Orvaschel, E. Gruenberg, J.D. Burke, A.R. Darrel. Lifetime prevalence of specific psychiatric disorders in three sites Arch. Gen. Psychiatry, 41 (1984), pp. 949–958

Rosa et al., 2000a. A. Rosa, L. Fananas, H.S. Bracha, E.F. Torrey, J. van Os. Congenital dermatoglyphic malformations and psychosis: a twin study. Am. J. Psychiatry, 157 (9) (2000), pp. 1511–1513

Rosa et al., 2000b. A. Rosa, M. Marcelis, L. Fananas, J. van Os, A–b ridge count and schizophrenia. Schizophr. Res, 46 (2–3) (2000), pp. 285–286

Rosa et al., 2000c. A. Rosa, J. van Os, L. Fananas, N. Barrantes, B. Caparros, B. Gutierrez, J. Obiols. Developmental instability and schizotypy. Schizophr. Res, 43 (2000), pp. 125–134

Selemon et al., 1995. L.D. Selemon, G. Rajkowska, P.S. Goldman-Rakic. Abnormally neuronal density in the schizophrenic cortex. Arch. Gen. Psychiatry, 52 (1995), pp. 805–818

Susser et al., 1996. E.S. Susser, R. Neugebauer, H.W. Hoek, A.S. Brown, S. Lin, D. Labovitz, J.M. Gorman. Schizophrenia after prenatal famine. Arch. Gen. Psychiatry, 53 (1996), pp. 25–31

Turek, 1990. S. Turek. Dermatoglyphics and schizophrenia: analysis of quantitative traits Coll. Antropol, 14 (1) (1990), pp. 137–150

van Oel et al., 2001. C.J. van Oel, W.F.C. Baare, H.E. Hulshoff Pol, J. Haag, J. Balazs, A. Dingemans, R.S. Kahn, M.M. Sitskoorn. Differentiating between low and high susceptibility to schizophrenia in twins: the significance of dermatoglyphic indices in

relation to other determinants of brain development. Schizophr. Res, 52 (3) (2001), pp. 181–193

van Os et al., 1997. J. van Os, L. Fananas, M. Cannon, A. Macdonald, R. Murray. Dermatoglyphic abnormalities in psychosis: a twin study. Soc. Biol. Psychiatry, 41 (1997), pp. 624–626

van Os et al., 2000. J. van Os, P.W.R. Woodruff, L. Fananas, F. Ahmad, N. Shuriquie, R. Howard, R.M. Murray. Association between cerebral structural abnormalities and dermatoglyphic ridge counts in schizophrenia. Compr. Psychiatry, 41 (5) (2000), pp. 380–384

Varma et al., 1995. S.L. Varma, T.R. Chary, S. Singh, M.Z. Azhar, A.S. Dharap. Dermatoglyphic patterns in schizophrenic patients. Acta Psychiatr. Scand, 91 (1995), pp. 213–215

Walker, 1977. H.A. Walker. A dermatoglyphic study of autistic patients. J. Autism Child. Schizophrenia, 7 (1) (1977), pp. 11–21

Weinberger, 1987. D.R. Weinberger. Implication of normal brain development for the pathogenesis of schizophrenia. Arch. Gen. Psychiatry, 44 (1987), pp. 660–669

Weinstein et al., 1999. D.D. Weinstein, D. Diforio, J. Schiffman, E. Walker, R. Bonsall

Minor physical anomalies, dermatoglyphic asymmetries, and cortisol levels in adolescents with schizotypal personality disorder. Am. J. Psychiatry, 156 (4) (1999), pp. 617–623