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Directional and fluctuating asymmetry in finger and *a-b* ridge counts in psychosis: a case-control study

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Published: 23 March 2003

Received: 18 November 2002

BMC Psychiatry 2003, 3:3

Accepted: 23 March 2003

This article is available from: <http://www.biomedcentral.com/1471-244X/3/3>

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Abstract

Background: Several studies have reported alterations in finger and *a-b* ridge counts, and their derived measures of asymmetry, in schizophrenia compared to controls. Because ridges are fully formed by the end of the second trimester, they may provide clues to disturbed early development. The aim of this study was to assess these measures in a sample of patients with psychosis and normal controls.

Methods: Individuals with psychosis ($n = 240$), and normal controls ($n = 228$) were drawn from a catchment-area case-control study. Differences in finger and *a-b* ridge count and Fluctuating Asymmetry were assessed in three group comparisons (non-affective psychosis versus controls; affective psychosis versus controls; non-affective psychosis versus affective psychosis). The analyses were performed separately for males and females.

Results: There were no significant group differences for finger nor *a-b* ridge counts. While there were no group difference for Directional Asymmetry, for Fluctuating Asymmetry measures men with non-affective psychosis had significantly higher fluctuating asymmetry of the index finger ridge count (a) when compared to controls (FA-correlation score, $p = 0.02$), and (b) when compared to affective psychosis (adjusted FA-difference score, $p = 0.04$).

Conclusion: Overall, measures of finger and *a-b* ridge counts, and their derived measures of directional and fluctuating asymmetry were not prominent features of psychosis in this sample. While directional asymmetry in cerebral morphology is reduced in schizophrenia, this is not reflected in dermatoglyphic variables.

Background

While the aetiology of schizophrenia is still poorly understood, many researchers believe that the disorder is a consequence of genetic and environmental factors that

impact on brain development [1–3]. Because prenatal brain development is not open to direct scrutiny, researchers have resorted to the study of minor physical anomalies and dermatoglyphic variables. Dermatoglyphic patterns

form on the finger pad and on the palm by the end of the second trimester and remained unchanged thereafter [4,5], thus these features may serve as proxy markers of altered early development in psychosis [6,7].

In recent decades a large body of biological research has developed around the concept of fluctuating asymmetry (FA), which has been defined as random differences between the right (R) and left (L) sides of a morphological trait [8]. When the distribution of the right minus left (R-L) differences in a population sample approximates a normal curve with a mean of zero (or close to zero), the variance of the distributions of R-L difference is a measure of FA [8–11]. FA has been regarded by many researchers as primarily being an expression of environmental 'noise' [12,13] disrupting the fidelity of the genetic 'signal'. However, genetic factors may also have a weak link to FA in finger ridge counts [13] and *a-b* ridge counts [12]. It has been proposed that the degree of FA in an organism reflects the 'developmental instability' of that organism [14,15].

While FA requires that the R-L differences are random and non-directional, directional asymmetry (DA) involves a significant departure from zero of the normally distributed mean of R-L differences. Examples in humans include the asymmetry of the planum temporal, branching of the bronchi, and the distribution of certain internal organs. There is evidence demonstrating rightward DA in finger ridge counts [16,17]. Loesch & Martin [17] and Martin et al. [13] emphasizes that genetic factors affect directional asymmetry. In the light of the robust evidence showing a loss of normal cerebral asymmetry in schizophrenia [18], it is surprising that DA in dermatoglyphic variables has received little attention. Only two studies have compared DA in schizophrenia versus control groups [9,19] in which there were no significant group differences for DA.

The early literature reported a relatively diverse and inconsistent range of dermatoglyphic alterations in schizophrenia (see review by Loesch [20]). However, more consistent results have emerged from recent studies that have examined ridge count variables in patient group assessed with modern diagnostic criteria. For example, most recent studies have found that patients with schizophrenia have reduced palmar *a-b* counts (between the palmar triradi below the index and middle fingers) compared to controls [21–26], but not all studies have found this [27]. However, studies that have examined other quantitative dermatoglyphic variables in schizophrenia and psychosis have produced inconsistent results. At least four studies in the last decade reported reduced total finger ridge counts in schizophrenia compared with normal controls [23,25,26,28] while others have found no significant differences between patients and controls [21,22,24]. In addition, two studies have demonstrated significantly

greater intra-pair differences in ridge counts for monozygotic twins discordant for schizophrenia compared to monozygotic twin pairs concordant for schizophrenia [21,29].

Several studies have reported greater dermatoglyphic FA for both *a-b* ridge counts and fingerprint patterns in individuals with schizophrenia compared to normal controls [14]. Later studies reported similar findings for finger ridge counts [9,25,30,31], and palmar *a-b* ridge counts [9,25,29,30]. Greater FAs were also observed in twins concordant for schizophrenia in finger ridge counts compared to pairs in which one co-twin was normal [19].

With respect to affective psychosis, the studies of dermatoglyphic variables have been inconsistent. Some studies have reported lower finger and *a-b* ridge counts and an increase of aberrant dermatoglyphic features in affective psychosis (mainly bipolar disorders) [25,32,33], while other studies have reported no group differences [14,34].

The lack of consistency in the literature may be due to the differences in sample characteristics, methodology, or analytical techniques. Most of the recent studies that have examined dermatoglyphic variables in schizophrenia have been based on relatively modest sample sizes, thus small group differences may have been missed due to lack of power. In addition, different methods of FA measure have been used. Some studies have used FA scores based on the spread or variability of the measures- the 'correlation method' [9], while other have used simpler measures derived from right or left shift in the mean scores- the 'difference method' [12,25,30]. We have examined dermatoglyphic variables in a relatively large case-control study where subjects were drawn from a population-based prevalence study of psychosis using both the 'correlation' and the 'difference' methods. We compared non-affective psychosis and affective psychosis with controls. We hypothesised that (a) those with non-affective psychosis would have higher FA compared to well controls, and (b) those with affective psychosis would have higher FA compared to well controls. We also predicted that those with non-affective psychosis would have less DA compared to well controls.

Methods

Subjects and diagnostic assessment

Subjects were drawn from a case-control study designed to examine risk factors for psychosis [35,36]. Individuals with psychosis were drawn from one of the four catchment areas in the recent Australian national prevalence study of psychosis (the Queensland segment of the National Survey of Mental Health and Well-being- Low prevalence (psychotic) disorders) [37,38]. Within an area of south-east Queensland (eligible population = 581,332)

we undertook a one-month census at a wide range of sites (inpatient, outpatient, clinic, general practice, private psychiatrists, hostels, boarding homes, homeless shelters, day centres) in order to identify persons aged 18–64 who were in contact with mental health services in the area and who met screening criteria for psychotic disorders. During the census month (June 1997), a total of 2,180 individuals were screened for symptoms of psychosis using the Psychosis Screen [37]. This instrument, derived from psychosis screening items of the Composite International Diagnostic Interview (CIDI) [39] and the Psychosis Screening Questionnaire [40](), is a six-item screen for specific psychotic symptoms current or at any time in the past with one item for the rater to record their judgement whether psychotic symptoms may be present. Of the 2,180 individuals screened, 1,513 were positive for psychosis. Over an 18-month period, we recruited 310 of the positive survey members to participate in the current study (cases were randomly selected for screen-positive survey members). Concerning the representativeness of the final 310 subjects, there were no significant differences in age ($t = -1.31$, $df 1511$, $p = 0.20$) nor sex (chi-square = 1.73, $df 1$, $p = 0.19$) between this group and the remaining 1203 positive individuals. The normal control subjects were recruited from the same catchment area via advertisements in local newspapers. All subjects included in this study provided written informed consent, and the study was approved by the Wolston Park Hospital Institutional Ethics Committee.

All patients and normal controls were assessed with the Diagnostic Interview for Psychosis (DIP) [37] which uses probes derived from the Schedule for the Assessment of Clinical Neuropsychiatry (SCAN) [41]. The raters (clinical psychologists and experienced research nurses) attended national training programs in the use DIP and participated in inter-rater reliability exercises as part of the national study (agreement on diagnosis weighted kappa = 0.60, $p < 0.01$). Normal control status or patient's diagnosis was confirmed with the Operational Criteria for Psychosis (OPCRIT), a 90-item checklist linked to a computer diagnostic algorithm- DSM-III-R [42]. DSM-III-R diagnoses were divided into non-affective psychosis (schizophrenia, schizophreniform psychosis, delusional disorder, atypical psychosis) and affective psychosis (bipolar disorder and mania with psychosis, depression with psychotic features, schizoaffective psychosis).

Dermatoglyphic measures

Finger and palm prints of both hands were taken from subjects using a standard inkless technique. We measured (a) finger ridge counts (FRC) for each digit separately, and (b) *a-b* ridge counts (*a-bRC*), which is the number of ridges intersected by a line drawn between the *a* triradius (at the base of the index finger) and *b* triradius (at the base

of the middle finger) of the palm in each hand. All measures were assessed by one trained rater, who was blind to the subject's group status, under the supervision of one of the authors (DL). We followed standard scoring procedures as outlined in Loesch [20].

Statistical analysis

While the overall difference between the three groups was assessed, the primary analyses involve three group comparisons; (a) non-affective psychosis (schizophrenia, schizophreniform psychosis, delusional disorder, atypical psychosis) versus controls, (b) affective psychosis (depression with psychosis, bipolar disorder, schizoaffective psychosis) versus controls and (c) non-affective psychosis versus affective psychosis. Because we expected the prevalence sample to have different sex ratios (e.g. more men than women in the non-affective psychoses compared to the affective psychosis), and as dermatoglyphic variables such as FRC differ between males and females [20], all comparisons were undertaken in males and females separately. Group differences (among three groups and between any two groups) were assessed using a General Linear Model (GLM) while groups were further compared by the Tukey-Kramer test for adjusted probability estimates.

Following standard recommendations, we used two different methods to calculate FA [10,11]. In the first method, comparisons were made in ridge counts between homologous fingers of the right and left hands using Pearson product-moment correlation coefficients (r) [43]. The difference in correlation coefficients between cases and controls was calculated using Fisher's z -transformation [44,45]. The square of the product-moment correlation coefficient (r) is a measure of their common variance, and $1 - r^2$ is an estimate of error variance, and thus a measure of FA [46,47]. This method (henceforth referred to FA-correlation) is not affected by directional asymmetry [10,11] and has been used in previous schizophrenia research [9]. As a second measure, FA was obtained by subtracting the left hand count from the right hand count and dividing by the average of the sum of both hand counts, and then taking the absolute value of the quotient [10,11]. This second method has also been used in previous schizophrenia research [30].

In keeping with earlier dermatoglyphic studies in schizophrenia [9,19], we analysed ridge counts separately in each homologous finger. Antisymmetry was assessed using tests of platykurtic or bimodal distribution of signed R-L differences of the distribution. Because directional asymmetry (DA) has been reported for ridge counts on the thumb [48], we assessed DA following the recommendation of Palmer [10]. The presence of DA artificially inflates the values of the FA-difference scores used in this study.

Table 1: Mean and standard deviation for dermatoglyphic variables by sex and by groups

	Mean (Standard deviation)		
	Controls N = 228† (M:F 122:106)	Non-affective psychosis N = 181† (M:F 118:63)	Affective psychosis N = 59† (M:F 25:34)
Males			
Right subtotal FRC	74.47 (27.60)	73.58 (22.77)	66.95 (29.94)
Left subtotal FRC	71.66 (23.85)	69.75 (22.84)	63.52 (29.83)
Total FRC	144.67 (50.13)	143.51 (44.82)	130.65 (59.84)
Right <i>a-b</i> RC	41.12 (6.34)	42.15 (5.41)	41.81 (5.52)
Left <i>a-b</i> RC	41.45 (6.21)	42.49 (5.57)	42.83 (5.15)
Total <i>a-b</i>RC	82.58 (11.71)	84.51 (10.23)	84.40 (10.14)
Females			
Right subtotal FRC	68.30 (26.05)	66.16 (26.19)	68.64 (26.69)
Left subtotal FRC	66.01 (27.51)	62.34 (26.44)	64.12 (27.06)
Total FRC	133.71 (53.06)	126.68 (51.17)	135.12 (49.39)
Right <i>a-b</i> RC	42.04 (5.49)	41.38 (5.71)	41.60 (6.85)
Left <i>a-b</i> RC	42.86 (4.91)	42.23 (4.73)	41.87 (6.87)
Total <i>a-b</i>RC	85.26 (9.86)	83.94 (9.09)	83.28 (12.96)

FRC = finger ridge count, *a-b*RC = *a-b* ridge count †The number of subjects available for each dermatoglyphic variable varies slightly

Thus, if significant DA was identified, FA-difference scores for all measures were adjusted by subtracting average mean right left difference (mean(R-L)/2) from the side with the larger mean and adding it to the smaller side of all individuals in the sample [11]. This variable will be referred as 'adjusted FA-difference'. In addition, we examine DA in the primary analyses involving all three group comparisons.

All analyses used SAS programming (version 8), and all significance tests were two-tailed with an alpha levels of $p < 0.05$.

Results

Of the 310 patients with psychosis and 303 normal controls recruited into the original risk factor study, finger and palm prints were available in 240 individuals with psychosis and in 228 normal controls. Three percent (7) of cases and seven percent (16) control subjects declined to provide prints. The quality of prints for fingers and the *a-b* palmar area of the remaining 63 patients and 59 controls was not suitable for inclusion in the study. There was no significant group difference in the distribution of missing prints ($\chi^2 = 0.05$, $p = 0.81$)

There were no statistically significant differences in sex distribution when the 'included subjects' were compared to 'subjects not suitable for assessment' ($\chi^2 = 0.21$, $p = 0.65$). Similarly, no sex differences were noticed when the three groups were examined separately between 'included subjects' and 'not suitable for assessment' subjects (data not shown). However, the mean age of the subjects 'not

suitable for assessment' was significantly lower than the 'included subjects' in the study (29.1 ± 13.9 versus 43.8 ± 9.6 years respectively; $t = -14.4$, $df = 611$, $p < 0.001$). This difference remained significant when the three groups were analysed separately (data not shown). However, since the dermatoglyphic variables examined in this study are stable after birth, it is unlikely that this difference would impact on the validity of the results.

Of the 240 individuals with psychosis, 181 had a non-affective psychosis (males:females = 118:63; schizophrenia $n = 166$, delusional disorder $n = 10$, atypical psychosis $n = 5$), while 59 had an affective psychosis (males:females = 25:34; bipolar disorder $n = 24$, mania with psychosis $n = 12$, depression with psychotic features $n = 14$, schizoaffective psychosis $n = 9$). Within the control group there were 122 males and 106 females.

Means and standard deviations for dermatoglyphic scores are shown in Table 1. There were no significant group differences for any of the ridge count measures when assessed in the three group comparisons, nor in comparing non-affective psychosis versus control, affective psychosis versus control, and non-affective psychosis versus affective psychosis.

There was no platykurtic and/or bimodal distributions of right-left differences (indicative of antisymmetry) for any of dermatoglyphic variables (data not shown).

When assessed within groups, we identified significant directional asymmetries (DA) for certain finger ridge counts

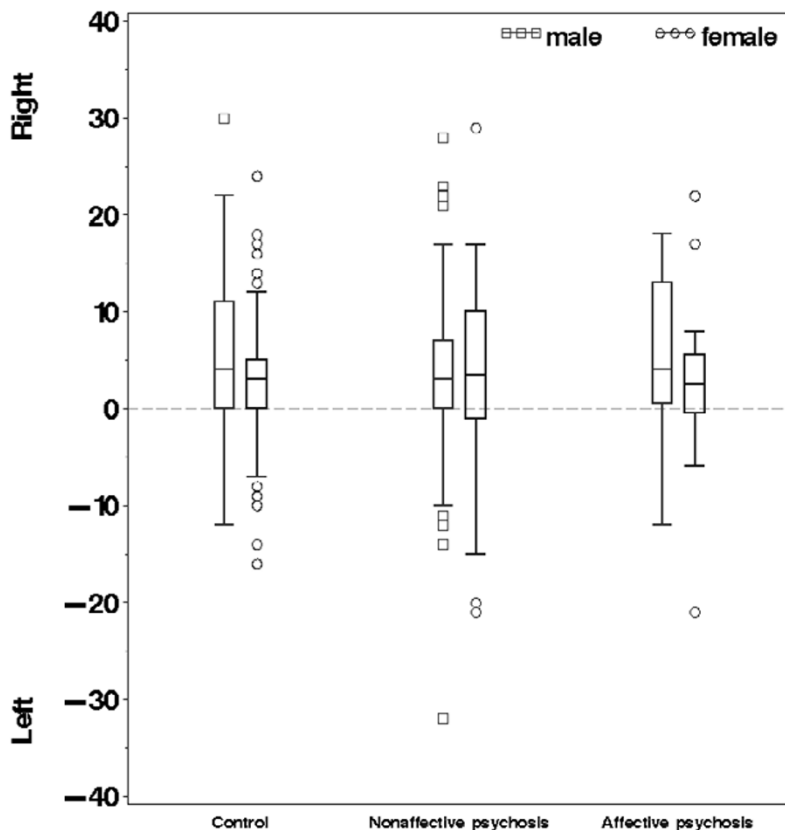


Figure 1

but not for *a-b* ridge counts. Significant DA in ridge counts for the thumb was found in each of the three groups for males (all $p < 0.01$) and in females from the non-affective psychosis and normal control groups ($p < 0.05$) (Figure 1). For total ridge counts, significant DA was found in male non-affective group ($p < 0.01$) and female normal controls and female affective psychosis ($p < 0.02$). Significant DAs was also identified in the ring finger (4th finger), (a) males with non-affective psychosis ($p < 0.02$), and (b) females normal controls ($p < 0.01$) (data not shown). In every case, the direction of the asymmetry was towards the right (i.e. more ridge counts on the right compared to the left at the group level). When we compared DA in the three group comparisons, no significant group differences were noted (data not shown).

Few significant group differences were identified on the measures of FA (Tables 2 and 3). The FA-correlation method identified significantly more FA in the second digit (the index finger) in males with non-affective psychosis

compared to normal controls ($p = 0.02$), and when comparing non-affective psychosis with affective psychosis ($p < 0.001$) (Table 2). There was significantly more FA in the fifth digit (little finger) in males with non-affective psychosis when compared with the affective group ($p = 0.02$). There were no significant FAs in *a-b* ridge counts when using the FA-correlation method.

Applying the adjusted FA-difference method, there was a significant difference in FA for the index fingers when male from the non-affective and affective groups were compared ($p = 0.04$). However, no statistically significant group differences were found in males for the other variables using the adjusted FA-difference method (Table 3). For *a-b*, there was significantly more FA in the female affective group when compared with the female controls ($p < 0.05$).

Table 2: Fluctuating asymmetry – correlation method: Correlation co-efficients of finger ridge counts and a-b ridge counts in male and females

Measure	Controls	Non-affective psychosis	Affective psychosis
	I - r ² (a)	I - r ² (b)	I - r ² (c)
Males FRC			
Digit I	0.33	0.51	0.33
Digit II	0.34	0.59*	0.15
Digit III	0.41	0.42	0.39
Digit IV	0.36	0.38	0.38
Digit V	0.45	0.66**	0.26
FRC (right vs left)	0.14	0.23	0.09
a-bRC (right vs left)	0.45	0.44	0.42
Females FRC			
Digit I	0.38	0.48	0.47
Digit II	0.48	0.39	0.19
Digit III	0.33	0.33	0.33
Digit IV	0.31	0.26	0.47
Digit V	0.38	0.50	0.33
FRC (right vs left)	0.15	0.19	0.15
a-bRC (right vs left)	0.31	0.48	0.38

NA = not applicable, FRC = finger ridge count, a-bRC = a-b ridge count *Fishers Z test for significant differences : Non-affective versus controls, (p < 0.02) **Fishers Z test for significant differences : Non-affective versus affective psychosis (p < 0.02)

Table 3: Adjusted fluctuating asymmetry-difference method: right-left differences of finger ridge counts and a-b ridge counts in male and females

	Mean (SD)		
	Controls	Non-affective Psychosis	Affective Psychosis
Males			
Digit I	0.35 (0.03)	0.31 (0.03)	0.41 (0.05)
Digit II	0.39 (0.04)	0.54 (0.05)*	0.46 (0.10)
Digit III	0.42 (0.05)	0.39 (0.04)	0.55 (0.14)
Digit IV	0.30 (0.03)	0.27 (0.03)	0.29 (0.05)
Digit V	0.29 (0.03)	0.37 (0.04)	0.32 (0.04)
FRC (right vs left)	0.13 (0.02)	0.15 (0.01)	0.30 (0.13)
a-bRC	0.08 (0.01)	0.07 (0.01)	0.08 (0.01)
Females			
Digit I	0.35 (0.03)	0.43 (0.06)	0.27 (0.05)
Digit II	0.54 (0.05)	0.50 (0.08)	0.49 (0.11)
Digit III	0.40 (0.05)	0.43 (0.06)	0.34 (0.06)
Digit IV	0.37 (0.04)	0.32 (0.04)	0.34 (0.07)
Digit V	0.29 (0.03)	0.37 (0.05)	0.28 (0.05)
FRC (right vs left)	0.17 (0.03)	0.17 (0.03)	0.14 (0.03)
a-bRC	0.06 (0.01)	0.07 (0.01)	0.09 (0.01)**

FRC = finger ridge count, a-bRC = a-b ridge count, Significant difference (F-test based on General Linear Model with adjusted probability estimates with Tukey-Kramer Test): * between non-affective psychosis and affective psychosis (p < 0.05) ** between affective psychosis and controls (p < 0.05)

Discussion

Despite the large sample size, this study found little evidence of altered dermatoglyphic variables in either non-affective psychosis or affective psychosis compared with normal controls. While our findings of lack of group difference was consistent with some studies [21,23], most groups have reported significantly reduced total finger ridge counts as well as in *a-b* ridge counts in schizophrenia versus well controls [22,24,25,28]. In addition, the lack of differences in finger and *a-b* ridge counts in affective psychosis compared to normal controls was consistent with other studies [14,25,32,34].

Our study did not find any statistically significant difference in fluctuating asymmetry for total finger ridge counts. However, there was a significant difference in FA for Digit II (index finger), where men with nonaffective psychosis had significantly more FA in finger ridge count compared to both normal controls and affective psychosis. This result was consistent over the two methods, and agrees broadly with the findings of Mellor [9] in which the greatest FA difference between schizophrenia versus controls was reported in the index finger. However, it is difficult to speculate on the meaning of this isolated function. Jantz showed that difference in right-left ridge counts was greatest for digit II compared to other digits (in normal subjects). Based on this higher directional asymmetry in digit II, we speculate that this digit may be more likely to differentiate groups with low versus high developmental instability. However, we recommend caution in the interpretation of this finding in the light of the many comparison undertaken in this study, and in the light of the fact that a similar difference was not found in females.

The result of overall no difference was in contrast with the findings reported by several other groups [19,25,30,31]. We note that the two methods used in this paper (FA-correlation and FA-difference methods) were not in agreement for all variables. Thus inconsistencies in the literature may be related to the different formulae used to assess dermatoglyphic FA.

While our sample was predominantly Caucasian, each of major group differences may reflect ethnic/genetic difference between our sample and the samples included in the studies based in Ireland, USA, and UK. In addition, perhaps there are regional variations in the prevalence or dose of putative non-genetic factors that may impact on both dermatoglyphic and brain development. For example, we have previously published evidence suggesting that the size of the season of birth winter/spring excess is smaller in the southern hemisphere studies compared to northern hemisphere studies [49]. However, in contrast to the lack of group differences in ridge counts, we have previously reported significantly more minor physical anom-

alies (MPA) and altered craniofacial measures in this same sample [36]. However, the subjects in the current dermatoglyphic study ($n = 468$) were a subset of the previously published MPA study ($n = 613$). Separate to the current analyses, we have looked for association between MPA (a summary score of qualitative measures of the head and face) and the main dermatoglyphic measures, but no significant association was found (data not shown).

There are several limitations in our study. The patients in this study were recruited during a prevalence study of psychosis, thus those with chronic, persisting types of illnesses would be over-represented in this sample. As increased fluctuating asymmetry has been associated with increased severity of the illness [14], our result of lack of group difference is surprising. The use of community advertisement for recruitment of normal controls may have also introduced some unspecified bias in the sample. Another possible weakness of the study was the significantly higher age in the 'included' group compared to 'not included' (whose hands prints were not available) group. Ridge counts do not vary with age [5,20]. It seems unlikely that secular or cohort changes extending over one or two decades in exposures would lead to appreciable changes in these variables. Measurement error can lead to greater variance and thus inflate FA. However, in this study measurement error was considered minimal as there was a single, experienced rater supervised by one of the author (DL). Finally, the alpha level was not adjusted for multiple comparisons, thus Type I errors cannot be excluded.

Yeo and colleagues [50] have proposed that properties related to increased developmental instability may underlie several of the features of schizophrenia, including increased minor physical anomalies, increased dermatoglyphic FA, and loss of normal cerebral asymmetry. Perhaps individuals at increased genetic risk of developing schizophrenia have reduced buffering capacity leading to the loss of phenotypic fidelity in the face of environmental stressors. Alternatively, perhaps environmental factors are responsible for the disruption in normal development, leading to immature structures that lack 'normal' asymmetries. For example, the factors that lead to the loss of normal brain asymmetry [18] may also lead to the disruption of other forms of subtle asymmetry such as FA. Regardless of the underlying mechanisms, we found that FA was not a prominent feature of psychotic disorders in this sample. We also draw attention to the curious fact that while normal cerebral laterality is reduced in schizophrenia, this does not seem to be case for dermatoglyphic variables. While there is a substantial evidence showing altered brain morphology in groups of individuals with schizophrenia, there is a little evidence to link these features with altered dermatoglyphic variables within subjects [7]. The overall lack of FA and DA differences

between schizophrenia and normal controls found in this study weakens the case that early developmental disruptions impact equally on the brain, and on finger and *a-b* ridge counts.

Overall, measures of finger and *a-b* ridge counts, and their derived measures of directional and fluctuating asymmetry were not prominent features of psychosis in this sample. While directional asymmetry in cerebral morphology is reduced in schizophrenia, this is not reflected in the dermatoglyphic variables assessed in this study.

Authors' contributions

SS and JM carried out statistical analysis, and prepared the manuscript. JM conceived the study, participated in its design and coordination. DC assisted in statistics. DL carried out dermatoglyphic assessments and assist in statistics. LF participated in design and data analysis. JW and OS participated in the study design and analysis.

Competing Interests

None

Acknowledgements

This study arose from the study on low-prevalence (psychotic) disorders, which is part of the National Survey of Mental Health and Well-being, Australia 1997–1998. The National Survey of Mental Health and Well-being was funded by the Commonwealth Department of Health and Aged Care. We wish to gratefully acknowledge the contribution of the late Dr Bogdan Mdzewski, who scored the dermatoglyphic variables. The Stanley Medical Research Institute supported this project.

References

- Weinberger D **Implication of normal brain development for the pathogenesis of schizophrenia** *Arch Gen Psychiatry* 1987, **44**:660-669
- Murray R, O'Callaghan E, Castle D and Lewis S **A neurodevelopmental approach to the classification of schizophrenia** *Schizophr Bull* 1992, **18**:319-332
- Harrison G, Glazebrook C, Brewin J, Cantwell R, Dalkin T, Fox R, Jones P and Medley I **Increased incidence of psychotic disorders in migrants from the caribbean to the United Kingdom** *Psychol Med* 1997, **27**:806
- Cummins H and Midlo C *Finger prints, Palms and Soles Philadelphia: The Blakiston Company* 1943,
- Babler W **Embryologic development of epidermal ridges and their configurations** In: *Dermatoglyphics: Science in transition. Birth defects* (Edited by: CC P, RM G, BA S) New York: Wiley-Liss 1991, 95-112
- McGrath J and Murray R **Risk factors for Schizophrenia: from conception to birth** In: *Schizophrenia* (Edited by: SR H, DR W) Victoria: Blackwell Science Ltd 1995, 187-205
- van Os J, Woodruff P, Fananas L, Ahmed F, Shuriquie N, Howard R and Murray R **Association between cerebral structural abnormalities and dermatoglyphic ridge counts in schizophrenia** *Compr Psychiatry* 2000, **41**:380-384
- Van Valen L **A study of fluctuating asymmetry** *Evolution* 1962, **16**:125-142
- Mellor C **Dermatoglyphic evidence of fluctuating asymmetry in schizophrenia** *Br J Psychiatry* 1992, **160**:467-472
- Palmer A **Fluctuating asymmetry analysis: a primer** In: *Developmental Instability: its origins and evolutionary implications* (Edited by: TA M) Dordrecht: Kluwer 1994, 335-364
- Palmer R and Strobeck C **Fluctuating asymmetry: measurement, analysis, patterns** *Annu Rev Ecol Syst* 1986, **15**:479-499
- Arrieta M, Criado B, Martinez B, Lobato M, Gil A and Lostao C **Fluctuating dermatoglyphic asymmetry: genetic and prenatal influences** *Annals Hum Biol* 1993, **20**:557-563
- Martin N, Jinks J, Berry H and Loesch D **A genetical analysis of diversity and asymmetry in finger ridge counts** *Heredity* 1982, **48**:393-405
- Markow T and Wandler K **Fluctuating dermatoglyphic asymmetry and the genetics of liability to schizophrenia** *Psychiatr Res* 1986, **19**:323-328
- Livshits G and Kobylanski E **Dermatoglyphic traits as possible markers of developmental processes in humans** *Am J Med Genet* 1987, **26**:111-122
- Jantz R and Brehme H **Directional and fluctuating asymmetry in the palmar interdigital ridge-counts** *Anthropol Anz* 1993, **51**:59-67
- Loesch D and Martin N **Directional and absolute asymmetry of digital ridge counts** *Acta Anthropologic* 1982, **6**:85-98
- Harrison P **The neuropathology of schizophrenia. A critical review of the data and their interpretation** *Brain* 1999, **122**:593-624
- Markow T and Gottesman I **Fluctuating dermatoglyphic asymmetry in psychotic twins** *Psychiatr Res* 1989, **29**:37-43
- Loesch D *Quantitative dermatoglyphics classification, genetics and pathology* New York: Oxford University Press 1983,
- Davis J and Bracha H **Prenatal growth markers in schizophrenia: a monozygotic co-twin control study** *Am J Psychiatry* 1996, **153**:1166-1172
- Fearon P, Lane A, Airie M, Scannell J, McGowan A, Bynre M, Cannon M, Cotter D, Murphy P, Cassidy B, Waddington J, Larkin C and Callaghan E **Is reduced dermatoglyphic a-b ridge count a reliable marker of developmental impairment in schizophrenia** *Schizophr Res* 2001, **50**:151-157
- Fananas L, Moral P and Bertranpetit J **Quantitative dermatoglyphics in schizophrenia: study of family history subgroups** *Hum Biol* 1990, **62**:421-427
- Fananas L, van Os J, Hoyos C, McGrath J, Mellor C and Murray R **Dermatoglyphic a-b ridge count a s possible marker for developmental disturbance in schizophrenia: replication in two samples** *Schizophr Res* 1996, **20**:307-314
- Jelovac N, Milicic J, Milas M, Dodig G, Turek S and Ugrenovic Z **Dermatoglyphic analysis in bipolar affective disorder and schizophrenia-" continuum of psychosis" hypothesis corroborated** *Coll Anthropol* 1999, **23**:589-595
- Turek S **Dermatoglyphics and schizophrenia: analysis of quantitative traits** *Coll Anthropol* 1990, **14**:137-150
- Rosa A, Fananas L, Marcellis M and van Os J **a-b ridge counts and schizophrenia** *Schizophr Res* 2000, **46**:285-286
- Torrey E, Taylor E, Bracha H, Bowler A, McNeil T, Rawlings R, Quinn P, Bigelow L, Rickler K, Sjostrom K, Higgins E and Gottesman I **Prenatal origin of schizophrenia in a subgroup of discordant monozygotic twins** *Schizophr Bull* 1994, **20**:423-431
- Bracha H, Torrey F, Gangestad I, Bigelow L and Cunniff C **Second-trimester markers of fetal size in schizophrenia: a study of monozygotic twins** *Am J Psychiatry* 1992, **149**:1355-1361
- Reilly J, Murphy P, Bynre M, Larkin C, Gill M, Callaghan E and Lane A **Dermatoglyphic fluctuating asymmetry and atypical handedness in schizophrenia** *Schizophr Res* 2001, **50**:159-168
- Green F, Green H, Bracha S, Satz P and Christenson C **Preliminary evidence for an association between minor physical anomalies and second trimester neurodevelopment in schizophrenia** *Psychiatr Res* 1994, **53**:119-127
- Balgir R **Dematoglyphic studies in affective disorders: an appraisal** *Biol Psychiatry* 1982, **17**:69-82
- Chakraborty D, Mazumdar P, Than M and Singh R **Dermatoglyphic analysis in Malay subjects with bipolar mood disorder** *Med J Malaysia* 2001, **56**:223-226
- Gutierrez B, Van-Os J, Valles V, Guillamat R, Campillo M and Fananas L **Congenital dermatoglyphic malformations in severe bipolar disorder** *Psychiatr Res* 1998, **78**:133-140
- McGrath J, El-Saddi O, Cardy S, Chapple B, Chant D and Mowry B **Urban birth and migrant status as risk factors for psychosis: an Australian case-control study** *Soc Psychiatry Psychiatr Epidemiol* 2001, **36**:533-536
- McGrath J, El-Saadi O, Grim V, Cardy S, Chapple B, Chant D, Lieberman D and Mowry B **Minor physical anomalies and quantitative**

- measures of the head and face in patients with psychosis *Arch Gen Psychiatry* 2002, **59**:458-464
37. Jablensky A, McGrath J, Herman H, Castle D, Gureje O, Morgan V and Korten A *People living with psychotic illness: An Australian study 1997-1998 Canberra: Commonwealth of Australia* 1999,
 38. Jablensky A, McGrath J, Herrman H, Castle D, Gureje O, Evans M, Carr V, Korten A and Harvey C **Psychotic disorders in urban areas: an overview of the Study on Low Prevalence Disorders** *Aust N Z J Psychiatry* 2000, **34**:221-236
 39. Robins L **The Composite International Diagnostic Interview. An eoidemiological Instrument suitable for use in conjunction with different diagnostic systems and in different cultures** *Arch Gen Psychiatry* 1988, **45**:1069-1077
 40. Bebbington P and Nayani T **The Psychosis Screening Instrument Questionnaire** *Int J Methods Psychiatric Res* 1995, **5**:11-19
 41. Wing J, Babor T, Brugha T, Burke J, Cooper J, Giel R, Jablensky A, Regier D and Sartorius N **SCAN: Schedule for Clinical Assessment in Neuropsychiatry** *Arch Gen Psychiatry* 1990, **47**:589-9315
 42. Association AP *DSM-III-R: Diagnostic and Statistical Manual of mental Disorders* Washington DC: American Psychiatric Association 1987,
 43. Holt S **The correlations between ridge counts on different fingers estimated from a population sample** *Hum Genet* 1959, **23**:459-460
 44. Miller R *Beyond ANOVA: Basics of applied statistics* New York: Wiley & Sons 1986,
 45. Fisher R **On the 'probable error' of a coefficient of correlation deduced from a small sample** *Metron* 1921, **1**:3-32
 46. Micle S and Kobylanski E **Sex differences in the intra-individual diversity of finger dermatoglyphics: pattern types and ridge counts** *Hum Biol* 1988, **60**:123-134
 47. Sokal R and Rohlf F *Biometry* New York: W H Freeman 1981,
 48. Jantz R **On the levels of dermatoglyphic variation** *Dermatoglyphics-Fifty Years Later (Edited by: W W, C P)* New York: Alan R Liss 1979, 53-61
 49. McGrath J and Welham J **Season of birth and schizophrenia: a systematic review and meta-analysis of data from the Southern Hemisphere** *Schizophr Res* 1999, **35**:242
 50. Yeo R, Gangestad S, Thoma R, Shaw P and Repa K **Developmental instability and cerebral lateralization** *Neuropsychology* 1997, **11**:552-561

Pre-publication history

The pre-publication history for this paper can be accessed here:

<http://www.biomedcentral.com/1471-244X/3/3/prepub>

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