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The Presentation of Dermatoglyphic Abnormalities in Schizophrenia: A Meta-Analytic Review

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Abstract

Within a neurodevelopmental model of schizophrenia, prenatal developmental deviations are implicated as early signs of increased risk for future illness. External markers of central nervous system maldevelopment may provide information regarding the nature and timing of prenatal disruptions among individuals with schizophrenia. One such marker is dermatoglyphic abnormalities (DAs) or unusual epidermal ridge patterns. Studies targeting DAs as a potential sign of early developmental disruption have yielded mixed results with regard to the strength of the association between DAs and schizophrenia. The current study aimed to resolve these inconsistencies by conducting a meta-analysis examining the six most commonly cited dermatoglyphic features among individuals with diagnoses of schizophrenia. Twenty-two studies published between 1968 and 2012 were included. Results indicated significant but small effects for total finger ridge count and total A-B ridge count, with lower counts among individuals with schizophrenia relative to controls. Other DAs examined in the current meta-analysis did not yield significant effects. Total finger ridge count and total A-B ridge count appear to yield the most reliable dermatoglyphic differences between individuals with and without schizophrenia.

Keywords

schizophrenia; dermatoglyphics; meta-analysis; neurodevelopment

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1. Introduction

The neurodevelopmental model posits that schizophrenia is caused in part by disruptions in central nervous system (CNS) development beginning as early as the prenatal period (Weinberger, 1987 and 1995). In addition to genetic factors, prenatal factors such as infection (see Brown and Derkits, 2010; Mittal et al., 2008a), maternal stress exposure (Khashan et al., 2008), and obstetric complications (OCs; Dalman et al., 1999; Lewis and Murray, 1987; McNeil and Cantor-Graae, 2000) have all been linked with increased risk for schizophrenia (for review see Mittal et al., 2008b; Walder et al., in press[a]). These prenatal insults are thought to adversely impact CNS development and can be assessed indirectly in the atypical presentation of morphologic traits (e.g., Haukvik et al., 2010; Qiu et al., 2010; Van Erp et al., 2002).

One such class of morphologic traits is dermatoglyphic abnormalities (DAs). Dermatoglyphic features are relatively stable and cosmetically insignificant epidermal ridge patterns that form prints on the fingers, hands, and soles. DAs are thought to represent, in part, the impact of prenatal insults, thus providing a window into the timing and nature of early development (Cummins and Midlo, 1961; Davis and Bracha, 1996; Lobato et al., 2001). The development of dermatoglyphics overlaps temporally with neuronal migration (Bracha et al., 1991). Although the precise origins of DAs remain unclear, their presence may suggest prenatal disruption relevant to the formation of neural structures implicated in future development of schizophrenia.

Several indices have been used to capture DAs. Measures typically rely on epidermal ridge counting, which makes use of the triradius, or meeting point of three opposing ridge systems. Commonly used indices include 1) finger ridge counts (the number of ridges between the core of the finger pattern and its corresponding triradius), 2) palmar ridge counts (the number of ridges crossing a line superimposed on the palm print connecting two triradii), 3) fingertip patterns (shapes created by the patterns of finger ridges characterized as whorls, ulnar/radial loops, and arches), 4) ATD angle (the angle formed by lines drawn from triradius *t*, the most distal axial triradius near the base of the palm, to triradii *a* and *d*, located proximal to the index and little fingers, respectively), and 5) fluctuating asymmetries (differences in ridge counts or pattern types between parallel structures on the left and right hands) (Cummins and Midlo, 1961, Palmer, 1994).

DAs such as ridge counts and fingertip patterns may reflect different developmental responses to a range of specific insults rather than a unitary construct indicating generic developmental disruption (Compton and Walker, 2009). Some DAs appear to be more influenced by environmental causes, with others closely linked to genetic factors (e.g., Bracha et al., 1991; Bracha et al., 1992; Bramon et al., 2005; Holt, 1968; van Oel et al., 2001). For example, from an environmental perspective, mild prenatal stress is associated with greater dermatoglyphic asymmetry among macaque offspring (Newell-Morris et al., 1989). Similarly, among humans, prenatal maternal stress during the period of finger ridge development (weeks 14–22) is associated with more DAs among offspring (King et al., 2009). Evidence indicating a link between chromosomal anomalies and alterations in dermatoglyphic and palmar flexion creases suggests genetic influences on the formation of DAs as well (Reed, 1981). Deviant ATD angles have been associated with chromosomal syndromes; namely, in 22q Deletion Syndrome patients, those with mental retardation had greater ATD angles than those with psychotic symptoms, who both had greater angles than healthy controls (Martín et al., 2004). More specifically pertaining to schizophrenia, a family study of people with a schizophrenia spectrum disorder and their first-degree relatives found associations between both genetically-influenced ectodermal derivate abnormalities (e.g., ridge dissociation, abnormal palmar flexion creases) and

environmentally-influenced abnormalities such as total a-b ridge count and schizophrenia vulnerability (Fatjó-Vilas et al., 2008).

Although the noted studies have suggested that specific DAs share some etiology with schizophrenia, the overall association between DAs and schizophrenia remains unclear. Bramon and colleagues' (2005) meta-analytic review of nine studies (published between 1983 and 2003) examining palmar a-b ridge count in individuals with schizophrenia compared to controls yielded a pooled standardized effect size of 0.39 ($p = .03$). There is, however, substantial variability in effect sizes across published DA studies. For example, Bramon and colleagues found significant heterogeneity in effect sizes ($p<.0001$) ranging from −0.05 to 1.15, indicating between-group differences ranging from negligible to substantive.

To date, research on dermatoglyphics has been relatively limited, and the available literature has been complicated by methodological inconsistencies (for a review see Compton and Walker, 2009). For example, variation in techniques for quantifying DAs has likely contributed to discrepancies in the literature, rendering cross-study comparisons challenging. Further, modest sample sizes typically used in this line of research may limit statistical power to detect group differences. Efforts to clarify dermatoglyphic differences between people with schizophrenia and controls using systematic review approaches have been limited. Bramon and colleagues' meta-analysis, for example, included only nine studies and limited examination to a single dermatoglyphic measure (2005). Understanding DAs in the context of a more comprehensive meta-analysis stands to correct for the limited power problem in the dermatoglyphic literature and enable comparison between multiple dermatoglyphic markers. The current study aims to fill this gap in the literature by conducting an updated (including studies from 1968 to 2012) and comprehensive (examining the six most cited dermatoglyphic features) meta-analytic review of dermatoglyphic abnormalities in schizophrenia patients relative to nonclinical (healthy) controls.

2. Methods

2.1 Literature Search and Selection

This meta-analysis included peer-reviewed articles examining a range of dermatoglyphic measures in individuals with schizophrenia and controls. Relevant articles were identified using the electronic databases PubMed and PsycINFO, using search terms "schizophrenia and dermatoglyphic*" and "psychosis and dermatoglyphic*" between January 1968 to June 2012. Reference lists of identified articles were explored for additional articles. Inclusion criteria were modeled after Weinberg and colleagues' (2007) meta-analysis of minor physical anomalies, which similarly reflect neurodevelopmental disruption (Compton and Walker, 2009). Inclusion criteria were 1) case-control design, 2) available published means and standard deviations, 3) participants with specific diagnoses of schizophrenia obtained using established diagnostic procedures, 4) data not overlapping between published studies, and 5) availability in English. Studies that included unique dermatoglyphic features that were only represented in one publication, and did not appear in any other published research, were excluded. In total, 22 studies were included and 43 were excluded (see Table 1 for a list of studies excluded from current analyses).

2.2 Dermatoglyphic Measures

Six dermatoglyphic indices were examined: total finger ridge count, total a-b palmar ridge count, fingertip pattern asymmetry, ATD angle, fluctuating asymmetry of finger ridge count, and palmar a-b fluctuating asymmetry.

2.2.1 Ridge counts—Ridge counting uses the triradius, or the meeting point of three opposing ridge systems (see Figure 1). Total finger ridge count (TFRC; number of ridge counts between the core of the finger pattern and its corresponding triradius; arch patterns do not have triradii and receive a ridge count of zero) and total a-b palmar ridge count (TABRC; number of palmar ridges crossing a line superimposed on a palm print that connects triradius *a*, located proximal to the index finger, and triradius *b*, located proximal to the middle finger) are two such types of epidermal ridge counts (Green et al., 1994).

2.2.2 Fingertip patterns—Fingertip patterns are typically identified as whorls, loops, or arches, depending on the number of triradii, namely two, one, and zero, respectively (Mellor, 1968). Whorls are purported to be more complex than loops, which are in turn more complex than loops and arches; individuals with more arches than whorls are theorized to have simplified fingertip ridge patterns (Cummins and Midlo, 1961). As whorls by definition have higher ridge counts than loops and arches, the same mechanisms leading to lower ridge count may account for pattern simplification. Figure 1 shows an example of the finger ridge count technique of an ulnar loop pattern and the identification of the corresponding triradius. Figure 2 shows an illustration of the landmarks used to quantify a-b palmar ridge counts.

2.2.3 ATD Angle—The ATD angle is a dermatoglyphic feature that compares the length of the hand to the width by measuring the angle created by superimposing lines on the palm print from the axial triradius ("t") at the base of the palm to the "a" and "d" triradii of the palm located proximal to the 2nd and 5th digits, respectively (Elizarrarás-Rivas et al., 2002).

2.2.4 Fluctuating Asymmetry (FA)—FA refers to random differences between parallel structures on the left and right sides of the body (Mellor, 1992). Greater right-left (R-L) differences indicate greater FA (van Valen, 1962; Palmer and Strobeck, 1986; Mellor, 1992; Palmer, 1994). Palmar A-B fluctuating asymmetry (or fluctuating TABRC; FABRC) is determined by subtracting right TABRC from left TABRC, and dividing that number by right TABRC plus left TABRC. Similarly, TFRC fluctuating asymmetry (or fluctuating asymmetry of total finger ridge count; FAFRC) is determined by subtracting right TFRC from left TFRC and dividing that number by the sum of TFRC from both hands (Green et al., 1994; van Oel et al., 2001).

2.3 Statistical analysis

MIX 2.0 (Bax, 2011) meta-analysis software was used for all analyses. Each dermatoglyphic feature was analyzed separately. Thus, data from an individual study can be used in multiple sub-analyses. Hedges' *g* effect sizes, 95% confidence intervals, and *z*-scores were calculated separately for each study subset. Hedges' *g* provides a measure of effect size while correcting for biases due to small sample sizes. As with Cohen's *d*, a Hedges' *g* effect size of 0.2 is considered small, 0.5 moderate, and 0.8 large (Cohen, 1992). The direction of the effect was positive when scores for the schizophrenia group were larger than scores for the control group. A *Q* statistic was then calculated to examine variance across effect sizes within each data subset. The *Q* statistic tests the null hypothesis that the studies' effect sizes are homogeneous (Cochran, 1954). Data subsets were analyzed using fixed-effects models when the *Q* statistic test demonstrated that the effect size of the population did not demonstrate significant heterogeneity; those whose *Q* statistic suggested significant heterogeneity among studies were analyzed using a random-effects model (Becker, 1996; Field, 2003; Hunter and Schmidt, 2000).

To determine the potential effects of the "file-drawer problem" (i.e., negative or non-significant findings tend to go unpublished), Rosenthal's fail-safe *N* statistic was calculated

for each data subset (Rosenthal, 1979). The fail-safe *N* provides an assessment of sampling bias in that it estimates how many hypothetical studies with an effect size of zero would need to be incorporated into the analysis to render the found *p*-value non-significant. In addition, Begg and Mazumdar's adjusted rank correlation test was employed to test for publication bias (1994).

3. Results

The results of each individual meta-analysis are summarized in Table 2.

3.1 Total Finger Ridge Count

Table 3 provides raw data, effect sizes, and *z*-values for the 18 studies reporting TFRC data. Absolute effect sizes for the three TFRC datasets were significantly heterogeneous ($Q = 56.96$, $p<.001$) and ranged from $g = 0.01$ (small) to $g = 0.71$ (moderate) (Cohen, 1988). Begg's test indicated that there was no evidence of a significant publication bias ($z = 0.95$, $p = .34$). The tests performed on TFRC studies resulted in a significant effect size ($g = -0.20$, 95% C.I. = −0.27–0.13, $p<.05$), with lower TFRC among patients with schizophrenia relative to controls. Figure 3 presents a forest plot of TFRC effect sizes.

3.2 Total A-B Ridge Count

Table 4 provides raw data, effect sizes, and z-values for 18 studies reporting TABRC data (including 9 studies not included in the Bramon et al. [2005] meta-analysis). Absolute effect sizes for TABRC were significantly heterogeneous ($Q = 151.00$, $p<.001$) and ranged from $g = 0.03$ (small) to $g = 1.11$ (large). Begg's test indicated that there was no evidence of a significant publication bias ($z = 0.27$, $p = .79$). Analyses of TABRC across all studies resulted in a significant effect size ($g = -0.31$, 95% C.I. = −0.38–0.24, $p<.01$), with lower TABRC among patients with schizophrenia. Figure 4 presents a forest plot of all TABRC effect sizes.

3.3 ATD Angle

Table 5 provides raw data, effect sizes, and z-values for 10 studies reporting total ATD angle data. ATD angle absolute effect sizes were significantly heterogeneous ($Q = 44.49$, $p<.001$) and ranged from $g = 0.01$ (small) to $g = 0.84$ (large). The meta-analysis was non-significant ($g = -0.10$, 95% C.I. = −0.20–0.01). Begg's test indicated that there was no evidence of a significant publication bias ($z = -0.26$, $p = .79$). Figure 5 presents a forest plot of ATD angle effect sizes.

3.4 Fingertip Pattern Asymmetry - Three-Pattern Classification

Table 6 provides raw data, effect sizes, and *z*-values for four studies reporting three-pattern classification (i.e., whorl, loop, or arch) fingertip pattern asymmetry data. Overall, there was a non-significant effect size of $g = 0.25$ (95% C.I. = −0.08–0.59) and significant between-study heterogeneity ($Q = 11.75$, $p<.05$). Begg's test indicated that there was no evidence of a significant publication bias ($z = -0.25$, $p = .81$).

3.5 Fluctuating Asymmetry Finger Ridge Count (FAFRC)

Table 7 provides raw data, effect sizes, and *z*-values for three studies reporting FAFRC data. The effect size for all datasets combined was not significant ($g = 0.31$, 95% C.I. = −0.50–1.12); there was significant heterogeneity across studies ($Q = 54.17$, $p<.001$). Begg's test indicated that there was no evidence of a significant publication bias ($z = -0.68$, $p = .50$).

3.6 Fluctuating Asymmetry A-B Ridge Count (FABRC)

Table 8 provides raw data, effect sizes, and *z*-values for three studies reporting FABRC data. The absolute effect size was moderate ($g = 0.75$) and non-significant; there was significant heterogeneity among studies ($Q = 146.89$, $p < .001$). Begg's test indicated that there was no evidence of a significant publication bias ($z = 1.36$, $p = .17$).

4. Discussion

4.1 General Conclusions

This meta-analysis in part supports a neural diathesis-stress model of schizophrenia (See Bracha et al., 1991; Walker and Diforio, 1997), demonstrating small effect size differences between individuals with schizophrenia and controls across DAs that likely reflect early developmental deviation. The two DAs analyzed using the largest overall sample sizes revealed the most reliable and robust (albeit small) effect sizes; namely, TFRC ($g = -0.20$, $p < .05$; fail-safe $N = 83$) and TABRC ($g = -0.31$, $p < .01$; fail-safe $N = 238$). These findings are consistent with a prior meta-analysis that included approximately half the number of studies and focused on palmar ridge count in schizophrenia (Bramon et al., 2005). Among DA measures, TFRC and TABRC appear to most reliably differentiate people with and without schizophrenia and thus, appropriately, are the DAs most often reported in the schizophrenia research literature. It is notable, however, that these two features are also the most widely studied, resulting in sample sizes far greater than other dermatoglyphic features. Although this disparity should be taken into account when considering the current results, the present state of the literature precludes a definitive answer as to whether this robustness is due to true population differences or subject to change if future research were to further investigate the less-studied dermatoglyphic features.

4.2 Timing of Prenatal Disruptions

Some dermatoglyphic features have been tied to specific stages of prenatal development. This information can be used to make assumptions about the timing of neurodevelopmental disruptions influencing schizophrenia vulnerability. For example, TFRC is thought to reflect the speed of fetal growth, with more ridges indicating faster cell division during the first and second trimester of gestation (Cohen-Bendahan et al., 2005). In contrast, TABRC is largely believed to reflect later fetal development and to be more influenced by non-shared environmental factors (Bracha et al., 1991; Bracha et al., 1992; Bramon et al., 2005; van Oel et al., 2001). More specifically, although the a-b ridges are among the first to appear (i.e., interdigital area II), they develop over a longer period of time, and thus may be more sensitive to a variety of developmental disturbances relative to finger ridge formation (Fañanás et al., 1996a; Rose, 1987). That TFRC and TABRC yielded significant effects across studies suggests that schizophrenia may be more influenced by disruptions occurring at multiple stages across prenatal development.

4.3 DA Variability

Despite TFRC and TABRC yielding significant group differences between individuals with schizophrenia and controls, there was considerable variability across studies. For instance, some studies reported lower TFRC in people with schizophrenia, while others reported higher TFRC count in people with schizophrenia. Nonetheless, all statistically significant findings were in the direction of reduced TFRC among people with schizophrenia.

Due to local differences, samples may contain individuals with varying mean levels and types of prenatal disruptions, leading to mean differences in DA patterns and confounding omnibus results (Cantor-Graae et al., 1998; Green et al., 1994; Bracha et al., 1992). In other words, distinct prenatal events differentially impact specific dermatoglyphic features, all of

which may be etiologically related to schizophrenia. For example, disrupted neurodevelopment due to intrauterine growth restriction may result in abnormal decreases ("simplification") of finger and palmar ridges. Further, prenatal edema results in abnormal increases in ridges (Bracha et al., 1992; Bracha et al., 1995). In addition, both human and animal research suggests that prenatal maternal stress during the period of fingerprint development (weeks 14–22) results in greater dermatoglyphic asymmetry among offspring (King et al., 2009; Newell-Morris et al., 1989). In contrast, ectodermal derivate abnormalities (e.g., ridge dissociation; abnormal palmar flexion creases) appear to reflect primarily genetically influence (Fatjó-Vilas et al., 2008). It is also important to note that prenatal insults do not necessary reflect a purely environmental contribution, as an emerging literature suggests that genes can adversely affect the prenatal environment or lead to additional obstetric complications as well (Boin et al., 2001; Engel et al., 2005;Katila et al., 1999). For example, polymorphisms associated with abnormalities in immune function (e.g., an exaggerated inflammatory response to infection) may render a mother and fetus more susceptible to the deleterious effects of prenatal events and lead to fetal neuronal injury (see Ellman and Cannon, 2008 for a review). Any of these potential causes of developmental disruption may also vary locally between populations. Thus the heterogeneity of insults may differentially influence specific DAs among groups of people with schizophrenia, obscuring potential differences between patients and controls. In some cases it may be more fruitful to identify individuals who fall outside (above or below) the expected 'normative' range of DAs, as reflected by relatively increased or decreased ridge counts.

4.4 Limitations

There are a number of important limitations in this meta-analysis. First, we excluded studies of individuals with schizophrenia spectrum disorders or those at-risk for schizophrenia. In light of evidence of DAs in at-risk groups (Chok et al., 2005; Daly et al., 2008; Langsley et al., 2005), future reviews across a broader range of diagnostic groups is warranted to see if patterns hold along a continuum of psychotic symptom manifestation. Second, the overall number of studies was relatively small, limiting power to detect statistical significance. Third, utilizing fixed-effects models in meta-analyses has the potential of increasing Type I error rates. This is particularly problematic when analyses include more than 20 studies and when authors assume homogeneous population parameters. Nevertheless, alternative strategies are lacking with respect to analyses that include fewer studies. In the current study, we aimed to utilize the most accurate effects models by analyzing population heterogeneity (Field, 2003). Fourth, methodological and demographic variability across studies may have impeded accurate effect size estimates and contributed to the sizable heterogeneity in means and effect sizes. The anthropology literature cites longstanding evidence of racial/ethnic group differences (e.g., Cummins and Midlo, 1961; Jantz, 1987; Rosner and Steinberg, 1968), and more recently geographic region of origin differences (e.g., Arrieta et al., 2003; Karmarkar and Kobyliansky, 2009; Karmarkar et al., 2005; Zhang et al., 2010). For example, northern and southern Chinese populations can be differentiated based on dermatoglyphic analysis (Zhang et al., 2010). Not all studies included in the current meta-analysis matched cases and controls on ethnicity or region of origin. This collapsing (or lack of matching) across ethnically or geographically diverse samples may have masked true diagnostic group differences. Likewise, few studies reported data separately by sex, restricting examination of sex effects.

It is noteworthy, however, that in a series of studies, Karmakar, Kobyliansky and colleagues consistently and accurately classified males and females across various ethnic groups by applying discriminant analyses to several derived qualitative and quantitative dermatoglyphic traits (Karmakar and Kobyliansky, 2009; Karmakar et al., 2003; Kobyliansky and Micle, 1988; Micle and Kobyliansky, 1991; Micle and Kobyliansky,

1986). The authors posit that these sex differences indicate common genetic components within the two major classes of dermatoglyphic traits that are at least in part sexually dimorphic. The degree to which this carries across diagnostic groups is as yet unclear. As with any meta-analysis, results are only as reliable as the studies included.

4.5 Conclusions

Findings support DAs as one among other indicators of neurodevelopmental disruption, including other types of minor physical anomalies (e.g., Compton et al., 2007; Mittal et al., 2008b; Mittal and Walker, 2011; Weinstein et al., 1999; Schiffman et al., 2002; Golembo et al., 2012), structural and functional neuroanatomical abnormalities (Karlsgodt et al., 2010; Niznikiewicz et al., 2003; Shepherd et al., 2012), neuromotor abnormalities (Schiffman et al., 2004; Walker et al., 1994), cortisol abnormalities (Walder et al., in press[b]; Walker and Walder, 2002), immune alterations (DeLisi, 1996; Müller et al., 2000), and neurocognitive deficits (Walder et al., 2008) that may serve as biomarkers of schizophrenia risk. Future research examining the combined effects of these markers may lead to significant strides in the etiological understanding of schizophrenia.

Additionally, conceptual strategies such as viewing schizophrenia within the context of equifinality (Cicchetti and Rogosch, 1996) or as a series of subtypes, each the product of partially distinct and/or somewhat overlapping etiologies (Gay et al., 2012; Walsh et al., 2008), may help to explain the heterogeneous findings noted in the present review. Individuals with schizophrenia with more DAs may represent a subtype characterized by a more adverse prenatal environment. Studies examining minor physical anomalies and dermatoglyphics within an equifinality framework hold promise for elucidating our understanding of this theoretical point of view. Further research might also seek to resolve inconsistencies in DA findings attributable to poorly standardized methods. Consistency in types of dermatoglyphics measures, techniques for obtaining dermatoglyphic features, diagnostic ascertainment, and sample recruitment may yield more reliable and easily comparable results. In addition, reporting raw data for male participants separate from female participants may shed light on potential sex differences. Despite these methodological issues, the current study detected modest but significant evidence for differences in DAs between diagnostic groups, thus lending further support to the neurodevelopmental model of schizophrenia.

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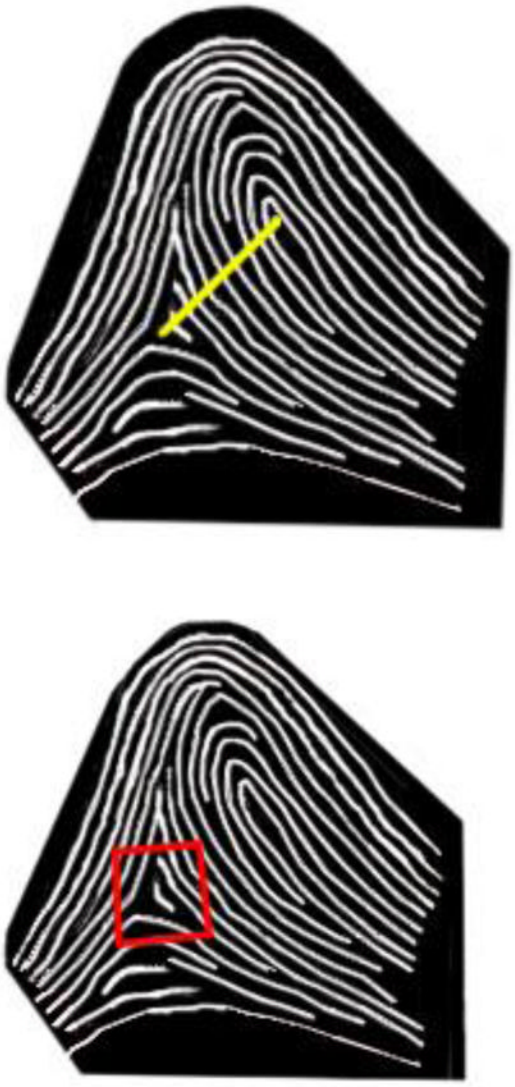

Figure 1. Ulnar loop with corresponding finger ridge line and triradius

The finger print pattern is taken from the left second finger (L2). In the first picture, a red box outlines the triradius of the ulnar loop. The triradius is formed by the convergence of 3 ridges (colored black). In the second picture, a yellow line from the triradius to the core of the fingerprint shows that the ulnar loop has a ridge count of 8. A ridge count is obtained by counting the ridges between the triradius and the core (not counting the ridges the make up the triradii or the core).

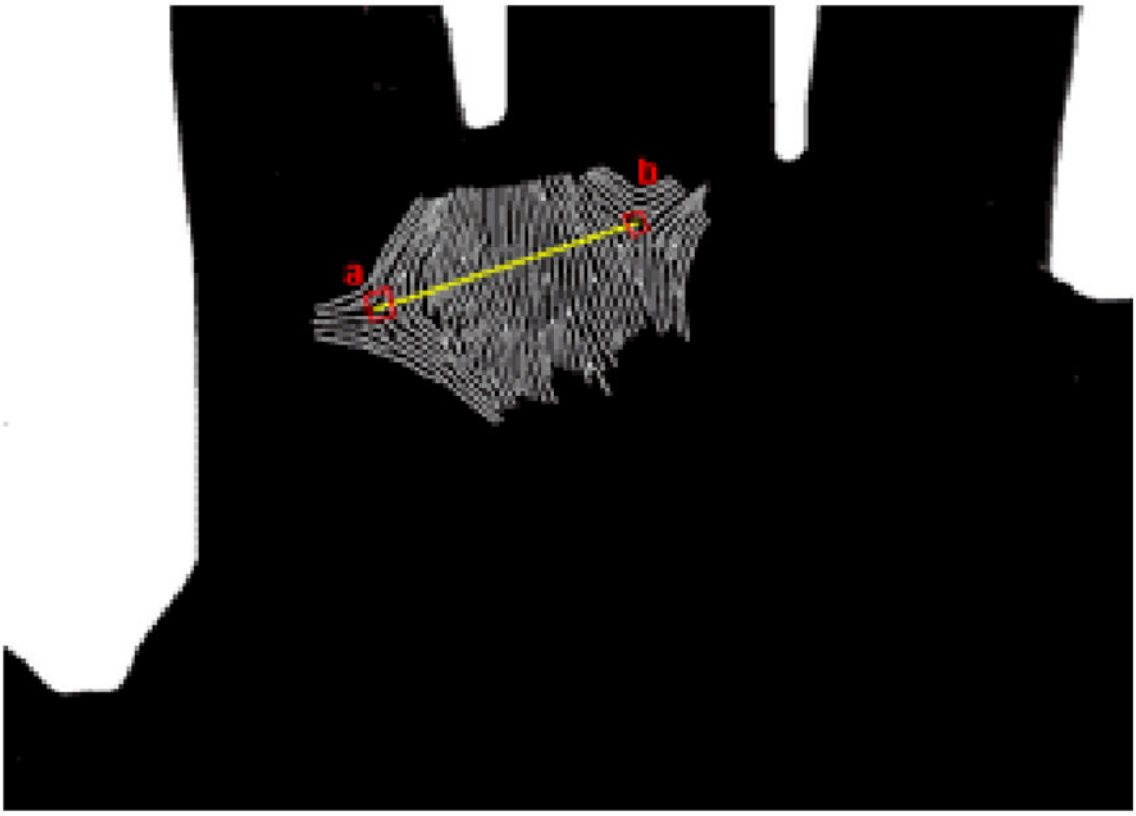

Figure 2. A-B ridge count example

AB Ridge count: The handprint shows an example of an AB ridge count. The AB ridge count is obtained by counting the ridges (colored black) between the triradii found below the second and third finger. The triradii are outlined in red boxes, and a yellow line drawn from one triradius (a) to the other (b) allows one to count the number of ridges between the triradii. Only the ridges that fully cross the line are counted. The AB ridge count is 34 in this example.

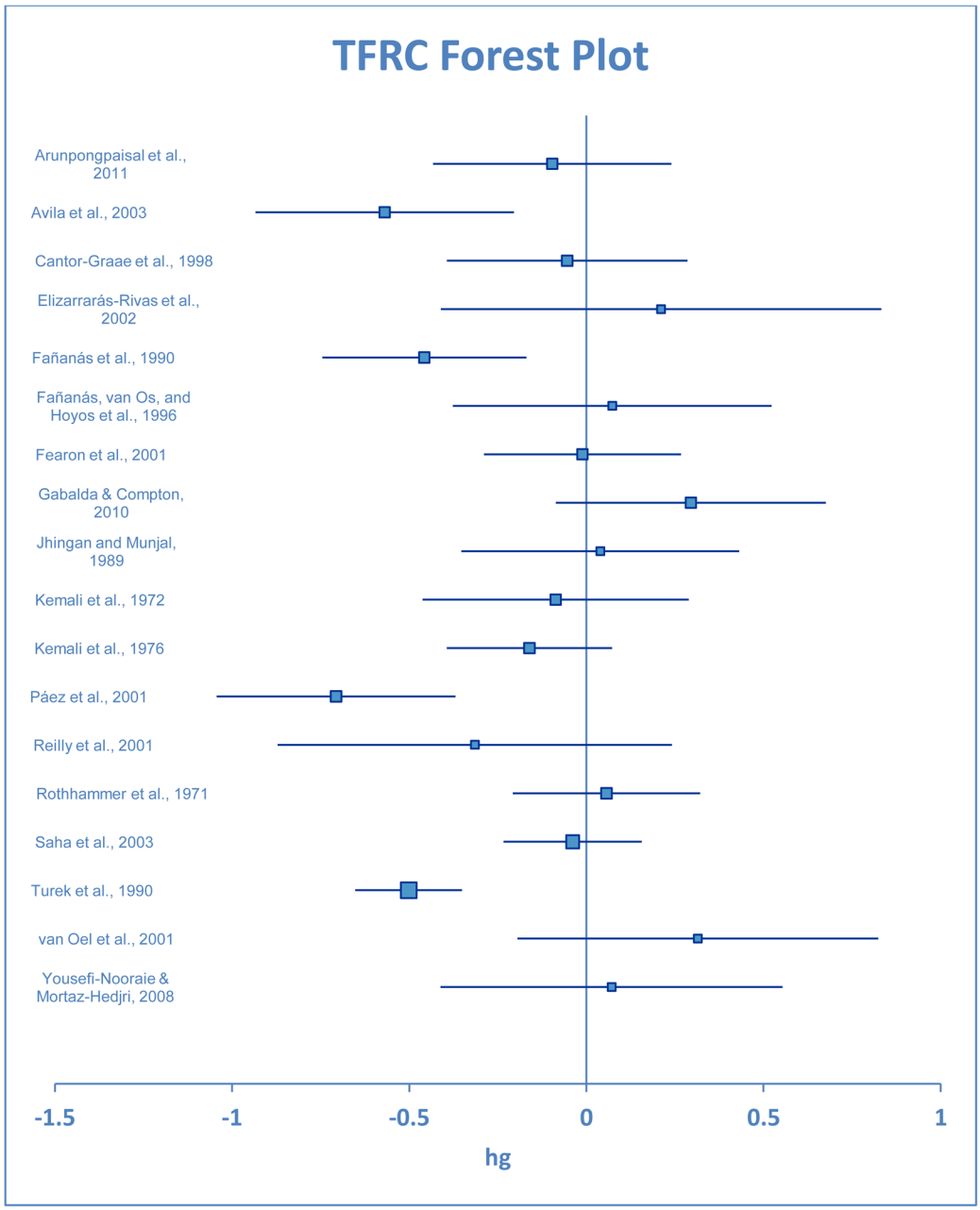

Figure 3.
Forest plot of TFRC meta-analysis

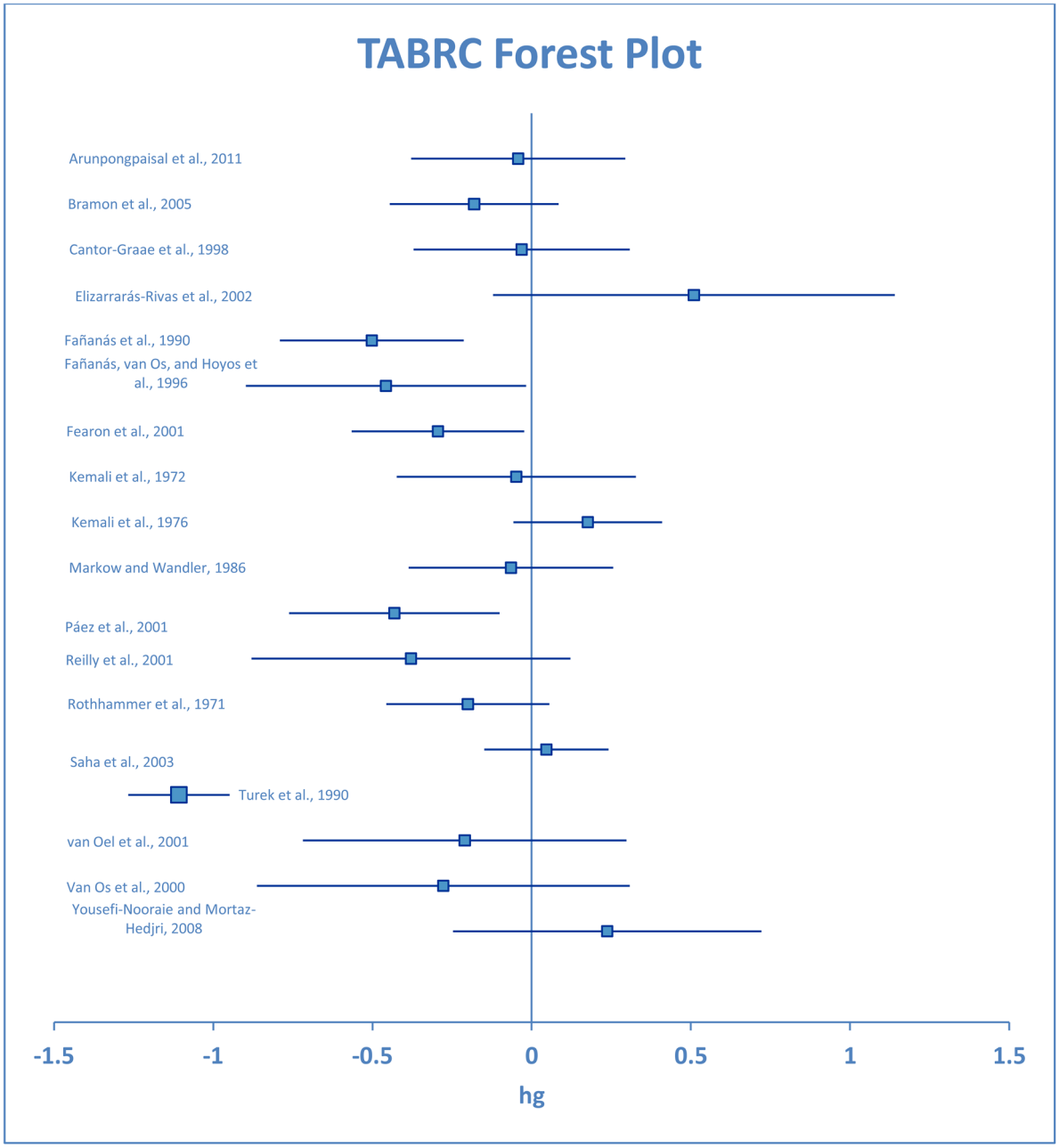

Figure 4.
Forest plot of TABRC meta-analysis

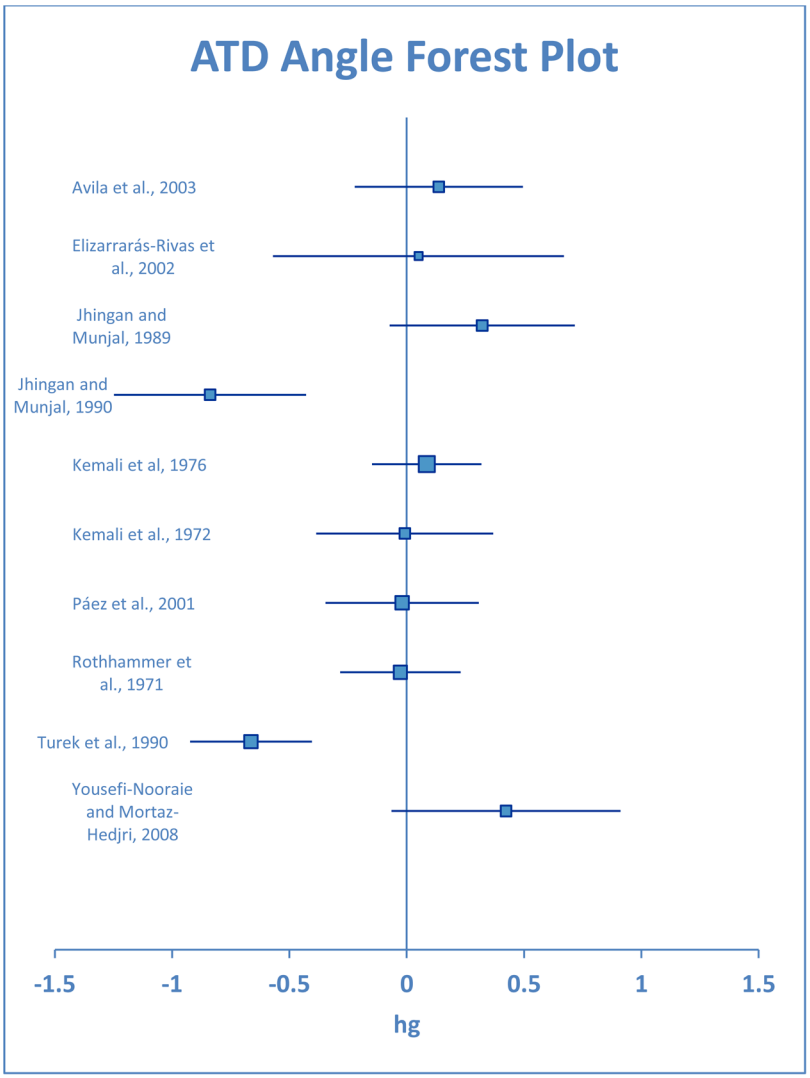

Figure 5.
ATD angle meta-analysis

Table 1

Studies excluded from current analyses

Primary reason for exclusion*	Studies
Not case-control design	Balgir et al., 1980; Bracha et al., 1992; Daly et al., 2008; Davis and Bracha, 1996; Fañanás et al., 1996; Green et al., 1994; Markow and Gottesman, 1989; Kelly et al., 2004; Rosa et al., 2000a; Rosa et al., 2000b; Rosa et al, 2002; Rosa et al., 2005; van Os et al., 1997; Zavala and Núñez, 1970
Raw data or mean scores not available	Arboleda-Flórez et al., 1998; Chary et al, 1996; Mellor, 1992; Srinivasa Murthy and Wig, 1977; Polednak, 1972; Ponnudurai, 1999; Rosner and Steinberg, 1968; Sank, 1968; Sivkov et al., 2007; Sivkov and Akabaliev, 1998; Stowens et al., 1970; Varma et al, 1995
Not purely schizophrenia diagnoses	Barrantes-Vidal et al., 2003; Chok and Kwapil, 2005; Chok et al., 2005; Fatjó-Vilas et al., 2008; Weinstein et al., 1999
Published elsewhere/Used previously published data	Fañanás et al., 1996 (CSM sample); Hilbun, 1970; Jelovac et al., 1999; Jelovac et al., 1995; Johnstone et al., 2005; Mellor, 1968; Rosa et al., 2003
Not available in English	Dvořáková and Zvolský, 1979; Náměstek and Hronek, 1974; Perkovic, 1977; Tényi et al., 2000
Unique dermatoglyphic measure	Shakibaei et al., 2011

* Some studies were excluded for multiple reasons

Table 2

Summary of meta-analyses results

DA Feature	*n* SZ	*n* Control	Effect size (*g*)	95% LL	95% UL	*z*-score	*Q*	Fail-safe *N*
TFRC	1626	1690	−0.20 *	−0.27	−0.13	−5.48	56.96 ***	83
TABRC	1699	1736	−0.31 **	−0.38	−0.24	−8.71	151.00 ***	238
ATD angle	819	687	−0.10	−0.20	0.01	−1.81	44.49 ***	43
Fingertip asymmetry (three-pattern)	249	227	0.25	−0.08	0.59	1.490	11.75 *	4
FAFRC	233	298	0.31	−0.50	1.12	0.75	54.17 ***	15
FABRC	241	298	0.75	−0.65	2.13	1.05	146.89 ***	14

* $p<0.05$,

** $p<0.01$,

*** $p<0.001$

Table 3

Total Finger Ridge Count (TFRC)

Study	*n* SZ	SZ mean (SD)	*n* Control	Control mean (SD)	Effect Size (*g*)	95% LL	95% UL	*Z*
Arunpongpaisal et al., 2011	68	135.50 (33.92)	68	139.10 (40.12)	−0.1	−0.43	0.24	−2.33
Avila et al., 2003	86	114.17 (42.24)	46	140 (50.09)	−0.57**	−0.93	−0.2	−3.059
Cantor-Graae et al., 1998††	60	143.44 (52.51)	75	146.09 (45.54)	−0.05	−0.4	0.29	−0.31
Elizarrarás-Rivas et al., 2002	20	161.1 (47.39)	20	153.2 (21.12)	0.21	−0.41	0.83	0.67
Fañanás et al., 1990††	139	122.03 (43.86)	72	143.31 (50.89)	−0.46**	−0.75	−0.17	−3.11
Fañanás et al., 1996b	29	131.7 (47.2)	55	128 (51.6)	0.07	−0.38	0.52	0.32
Fearon et al., 2001	126	126.4 (44.4)	82	126.9 (49)	−0.01	−0.29	0.27	−0.076
Gabalda and Compton, 2010	62	131.84 (56.15)	47	115.6 (52.65)	0.29	−0.09	0.68	−2.76
Jhingan and Munjal, 1989	50	145.5 (33.9)	50	144.06 (38.87)	0.04	−0.35	0.43	0.196
Kemali et al., 1972	55	142.78 (45.36)	54	146.89 (48.99)	−0.09	−0.46	0.29	−0.451
Kemali et al., 1976	217	134.9 (42.23)	105	142.06 (48.82)	−0.16	−0.39	0.07	−1.35
Páez et al., 2001†	72	130.6 (46.87)	72	163.9 (46.90)	−0.71***	−1.04	−0.37	−4.1
Reilly et al., 2001†	19	131.07 (40.71)	37	146.78 (52.95)	−0.32	−0.87	0.24	−1.12
Rothhammer et al., 1971††	80	133.78 (39.80)	176	131.26 (46.15)	0.06	−0.21	0.32	0.42
Saha et al., 2003††	181	137.65 (47.12)	228	139.57 (51.51)	−0.04	−0.23	0.16	−0.39
Turek et al., 1990††	310	116.26 (36.96)	400	137.21 (45.07)	−0.50***	−0.65	−0.35	−6.53
van Oel et al., 2001	19	139.74 (43.88)	70	125.39 (45.52)	0.31	−0.19	0.82	1.21
Yousefi-Nooraie and Mortaz-Hedjri, 2008†	33	154.2 (53.87)	33	150.4 (51.70)	0.07	−0.41	0.55	0.29

† means and standard deviations pooled from left- and right-hand data

†† means and standard deviations pooled from male and female data

* $p<0.05$,

** $p<0.01$,

*** $p<0.001$

Table 4

Total A-B Ridge Count (TABRC)

Study	*n* SZ	SZ mean (SD)	*n* Control	Control mean (SD)	Effect Size (*g*)	95% LL	95% UL	*Z*
Arunpongpaisal et al., 2011	68	76.31 (9.49)	68	76.68 (7.92)	−0.04	−0.38	0.29	−0.09
Bramon et al., 2005	125	79.1 (10.6)	98	81.1 (11.6)	−0.18	−0.45	0.08	−1.33
Cantor-Graae et al., 1998	60	79.94 (9.32)	75	80.24 (9.47)	−0.03	−0.37	0.31	−0.18
Elizarrarás-Rivas et al., 2002	20	89.1 (13.04)	20	83.7 (6.75)	0.51	−0.12	1.14	1.58
Fañanás et al., 1990	139	76.6 (9.78)	72	81.53 (9.8)	−0.50**	−0.79	−0.21	−3.41
Fañanás et al., 1996a	38	76.2 (11.1)	44	81.4 (11.4)	−0.46*	−0.9	0.02	−2.04
Fearon et al., 2001	140	78 (16.1)	85	82.6 (14.7)	−0.29*	−0.56	−0.02	−2.13
Kemali et al., 1972	55	81.38 (10.49)	54	81.89 (10.56)	−0.05	−0.42	0.33	−0.25
Kemali et al., 1976	217	82.94 (10.92)	105	81 (11.09)	0.18	−0.06	0.41	1.48
Markow and Wandler, 1986†	81	83.44 (13.89)	69	84.26 (10.80)	−0.06	−0.39	0.26	−0.4
Páez et al., 2001†	72	80.8 (11.52)	72	86.7 (15.42)	−0.43**	−0.76	−0.1	−2.56
Reilly et al., 2001†	27	79.19 (15.08)	37	84.17 (11.23)	−0.38	−0.88	0.12	−1.48
Rothhammer et al., 1971††	86	78.00 (10.67)	187	82.46 (12.96)	−0.20	−0.46	0.06	−1.53
Saha et al., 2003††	181	84.31 (9.85)	228	83.83 (10.89)	0.05	−0.15	0.24	0.47
Turek et al., 1990††	310	71.63 (10.14)	400	84.14 (12.09)	−1.11***	−1.27	−0.95	−13.64
van Oel et al., 2001	19	78.41 (12.19)	70	80.73 (10.59)	−0.21	−0.72	0.3	−0.81
van Os et al., 2000	28	74.4 (11.5)	19	77.7 (12)	−0.28	−0.86	0.31	−0.93
Yousefi-Nooraie and Mortaz-Hedjri, 2008†	33	84.8 (10.60)	33	82.3 (10.21)	0.24	−0.25	0.72	0.96

† means and standard deviations pooled from left- and right-hand data

†† means and standard deviations pooled from male and female data

* $p<0.05$,

** $p<0.01$,

*** $p<0.001$

Table 5

ATD Angle

Study	*n* SZ	SZ mean (SD)	*n* Control	Control mean (SD)	Effect size (*g*)	95% LL	95% UL	*Z*
Avila et al., 2003	86	84 (11.77)	46	82.53 (8.25)	0.14	−0.22	0.50	0.75
Elizarrarás-Rivas et al., 2002	20	85.84 (18.02)	20	85.16 (5.46)	0.05	−0.57	0.67	0.16
Páez et al., 2001†	72	89.1 (18.84)	72	89.5 (21.80)	−0.02	−0.35	0.31	−0.12
Jhingan and Munjal, 1989	50	84.16 (14.79)	50	79.46 (14.18)	0.32	−0.07	0.72	1.60
Rothhammer et al., 1971††	140	86.39 (13.67))	100	86.74 (11.87)	−0.03	−0.28	0.23	−0.21
Turek et al., 1990††	97	78.56 (10.28)	157	86.35 (12.50)	−0.66***	−0.92	−0.40	−5.01
Jhingan and Munjal, 1990†	50	74.48 (8.37)	50	82.55 (10.6)	−0.84***	−1.25	−0.43	−4.01
Kemali et al, 1976	217	92.54 (19.59)	105	90.96 (15.88)	0.09	−.15	.32	.718
Kemali et al., 1972	54	90.2 (16.27)	54	90.33 (14.24)	−0.008	−0.39	0.37	−0.04
Yousefi-Nooraie and Mortaz-Hedjri, 2008†	33	93 (18.70)	33	85.6 (15.72)	0.42	−0.06	0.91	1.70

† means and standard deviations pooled from left- and right-hand data

†† means and standard deviations pooled from male and female data

* $p<0.05$,

** $p<0.01$,

*** $p<0.001$

Table 6

Fingertip pattern asymmetry - three-pattern classification

Study	Sample composition	*n* SZ	SZ mean (SD)	*n* Control	Control mean (SD)	Effect size (g)	95% LL	95% UL	Z
Avila et al., 2003	Male + Female	86	1.27 (0.96)	46	0.95 (0.94)	0.33	−0.03	0.69	1.82
Markow and Wandler, 1986	Male + Female	81	1.54 (1.14)	69	1.13 (0.85)	0.40*	0.07	0.72	2.41
Reilly et al., 2001	Male + Female	22	1.63 (0.95)	37	1.14 (1.11)	0.46	−0.08	0.99	1.68
Cantor-Graae et al., 1998	Females	16	1.56 (1.1)	16	0.94 (0.9)	0.60	−0.11	1.31	1.66
Cantor-Graae et al., 1998	Male	44	1.09 (0.9)	59	1.46 (1.1)	−0.36	−0.75	0.03	−1.79

Table 7

Fluctuating Asymmetry finger ridge count (FAFRC)

Study	Sample composition	*n* SZ	SZ mean (SD)	*n* Control	Control mean (SD)	Effect size (g)	95% LL	95% UL	*Z*
Reilly et al., 2001	Male + Female	19	0.09 (0.08)	37	0.06 (0.06)	0.44	−0.12	1.00	1.54
Saha et al., 2003	Female	63	0.17 (0.03)	106	0.17 (0.03)	0	−0.31	0.31	0
Saha et al., 2003	Male	118	0.15 (0.01)	122	0.13 (0.02)	1.25***	0.98	1.53	8.870
Yousefi-Nooraie and Mortaz-Hedjri, 2008	Male	33	0.08 (0.02)	33	0.09 (0.02)	−0.49*	−0.98	0	−1.98

* $p<0.05$,

** $p<0.01$,

*** $p<0.001$

Table 8

Fluctuating Asymmetry A-B Ridge Count (FABRC)

Study	Sample composition	*n* SZ	SZ mean (SD)	*n* Control	Control mean (SD)	Effect size (g)	95% LL	95% UL	*Z*
Reilly et al., 2001	Male + Female	27	0.05 (0.04)	37	0.03 (0.03)	0.57*	0.07	1.08	2.21
Saha et al., 2003	Female	63	0.07 (0.01)	106	0.06 (0.01)	1.00***	0.67	1.32	5.92
Saha et al., 2003	Male	118	0.07 (0.01)	122	0.08 (0.01)	−1.00***	−1.26	−0.73	−7.28
Yousefi-Nooraie and Mortaz-Hedjri, 2008	Male	33	0.05 (0.004)	33	0.04 (0.004)	2.47***	1.83	3.11	7.46

*$p<0.05$,

**$p<0.01$,

***$p<0.001$