# FLUCTUATING ASYMMETRY AND SCHIZOTYPY:

# A TIMELINE OF DEVELOPMENTAL DISRUPTIONS

by

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### Abstract

The present study was designed to test if different indexes of *fluctuating asymmetry* (FA), each comprised of traits specific to certain periods of development, could be used to test in which of these spans individuals are vulnerable to the genetic and environmental stress factors that have been associated with the onset of the symptoms of schizotypy. Three FA indexes, composed of skeletal, facial, and dermatoglyphic features, were created. The individual traits that comprise these indexes were measured either from digital photographs taken of participants, or were directly measured via digital calipers. The participants' level of schizotypal symptoms was assessed via the short form version of the Wisconsin Schizotypy scales. Two way (Sides x Individuals) ANOVA tests found that FA was significantly greater than measurement error for all traits examined. However, the presence of significant *directional asymmetry* (DA) led to the exclusion of several traits from the total indexes. Correlational analysis found none of the resulting indexes to be significantly associated with scores on the Wisconsin Schizotypy scales. A small sample size, exacerbated by missing data for specific traits, is considered the most likely cause of this lack of association.

Keywords: Fluctuating Asymmetry, Developmental Instability, Schizotypy.

# Dedication

I dedicate this thesis to my wife Gracie, who has been very supportive of me throughout this process.

# Acknowledgments

I wish to express gratitude to my adviser, Dr. Hall, for his patience, insight, and guidance. I would also like to thank my committee members, Dr. Christensen and Dr. Steele for their support and suggestions. I also appreciated greatly the assistance of Grace Flood in the data collection stage of this study.

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### **Chapter 1: Purpose of Study**

*Fluctuating asymmetry* (FA) refers to the degree of deviation from perfect bilateral symmetry in features that are symmetrical at the population level (Van Valen, 1962). It is a widely used index of developmental disruptions. A number of studies have found elevated FA to be predictive of neurodevelopmental disorders, with schizophrenia-spectrum disorders being the most thoroughly documented. This linkage has been found when FA is indexed from dermatoglyphic features (skin ridges that are present on the hands and feet; Rosa et al., 2000; for a meta-review see Golembo-Smith et al., 2012) or skeletal features (Thomas, Gangestad, & Euler, 2008). While much of the existing research focuses on schizophrenia, the link between high FA and schizotypy has also been documented (Rosa et al., 2000; Saha et al., 2003; Thomas, Gangestad, & Euler 2008). Schizotypy refers to a set of symptoms that, while less severe, are similar to and predictive of the eventual manifestation of schizophrenia (Meehl, 1990). The present study is on the association between schizotypy and FA.

Dermatoglyphic patterns are formed by the end of the second trimester, and remain as they are after this point (Babler, 1991). The assumption in the existing research is that only developmental disruptions that occur before birth increase the likelihood of manifesting schizotypy. However, it has yet to be empirically documented that the window in which developmental disruptions lead to schizotypy is so narrow. In contrast to dermatological features, skeletal and facial asymmetry have been shown to increase postnatally until the end of puberty (Wilson & Manning, 1996). The primary purpose of the present study was to determine if skeletal FA has unique predictive value in regards to schizotypy after dermatoglyphic FA has been statistically accounted for. If skeletal FA does have unique predictive value, this would

suggest that postnatal development disruptions increase the likelihood of manifesting schizotypal symptoms.

The second goal of the present study was to establish if facial FA is more strongly associated with schizotypy than dermatoglyphic or skeletal FA. The morphogenesis of the face and brain suggests this is likely. Schizophrenia has been linked to atypical cerebral symmetry and lateralization, and in early fetal life cerebral and craniofacial development are tightly linked (Gruzelier, 1994, 1996; Heim, Kissler, Elbert, & Rockstroh, 2004; Sommer et al. 2001). In the present study it was expected that facial FA would be the best predictor of schizotypy.

#### **Chapter 2: Overview of Past Research**

## Schizophrenia

Schizophrenia is not an obscure psychiatric condition. Kraepelin (as cited in Yeo, Gangestad, Edgar, & Thomas, 1999) characterized it over a century ago as a distinct disorder and it affects roughly one percent of the world population (Gottesman, 1991). Schizophrenia is a disorder that imposes enormous costs on those who suffer from it, as well as on society as a whole; the CDC estimates that one out of ten people suffering from this condition will take his or her own life, and that the total yearly expense of schizophrenia—which includes the direct expense of medical care plus the loss of productivity caused by the disorder—is approximately \$6.85 billion in North America alone. It is therefore unsurprising that schizophrenia has long been a consistent subject of study; yet, the etiology of schizophrenia remains poorly understood. Some researchers question whether it is even a single disorder (Salem & Kring, 1998).

Researchers have long known that genetic factors relate to the manifestation of schizophrenia. Gottesman's review of the existing research suggested that children of schizophrenics have a tenfold increased risk of developing the disorder compared to the population average (as cited in Gajman, Sanders, & Duan, 2010). Twin studies have found the concordance rates for monozygotic twins to be 40-50% for schizophrenia, with heritability (in this context meaning the proportion of variance explained by genetic factors for a particular population) estimated to be 80% (Cardno & Gottesman, 2000). Further evidence that schizophrenia is the result of genetic factors comes from studies that looked at adopted children. These studies have found that children of schizophrenics had the same elevated likelihood of developing the disorder whether raised by adoptive or biological parents (Higgins et al., 1997). However, the fact that concordance rates for monozygotic twins are well below 100% implies

that environmental risk factors must play a part in the development of schizophrenia. Many such risk factors have been found to be associated with developing the disorder, including obstetric complications, malnutrition, prenatal infection and advanced paternal age (Byrne et al., 2003; Malaspina et al., 2001; McGrath, Saha, Chant, & Welham, 2008).

# Schizotypy

The term schizotypy is generally used to denote a set of correlated personality traits which are similar to, but less severe than, the symptoms of schizophrenia. These include reduced pleasure from social interaction, strange sensory experiences, and sometimes unusual or paranoid beliefs (Meehl, 1990). Just as in schizophrenia, researchers categorize these symptoms as either deficits in normal functioning (negative symptoms), or as unusual beliefs and experiences not shared by normally functioning individuals (positive symptoms; Claridge et al., 1996).

Because both genetic and environmental factors appear to contribute to the manifestation of schizophrenia, most researchers have adopted a diathesis-stress model of the condition (Chaptman & Chaptman, 1994). In this model, individuals vary in their levels of underlying biological psychosis-proneness, with high risk people requiring less environmental stress to manifest the condition than those with low psychosis-proneness (Gottesman 1991; Meehl, 1990). A number of studies suggest that schizotypy is the phenotypic expression of this underlying psychosis-proneness. It has been well documented that schizotypy and schizophrenia are underpinned by either the same, or similar, genetic factors (Cannon, van Erp, & Glahn 2001; Jang, Woodward, Lang, Honer, & Livesley 2005; Kendler, Gruenberg, & Strauss, 1981; Squires-Wheeler, Skodol, Bassett, & Erlenmeyer-Kimling, 1989 as cited in Cannon et al., 2011). Longitudinal studies have confirmed that an individual's degree of schizotypal symptoms is predictive of the eventual manifestation of schizophrenia (Cannon et al., 2001; Chapman et al

1994). Additionally, a large body of research suggests that individuals with high scores on measures of schizotypy do in fact exhibit psychological deficits akin to those exhibited by schizophrenics (for review see Fonseca-Pedrero et al., 2008).

Schizotypy Scales. Because of its relationship with schizophrenia, schizotypy is an important focus of research. By studying psychosis-prone individuals, researchers have the opportunity to see what environmental factors heighten or reduce the likelihood that schizophrenia will be manifested in adulthood. Additionally, developmental and genetic factors that underlie schizophrenia can be probed by investigating schizotypy.

Because psychosis proneness is believed to be normally distributed in the population, one of the great advantages of studying schizotypy, compared to schizophrenia, is that researchers can get access to much larger samples composed of individuals who are more likely to be willing to participate in research (Rosa et al., 2000; Verdoux & van Os, 2002) However, the earliest method of identifying schizotypal individuals— looking at the immediate family of schizophrenics—was not a convenient way to get these large samples (Erlenmeyer-Kimling & Cornblatt, 1987; Fish, 1987; Mednick, Parnas, & Schulsinger, 1987). Many researchers have therefore developed measures to identify schizotypal individuals by other means. While some have used biological measures, such as monoamine oxidase (MAO) activity (Holzman et al, 1988) or smooth-pursuit eye tracking (Siever, 1985), the majority have sought to develop paper and pencil measures, including Golden and Meehl's (1979) Schizoidia Scale, Eysenck and Eysenck's (1975) Psychoticism Scale and Claridge and Broks's (1984) Schizotypal Personality Scale. The Wisconsin Schizotypy Scales (WSS) are among the more popular, reliable and well validated measures (Chapman, Chapman, & Raulin, 1976; Chapman, Chapman, & Raulin, 1978; Eckblad & Chapman, 1983). Longitudinal studies have confirmed that individuals with elevated

scores on the WSS are more likely to eventually manifest schizophrenia. One study found that 5% of high scorers on two of the WSS subscales (Perceptual Aberration and Magical Ideation) developed a psychosis within ten years (Chapman et al., 1994). Kwapil (1998) found that 24% of high scorers on another subscale (revised Social Anhedonia) developed some form of schizophrenia-spectrum disorder before the age of thirty. One review of the state of modern schizotypy assessment found the WSS to have the best psychometrics, when compared to other popular measures of schizoptypy (Fonseca-Pedrero et al., 2008). Additionally, individuals' performance on the WSS has also been found to be heritable, supporting the contention that the WSS is tapping into the underlying biological susceptibility to schizophrenia (Linney et al., 2003).

As successful as the WSS has been found to be, some researchers consider it to be inconveniently long. Additionally, new measurement models such as Item Response theory (IRT) suggest that each subscale has a significant number of items with low discrimination values, implying that these items are redundant and inefficient (Winterstein et al., in press). Winterstein et al. (2011) created short form versions of each scale, using only items with high difficulty and high discrimination (as defined by IRT). A study by Gross, Silvia, Barentes-Vidal, and Kwapil (2012) found the short form of the WSSs to be strongly correlated with the long forms, and to have comparable internal consistency (Table 1). The Wisconsin Schizotypy Short Scales were also found to be comparable to the original, in both statistical significance and effect size, in their association with the results of interview measures of psychotic symptoms. This suggests that the short form, while not as extensively validated as the original, is a comparably accurate measure of schizotypy (Gross, Silvia, Barrantes-Vidal, & Kwapil, 2011). This study utilized the short form version of the WSS.

## **Developmental Instability**

Existing research strongly suggests that schizotypy represents an underlying liability to manifest schizophrenia, and is influenced by both genetic and environmental factors. However, the process through which these factors interact to increase individuals' propensities to develop psychosis has yet to be well understood. Yeo, Gangestad and Thomas (1999, 2007) have proposed a neurodevelopmental model to answer this question, based on the concept of developmental instability (DI). DI is generally defined as the degree to which an organism's developmental program fails to produce its target phenotype as defined by its species in a particular environment (Møller & Swaddle, 1997). More specifically, DI is the product of two opposed factors. The first factor is developmental noise. This term refers to both environmental stressors (including, but not limited to, pathogens, free radicals, toxins, and thermal stress), or genetic stressors (including a high mutational load, or poorly co-adapted gene lines as in the case of hybrids; Graham, Raz, Hel-Or, & Nevo, 2010; Palmer & Strobeck, 2001). The second factor is *developmental stability*, which refers to the ability of an organism's developmental program to counteract and correct for the effects of developmental noise (Palmer & Strobeck, 2001). When pushed off its original developmental trajectory by some perturbation, an organism with high developmental stability is able to course correct, eventually producing the intended phenotype. While the underlying mechanisms responsible for developmental stability are unknown, it appears genetic factors, including high heterozygosity and well co-adapted gene lines, are positively related (Raz, Hel-Or, & Nevo, 2010; for a review of the literature on the relationship between gene-coadaptaion and DI see Alibert and Auffray, 2003). It should be noted, though, that one meta-analysis found the relationship between DI and heterozygosity to be relatively weak (Vollestad, Hindar, & Moller, 1999).

According to Yeo, Gangestad and Thomas's model (1999, 2007), an individual's genotype influences his or her likelihood of manifesting a neurodevelopmental disorder in two distinctly different ways. The first is by increasing DI, which could be the result of mutations either directly increasing developmental noise or lowering the efficacy of the developmental system's ability to buffer developmental noise (genetic or environmental). This elevated DI increases the liability of developing a broad range of neurodevelopmental disorders. According to Yeo, Gangestad and Thomas (1999, 2007), this explains the elevated level of comorbidity among neurodevelopmental disorders such as dyslexia and schizophrenia, as well as the fact that many neurodevelopmental disorders often produce similar physical abnormalities. The second way an individual's genotype influences the development of neurodevelopmental disorders is through specific genetic factors that are unique to each disorder.

The model proposed by Yeo, Gangestad and Thomas (1999, 2007) suggests that neurodevelopmental disorders can result from either environmental or genetic factors, via their effects on DI. A third factor that has not been explicitly addressed yet is the timing of exposure to developmental noise. This is as critical a factor as any, but before it can be addressed we must first look at how DI is measured.

#### **Fluctuating Asymmetry**

Because it cannot be directly observed, researchers have to use a number of different methods to index DI, the most common being *fluctuating asymmetry* (FA) (Palmer, 1994). FA is the asymmetry of bilateral traits that are symmetric at the population level (Van Valen, 1962).

FA is one of three kinds of "subtle asymmetries," which are each defined by a distinct pattern of right-minus-left (R-L) differences at the population level (Palmer 1994). FA is characterized by a normal distribution of R-L with a mean of zero. *Directional asymmetry* (DA)

exists where one particular side of a bilateral feature tends to be larger than the other within the population. If a structure is directionally asymmetric, the distribution of R-L differences will be normally distributed around a mean either above or below zero (Palmer & Strobeck, 1986). *Anti-symmetry* (AS) is characterized by a mean R-L difference of zero, where the distribution is either bimodal, or platykurtic (Palmer, 1994).

Of these three, FA offers the most unproblematic index of underlying DI. This is because DA and AS can be adaptive and part of the organism's developmental plan, and as such are the result of genetic and environmental factors unrelated to DI (Tomkins & Kotiaho, 2002). For this reason Palmer and Strobeck (2003) advise excluding traits that exhibit DA or AS. Others have seen traits exhibiting DA as less problematic, and suggested that DA can be factored out easily (Thomas, Gangestad & Euler, 2008). Palmer and Strobeck (1986; Palmer, 1994) have themselves suggested means of accomplishing this.

On the conceptual level, FA is an attractive proxy for DI because bilateral traits have an *a priori* ideal known to the researcher: perfect bilateral symmetry (Graham, Raz, Hel-Or, & Nevo, 2010). Additionally, there is a large body of research to support FA being a useful proxy for DI. Remembering that DI is determined by two factors, *developmental stability* and *developmental noise*, it should be expected that environmental stressors should be positively associated with FA, and high genetic quality (such as low levels of inbreeding, co-adapted gene-lines, and high heterozygosis) should be negatively associated with FA. Additionally, if FA is an index of overall DI, we should expect it to have a negative association with overall fitness. Research exists which has documented all three of these expected relationships.

FA has been found to be positively associated with many forms of environmental stress, including high population density (Zahkarov, Demin, & Baranov, 1997), nutritional stress

(Pravosudov & Kitaysky, 2006), and heat stress (Petavy et al., 2006). The relationship between parasitism and increased FA (in the host) has been extensively documented (for a review, see Moller, 1996; 2006; for a review focused on humans see Thornhill &Moller, 1997). As predicted, FA has been found to have a negative relationship with heterozygosity (Borrell et al. 2004), a positive association with hybridization (poorly co-adapted gene-lines; Andersen et al. 2006), and a positive association with inbreeding (Mazzi, Largiader & Bakker, 2002), though as previously noted one meta-analysis found the relationship between DI and heterozygosity to be relatively weak (Vollestad, Hindar, & Moller, 1999). Also as predicted, fitness is found to have a modest negative relationship with FA (for a review, see Moller, 1996).

FA has been found to be positively associated with several human neurodevelopmental pathologies, but its relationship with schizophrenia-spectrum is the best-documented (Yeo, Gangestad & Thomas, 1999). Some of these studies have focused on the asymmetry of dermatoglyphic features (skin ridges that are present on the hands and feet; Rosa et al., 2000; for a meta-review see Golembo-Smith et al., 2012) and others have calculated asymmetry based on skeletal features (Thomas, Gangestad, & Euler, 2008). Both types of studies have found FA to be predictive of schizotypal symptoms, but what they reveal about the timing of developmental disruptions differs. Dematoglyphic patterns are formed by the end of the second trimester, and remain as they are after this point (Babler, 1991). They can therefore serve as a measure of the degree of perturbation the fetus was experiencing between the 11<sup>th</sup> week (when ridges begin to form) and the 27<sup>th</sup> Week. Skeletal and facial asymmetry, in contrast, have been shown to increase post-natally through puberty (Wilson & Manning, 1996). As such, the significant positive associations found between both skeletal and dermatoglyphic asymmetry and schizotypy can be interpreted in multiple ways.

The possibility that seems to be implicitly endorsed by most researchers is that developmental disruptions only increase the likelihood of schizotypy during the first two trimesters. In this interpretation of the evidence, only the skeletal FA that was created during this period should be associated with schizotypy, and any subsequent increase in skeletal FA would not be predictive of schizotypy.

This would suggest that once dermatoglyphic asymmetry was statistically accounted for, skeletal asymmetry would not have any unique predictive value in regards to schizotypy. Another possibility is that developmental disruptions may increase the likelihood of schizotypy during a wider developmental window, perhaps all the way through puberty. If this were the case, skeletal asymmetry would be expected to have unique predictive value after the dermatoglyphic asymmetry was accounted for. The primary purpose of this study is to lend support to one of these interpretations.

The second goal of this study is to look at the potential relationship between facial FA and schizotypy. While facial FA has been investigated in relation with a variety of other traits, such as attractiveness and aggression, no prior study has linked facial FA to schizotypy (Grammer & Thornhill, 1994; Sanchez-Pages & Turiegano, 2010). This is surprising, considering that the link between schizophrenia spectrum disorders and other indexes of FA have been extensively studied (Rosa et al., 2000.;Saha et al., 2003; Thomas, Gangestad, & Euler 2008; for a meta-analysis of dermatoglyphic studies see Golembo-Smith et al., 2012). There are several reasons that facial FA, as indexed via the landmark method (in which traits are measured via the setting of markers on photographs of the feature), might be an especially good predictor of schizotypy. One reason has to do with the morphogenesis of the face itself. Early in fetal life, cerebral and craniofacial development are tightly linked, as evidenced by the fact that

neurodevelopmental disorders (such as Down's syndrome and velo-cardio-facial syndrome) are often characterized by dysmorphic facial features (Waddington et al., 1999). Given this linkage, and the fact that schizophrenia may be the result of atypical cerebral asymmetry and lateralization, facial asymmetry may be especially revealing (Gruzelier, 1994; Gruzelier et al., 1996; Sommer et al. 2001; Heim, Kissler, Elbert, & Rockstroh, 2004). This contention is supported by an earlier study that found differences in facial symmetry between schizophrenics and controls, though the researchers were investigating directional, rather than fluctuating asymmetry (Hennessy et al., 2004).

### Chapter 3: Method

# Participants

Data from 106 participants was collected for this study. Participants were drawn through the SONA system, from psychology courses at Radford University, a medium-sized university in the southeast (See Appendix A for SONA study description). All participants were males between the ages of 18 and 23 (M=19.45, SD = 1.37). Participants were prescreened for male gender and the inclusive age range of 18 to 24; this screening criterion was used because this is near the age range at which positive schizotypal symptoms tend to first emerge (negative symptoms tend to emerge somewhat earlier; Galdos et al., 1993; Messia, Chen, & Eaton, 2007; Salem & Kring, 1998). The gender restriction is in place because earlier studies have shown the relationship between FA and schizotypy to be dependent on gender (possibly because of differences in DA), necessitating that all analyses be conducted independently for males and females (Hennessy et al., 2004, Thornhill & Gangestad, 2006). Because the collection of data was labor intensive for this study, it was unlikely that enough participants could be included to create adequate sample sizes for both genders. This experiment used male, rather than female, participants because recent meta-analysis has suggested that males have a 30-40% greater risk of developing schizophrenia at some point in their lives (Mcgrath et al, 2004). The majority of participants (69.8%) were of European American descent, 18.9 percent identified as African American, 4.7% identified as Asian/Pacific Islander, 3.8% identified as Hispanic, and 2.8% identified as other. All procedures were approved by the IRB of Radford University.

### Measures

**Facial FA**. This study collected measurements of facial FA using the protocol developed by Sanchez-Pages and Turiegano (2010). Digital photographs of the participants were taken with

a Nikon D 160 camera. These photographs have a 3872x2592 pixel resolution. Photographs were taken from a constant distance of three meters. Participants were photographed looking toward the camera with neutral expressions. All participants were asked to remove any facial adornment, and were instructed to wear a disposable shower cap if their features were obscured by their hair. Three photographs were taken of each individual, the best of which was used in the final analysis. The tpsDIG2 program (by F.J. Rohlf, see http://life.bio.sunysb.edu/morph/) was used to set 12 landmarks (LM) on each image. These LMs were used because they can be unambiguously located in photographs, and have the same relative position on each participant's face. These LMs are the 12 used by Grammer and Thornhill (1994). In Sanchez-Pages and Turiegano's (2010) study each LM was set twice for each participant, once by each author. This was done in order to establish the degree of measurement error. LMs were set twice in this study for the same reason. This study deviates from the existing protocol in that only one author set all LMs. In order to prevent this from artificially reducing the assessed measurement error, the tpsUtil program (by F.J. Rohlf, see http://life.bio.sunysb.edu/morph/) was used to randomize the order that the participants' photographs will be presented in the tpsDIGS program, so both copies of one participant's picture will not have landmarks set during the same session. After landmarks were set twice for all participants, the MorphoJ program (by C. P. Klingenberg, see http://www.flywings.org.uk/MorphoJ page.htm) was used to perform a Procrustean fit of all LM data. This operation converts the LM data into a scale free form. This was an important step, because, as Palmer and Strobeck (2003) have pointed out, without conversion to a scale free form, larger individuals will appear to have greater FA than smaller individuals merely as a function of their size. The MorphoJ program was used to perform a Procrustean ANOVA. Conceptually, DA is the amount of asymmetry that can by accounted for by the difference in the

average size of the two halves of the face. In this calculation DA is the main effect of the variable Side. FA is the interaction of variables Side and Individual, or how much the individual deviates from the normal pattern of asymmetry (Klingenberg & McIntyre, 1998). MorphoJ gave each participant an FA score, based on the proportion of the Side x Individual interaction variance each individual was responsible for.

**Dermatoglyphic FA.** Earlier studies looking at dermatoglyphic FA have used either inkless or traditional ink printing procedures to gather data from participants. This approach has several disadvantages. Participants tend to dislike the mess involved with the process, as well as the length of time it can take for the researcher to take a good print. Dermatoglyphic printing is also fairly difficult for the researcher, usually requiring an assistant (Gupta & Gupta, 2013). It can also be fairly unreliable, especially for those with little experience. For example, Landers (2007) was forced to entirely exclude a-b ridge count FA from his final analysis, after around 1/3 of all prints were found to be unreadable. This study has forgone dermatoglyphic printing altogether. Instead, 3872x2592 pixel digital photographs of the participants' hands were taken with a Nikon D 160 camera. At this resolution, dermatoglyphic features can be evaluated from an enlarged image. Participants had each finger pad and the A-B triradii area of the palm marked with green highlighter in order to increase the contrast between dermal ridges and the area between them, making them easier to count. Three photos were taken for each hand of the upper palm, three of the thumb pad and three of the index and little fingers held together. Multiple photographs were taken to insure that at least one is usable.

Three measures of FA were taken from these photographs, following the procedure used by Reilly et al. (2001). The first was fingertip pattern asymmetry. The dermal ridges pattern of each fingertip (whorl, loop, or arch) was compared to the pattern of the same finger on the

opposite hand. The number of mismatches is the participant's FA score, ranging from 0-5 (Reilly et al., 2001). The second measure of FA was based on the number of ridges touching a straight line between the A and B triradii of the palm, triradii being the point where three distinct lines of dermal ridges converge, and A and B referring to the triradii under the index and middle finger respectively. The third measure was based on the total finger ridge count (TFRC); this was computed by drawing a line between the triradii and the core, and counting the total number of ridges this line intersects. For these second two measures, FA was calculated by subtracting the right hand ridge count from the left hand ridge count, dividing this number by the total ridge count, and then taking the absolute value of the outcome.

Measurements for each trait were taken twice (nonconsecutively), in order to allow for an estimation of ME to be computed. The sum of the standardized scores for each measure was used to make a composite index of dermatoglyphic FA.

**Skeletal FA.** This study assessed skeletal FA by measuring the width of participants' elbows, wrists, ankles, ears and feet with Neiko 12" Extra Large Digital Calipers (model 01409A), following the protocol developed by Gangestad (Personal communication from Gangestad to Landers, in Landers, 2007). This measurement was recorded on the Skeletal FA sheet (see Appendix B). Each structure was measured twice, non-consecutively, to allow for an estimation of measurement error. Index, middle, ring, and pinky finger length were recorded, but not via digital calipers (as in the protocol developed by Gangestad). Instead, the participant placed both hands side by side, palms down on a white piece of paper. A ruler was placed at the edge of the paper, and 3872x2592 pixel digital photographs of the participants' hands were taken from directly above with a Nikon D 160 camera. This modification of the existing protocol was done in order to reduce the amount of time the participant must be inconvenienced, as well as to

increase accuracy by allowing the experimenter to use the tpsDig2 program (by F.J. Rohlf, see http://life.bio.sunysb.edu/morph/) to measure from enlarged photographs. The sum of the standardized scores for each structure was used to make a composite index of Skeletal FA.

**Wisconsin Schizotypy Scales.** The following scales were presented to the participants in one intermixed form. As in previous studies of the link between schizotypy and FA, one index score of schizotypy was created by combining participants' scores on each individual subscale (Thomas, Gangestad & Euler, 2008).

The Perceptual Aberration Scale: Short form. The original Perceptual Aberration Scale (PerAb) was developed to assess the perceptual abnormalities that characterize schizotypy (Chapman, Chapman, & Raulin, 1976). All items are dichotomously scored (true or false) questions. The authors of the PerAb established internal consistency using several different samples. The largest was of college students, and in this sample the genders were analyzed separately. For male college students (n=631) Cronbach's alpha was .89. The measure was given to smaller samples of schizophrenics (n=66), a normal adult control (n=100), and nonpsychotic clinical patients (n=20), producing alphas of .92, .89, and .94 respectively (Chapman, Chapman, & Raulin, 1976). The PerAb has positive correlations with several other scales that are thought to measure psychosis proneness such as Golden and Meehl's Schizoidia Scale, and Eysenck and Eysenck's Psychoticism Scale (Chapman, Chapman, & Miller, 1982). The PerAb appears to have good discriminant validity, displaying weak negative relationships with unrelated constructs, such as Extraversion and Agreeableness, which correlate at -.16 and -.18 respectively (n=430; Gross, Silvia, Barrantes-Videl, Kwapil, 2012). The Short Form version consists of 15 of the original questions that Winterstein et al. (2011) found to have high difficulty and high discrimination (as defined by IRT). Gross, Silvia, Barentes-Vidal, and Kwapil (2012)

found the short form of the WSS to be strongly correlated with the long form, and to have comparable internal consistency (Table 1). Scores for this measure are summed.

*Revised Social Anhedonia Scale: Short Form*. The original Revised Social Anhedonia Scale (SocAnh) is a measure of the diminished social pleasure and asociality that are considered negative symptoms of schizotypy. All items are dichotomously scored (true or false) questions. This measure has strong internal consistency (Cronbach's Alpha=.89; Chapman, Chapman, & Miller, 1982). This scale's validity is reinforced by the longitudinal studies that have found that social anhedonia has a potentiating role in the development of later clinical psychosis (Kwapil, et al., 1997). The short form of the SocAnh is composed of 15 questions, selected in the same manner as the PerAb short form (Winterstein et al., 2011). The SocAnh short form is similar to the PerAb short form in its internal consistency and its correlation with the scale from which it is derived (see Table 1; Gross, Silvia, Barentes-Vidal, & Kwapil, 2012). Scores for this measure are summed.

*Physical Anhedonia Scale: Short Form.* The original Physical Anhedonia Scale (PhyAnh) measures deficits in aesthetic and sensory pleasure. All items are dichotomously scored (true or false) questions. It has strong internal reliability (Cronbach's Alpha =.88; Gross, Silvia, Barrantes-Videl, Kwapil, 2011). Additionally, it has been found that schizophrenic patients have statistically significant higher scores on this measure than controls (Chapman, Chapman, & Raulin, 1976). The short form of the Physical Anhedonia Scale consists of 15 of the original questions which Winterstein et al. (2011) selected using the same criteria as in the two previous short form scales. This short form scale is also strongly correlated with the original it is derived from, and has high internal consistency (see Table 1). Scores for this measure are summed.

*Magical Ideation Scale: Short Form.* The Magical Ideation Scale (MagicId) is a measure of paranormal and delusion-like beliefs about causality that violate cultural norms. All items are dichotomously scored (true or false) questions. This measure has good internal consistency (Cronbach's Alpha =.82), and high scores on this measure have been found to predict later psychotic experiences (Chapman et al., 1994; Eckblad & Chapman, 1983). The short form of the MagicId Scale consists of 15 of the original questions which Winterstein et al. (2011) selected using the same criteria as in the previous short form scales. As with the previous scales, the MagicID short form strongly correlates with the original it is derived from, and has high internal consistency (Table 1). Scores for this measure are summed.

Table 1: Comparisons betw	Item	Chronbach's Alpha	Correlation between Short and
	Count	1	long forms
Original Magical Ideation	30	.84	.92
Short Magical Ideation	15	.76	.92
Original Perceptual Aberration	35	.88	.89
Short Perceptual Aberration	15	.84	
Original Social Anhedonia	40	.84	
Short Social Anhedonia	15	.79	.88
Original Physical	61	.84	
Anhedonia			.81
Short Physical Anhedonia	15	.73	

Table 1: Comparisons between short and long form WSS

Data from Gross, Silvia, Barrantes-Vidal, & Kwapil, (2011).

# Procedure

Each session was scheduled to be conducted with two participants. If only one participant showed up the session was conducted. At the beginning of each session, participants were given an informed consent sheet (Appendix C). The consent form explained that the participants are

free to discontinue the session at any time. The consent form also contained information about how to get more information about the study, and who to contact if the participants have any complaints. Participants that chose to continue with the study were given a participant number which they used on all subsequent forms instead of any identifying personal information (such as their name or student ID number). The participants then filled out a brief demographics questionnaire (Appendix D). After both participants were finished with the questionnaire, the experimenter left one participant in the room to complete the WSS short form, and took the other to have FA measurements taken in another room. Measurements were taken for the face, dermatoglyphic, and skeletal features respectively, following the procedure outlined in the methods section. After both the FA measurements and schizotypy scales were completed by both participants, they were thanked for their time and the session was complete.

### **Statistical Analysis Plan**

The following data analysis plan closely followed the recommendations outlined by Palmer and Strobeck (2003). The first step was creating scatterplots of the raw measurement data for each trait. The first of these plots was based on the difference between measurement 1 (M1) and measurement 2 (M2) for a specific trait. Visual inspections of these scatterplots were made in order to see if recording errors had been made at any stage (initial measurements, data entry, etc). This was a vital step, because errors here can substantially increase the estimated measurement error (ME), and underestimate the actual FA, for a trait.

If this inspection led to ME values that appeared to be outliers, one of two steps were taken. The first and preferable option was to rerecord the raw trait measurements. This was done for facial and dermatoglyphic traits, as the original photographs could be referenced. Unfortunately, this is not an option for the skeletal traits (excepting those derived from

photographs). Outlier tests were performed on the anomalous measurement values to decide which should be excluded. Palmer and Strobeck (2003) recommend using Grubb's test statistic to decide whether outliers should be excluded, and the present study followed this recommendation.

The next step was to create scatterplots of the difference between the left and right side for each trait, in order to find outliers that could inflate FA estimates. Individual participants may have extreme asymmetry for a trait that is unrelated to underlying DI (such as asymmetry caused by injury), which could artificially inflate estimates of FA. Potential outliers were addressed in the same way as potential ME outliers.

After outliers were addressed, the next step was to determine whether the FA is significantly greater than ME. This was done via a two-way ANOVA (Sides x Individuals), calculated separately for each trait. Traits in which FA was not significantly greater than ME were excluded from later analysis. This test simultaneously tested for DA, and traits displaying significant DA would also be excluded.

Three composite indexes of FA were made from the individual trait measurements, as described in the measures section; one index of facial FA, one index of skeletal FA, and one index of dermatoglyphic FA. Correlational analysis was done to establish which indexes were significantly associated with scores on the WSS. Hierarchical multiple regressions were conducted on the indexes that are significantly associated with schizotypy scores. This was done to see if skeletal FA is still a significant predictor of schizotypy, after dermatoglyphic FA has been accounted for. Facial FA was to be included to see if it is an equivalent, or superior, predictor of schizotypy, compared to skeletal and dermatoglyphic FA.

#### **Chapter 4: Results**

## Wisconsin Schizotypy Scales

All 106 participants have complete data for all subscales of the WSS, with average scores of 2.05 for the Perceptual Aberration Scale (SD=2.929), 4.60 for the Magical Ideation Scale (SD = 3.402), 2.52 for the Revised Social Anhedonia Scale (SD = 2.546), and 2.86 for the Physical Anhedonia Scale (SD = 2.424). For subscale correlations, refer to Table 2.

# **Missing Data**

All physical traits that were measured had some degree of missing data. The primary cause of this in regards to skeletal traits was previous injury. Additionally, several participants had sufficient fleshy tissue around skeletal features that accurate measurements could not be made without an unacceptable level of discomfort being caused. For dermatoglyphic traits the primary cause of missing data was abrasion and callusing of the hand caused by exercise-related physical exertion. Additionally, a number of participants had very fine dermal ridges of the fingertips. This proved to be an insurmountable obstacle with regards to the total finger ridge count, leading to this measure being dropped. Due to the nature of the FA calculations used to generate the index of facial FA, each participant must have the exact same number of LMs. This led to the use of a circumscribed number of LMs (8) being included in the final analysis, as a large minority of participants had significant facial hair. For the number of missing data points of each individual trait, please refer to Table 3.

		Perceptual Aberration Scale	Magical Ideation Scale	Revised Social Anhedonia Scale	Physical Anhedonia Scale
Perceptual Aberration	Pearson Correlation	1			
Scale	Sig. (2-tailed)				
Magical Ideation Scale	Pearson Correlation	.603**	1		
	Sig. (2-tailed)	.000			
Revised Social Anhedonia Scale	Pearson Correlation	.230*	.115	1	
	Sig. (2-tailed)	.017	.239		
	Pearson Correlation	062	082	.132	1
Physical Anhedonia Scale	Sig. (2-tailed)	.527	.404	.176	

# Table 2: Wisconsin Schizotypy Subscale Correlations

\*\*. Correlation is significant at the 0.01 level (2-tailed).

\*. Correlation is significant at the 0.05 level (2-tailed).

Feature	Data Unmeasured	M1-M2 Outliers	R-L Outliers
Left Ear (Width)	1	3	0
Left Ear (Length)	1	2	0
Left Elbow	7	1	1
Left Wrist	7	2	0
Left Ankle	7	0	0
Left Foot	2	2	0
Right Ear (Width)	1	1	0
Right Ear (Length)	3	0	0
Right Elbow	7	1	1
Right Wrist	7	0	0
Right Ankle	8	0	0
Right Foot	3	0	1
Right Index Finger	2	1	1
Right Middle Finger	2	1	0
Right Ring Finger	2	1	0
Right Little Finger	2	0	0
Left Index Finger	2	0	1
Left Middle Finger	2	1	0
Left Ring Finger	2	1	0
Left Little Finger	2	0	0

# Table 3: Missing or Excluded Trait Measurements

## **Outlier Analysis**

Visual inspection of scatterplots of M1-M2 differences for skeletal features, followed by the calculation of Grubb's Test Statistic for each potential outlier, resulted in the exclusion of data points for several traits (Table 3). Visual inspection of scatterplots of R-L differences for skeletal features, also followed by the calculation of Grubb's Test Statistic, resulted in the exclusion of one participant's elbow width data, and another participant's foot width measurements. No outliers were identified for the facial traits or either dermatoglyphic trait.

# Reliability

All skeletal and dermatoglyphic traits showed high M1-M2 reliability (Table 4), which compares favorably to other morphometric studies (Swaddle, Witter, & Cuthill, 1994). The use of a procrustean ANOVA procedure for the facial traits makes calculating a correlation coefficient of M1-M2 landmarks impractical; however, this omission is justified by the fact that this procedure is itself a robust test of both FA and DA (Palmer, 1986, 1994, 2003).

## **Tests for FA and DA**

A two-way (Sides x Individual) ANOVA (Palmer, 1994), calculated separately for each skeletal trait, found FA to be significantly greater than ME for all traits; however, several traits displayed significant DA (Table 5). Procrustean ANOVA found facial FA to be significantly greater than ME, F(630, 1273) = 6.05, p = <.0001. This analysis also suggested significant DA, F(6,630) = 4.69, p = .0001. ANOVA methods of testing DA can be misleading when applied to metrical traits (Palmer, 1994), so AB ridge count was tested for DA via the one sample t-test. The right minus left side difference was not found to be significantly different from zero, t(86) = .271, p = .787, suggesting an absence of DA. The effect size d of 0.058 suggests a very small effect for Side. Fingertip pattern asymmetry was not tested for DA, as the scoring metric for this

trait does not allow for standard tests of DA. Traits found to have significant DA were not included in the multi-trait indexes.

Table 4: M1-M2 Reliability of Features

Feature	Ν	Pearson Correlation	Significance (2-Tailed)
Left Ear (Width)	102	.920	<.001
Left Ear (Length)	103	.935	<.001
Left Elbow	97	.931	<.001
Left Wrist	97	.928	<.001
Left Ankle	99	.953	<.001
Left Foot	102	.953	<.001
Right Ear (Width)	105	.913	<.001
Right Ear (Length)	103	.946	<.001
Right Elbow	97	.950	<.001
Right Wrist	99	.938	<.001
Right Ankle	98	.956	<.001
Right Foot	102	.958	<.001
Right Index Finger	102	.977	<.001
Right Middle Finger	102	.985	<.001
Right Ring Finger	103	.978	<.001
Right Little Finger	104	.981	<.001
Left Index Finger	103	.977	<.001
Left Middle Finger	103	.985	<.001
Left Ring Finger	103	.980	<.001
Left Little Finger	104	.982	<.001
Left AB ridge count	88	.963	<.001
Right AB ridge count	90	.968	<.001
Finger Tip Pattern	106	.985	<.001

Feature	Source	df	MS	F	Р
Ear (Width)	Side	1	3.441	3.793	.054
	Error	101	.907		
	Side x	101	.907	1.947	<.001
	Individual Error	207	.466		
Ear (Length)	Side	1	50.211	16.094	<.001
	Error	101	3.120		
	Side x	101	3.12	2.90	<.001
	Individual Error	205	1.08		
Elbow	Side	1	.162	.087	.769
	Error	97.378	1.872		
	Side x	97	1.878	2.579	<.001
	Individual Error	194	.728		
Wrist	Side	1	4.710	3.335	.071
	Error	96	1.412		
	Side x	96	1.41	2.19	<.001
	Individual Error	196	.654		
Ankle	Side	1	8.484	6.735	.011
	Error	98	1.260		
	Side x	98	1.261	1.828	<.001
	Individual Error	197	.690		
Foot	Side	1	6.735	19.634	<.001
	Error	101	3.146	17100	
	Side x	101	3.146	2.534	<.001
	Individual Error	204	1.242	2.554	<.001
Index Finger	Side	1	.002	.071	.791
muck i mgei	Error	103	.024	.071	.771
	Side x	103	.019	3.305	<.001
	Individual Error	205	.006	5.505	<.001
Middle Finger	Side	1	.000	.006	.938
Midule Filigei		103	.000	.000	.938
	Error	103		ΛΕΛΕ	<.001
	Side x Individual Error		.023 .005	4.646	<.001
Din - Fin		205		2 459	120
Ring Finger	Side	1 103	.068	2.458	.120
	Error		.027	2 406	< 001
	Side x	101	.028	3.406	<.001
L :441 a Eine	Individual Error	106	.008	( 0.01	010
Little Finger	Side	1	.138	6.821	.010
	Error	103	.020	4 202	- 001
	Side x	103	.020	4.303	<.001
	Individual Error	208	.005		

Table 5: Side x Individual ANOVA Test for Fluctuating Asymmetry/Directional Asymmetry

## **Correlational Analysis**

FA indexes were compiled in the manner described in the methods section. Because of traits being excluded due to the presence of DA, the index of skeletal traits was comprised of the standardized FA scores of the ear (width), the elbow, the wrist, and the index, middle, and ring fingers. The index of dermatoglyphic traits was comprised of the standardized FA scores of the AB ridge count, and fingertip pattern asymmetry. Correlational analysis was performed not just between FA indexes and the participants' total WSS score, but also between the FA indexes and the individual subscales. This is done because subscales of the WSS were not well correlated with each other, meaning that in this study there is no empirical evidence that they are all measuring aspects of the same construct. This undermines the legitimacy of the combined WSS score (necessitating independent subscale correlations with FA indexes), though it will still be included in the analysis so as to follow the protocol of earlier work on this subject (Thomas, Gangstad & Euler, 2008).

The skeletal FA index was not significantly correlated with participants' combined Wisconsin Schizotypy Scales (WSS) scores, r(84) = .157, p = .077. However, the index of skeletal FA was significantly correlated with scores for the Perceptual Aberration Scale, r(84) =.254, p = .010. Skeletal FA was not significantly correlated with the Magical Ideation scale, the other index of positive schizotypy symptoms, r(84)=.118, p = .142. Additionally, skeletal FA was not significantly correlated with the Revised Social Anhedonia Scale, r(84) = .040, p = .360, or the Physical Anhedonia Scale, r(84) = -.053, p = .316. The dermatoglyphic FA index was not significantly correlated with scores on WSS, r(87) = .014, p = .449. The Perceptual Aberration Scale was uncorrelated with dermatoglyphic FA, r(87) = -.132, p = .111, as was the Magical Ideation Scale, r(87) = .058, p = .297. Dermatoglyphic FA was also not significantly correlated with the Revised Social Anhedonia Scale, r(87) = -.039, p = .359, or the Physical Anhedonia Scale, r(87) = .154, p = .077.

Because none of the three FA indexes were significantly correlated with participants' combined Wisconsin Schizotypy Scales scores, and no single subscale was significantly correlated with both indexes of FA, the hierarchical regression analysis proposed in the statistical analysis plan was not conducted.

### **Chapter 5: Discussion**

The present study was designed to test if different indexes of FA, linked to specific developmental stages, could help determine when the disruptive genetic and environmental factors that have been linked to adult schizotypy are most influential. However, the necessary precondition for performing the hierarchical regression analysis to test this hypothesis, that each FA index be significantly positively correlated with the Wisconsin Schizotypy scales, was not met.

There are a number of possible reasons that the predicted statistically significant association between the indexes FA and the Wisconsin Schizotypy scales was not found. The most obvious is the small sample size of 106 participants in this study. This situation was exacerbated by the fact that each index of FA contained traits with missing data. Because the FA indexes were solely comprised of participants with complete data, the eventual sample size for each was meaningfully reduced, with the skeletal index having only 84 participants, and the dermatoglyphic index only 87. To see if this resulted in an underpowered test of these correlations, a post hoc power analysis was conducted using the software package GPower (Faul, Erdfelder, Buchner, & Lang 2009). Based on the observed effect size (r = .157), total sample size (N=84), and acceptable significance level (p=.05), the estimated power (1- $\beta$  = .42) of the test for correlation between skeletal FA scores and combined WSS scores is well below the conventional desired power of .80 (Cohen, 1977). While multi-trait indexes of FA tend to be superior gauges of underlying developmental instability versus single trait measures, in this case this advantage was most likely more than offset by the reduction in sample size (Palmer, 2003). Post hoc power analysis for the correlation between dermatoglyphic FA scores and combined WSS score, with a significance level of .05 and a sample size of 87, suggested that this was a very underpowered

test ( $1-\beta = .06$ ). However, as this estimate was based on an extremely small effect size (r=.014), rather than interpreting this as an underpowered study needing a larger sample size, it is more likely the result of some flaw in data collection. This interpretation is supported by the fact that two of the four correlations between dermatoglyphic FA and the WSS subscales showed unexpected negative relationships. Alternatively, the hypothesized association between dermatoglyphic FA and schizotypy may simply not exist, though this is considered unlikely, given that this relationship has been found in a number of earlier studies. While post hoc power analysis of the correlations between the FA indexes and the individual WSS subscales varied, they similarly show these tests of association to be either relatively or extremely underpowered, with the exception of skeletal FA and the Perceptual Aberration Scale (Table 6).

		Perceptual Aberration Scale	Magical Ideation Scale	Revised Social Anhedonia Scale	Physical Anhedonia Scale
Skeletal FA	Effect Size (r)	.254	.118	.04	053*
	Acceptable Sig.	.05	.05	.05	
	Sample Size	84	84	84	
	Power $(1-\beta)$	.770	.029	.100	
Dermatoglyphic FA	Effect Size (r)	132*	.058	039*	.154
	Acceptable Sig.		.05		.05
	Sample Size		87		87
	Power $(1-\beta)$		.134		.41

 Table 6: Wisconsin Schizotypy Subscale Post Hoc Power Analyses

\*. Negative correlation makes power analysis unjustified.

While the facial FA index included all 106 participants, this could only be accomplished by steeply reducing the number of LMs to 8 from the originally intended 12, due to the large number of participants with facial hair obscuring other potential LMs.

Inaccurate measurement of the individual traits would result in the lack of association between the FA indexes and schizotypy scales; however, the high reliability of all measures suggests measurement error was small. Additionally, all traits were found to have FA that was significantly greater than measurement error, suggesting that data collection was successfully carried out. However, traits that also were determined to have significant directional asymmetry (DA) complicate this interpretation, as DA could be subtly introduced by the handedness of the researcher (Palmer, 1994; Landers, 2007). The DA could also be caused by a variety of other factors, including differential use and genetic factors (Palmer, 2003). Regardless, because these traits were excluded from the final indexes, any bias introduced here cannot explain the lack of correlation between the final indexes and the Wisconsin Schizotypy scales scores.

Another potential cause of the lack of association between the FA indexes and the Wisconsin Schizotypy Scales is the lack of motivation on the part of participants to respond to the scale's questions in a careful or accurate manner. The researcher consistently observed participants answering the sixty question questionnaire in less time than would seem possible to thoughtfully respond to all items. This interpretation is reinforced by the number of participants (5) who answered the demographics question of what year they had been born, by listing their home town. However, this interpretation is not supported by the pattern of subscale correlation that is found in this study (Table 2), which was similar at least in pattern of correlations to other, more large scale studies using the short form of the Wisconsin Schizotypy Scales (Gross, Silvia, Barrantes-Videl, Kwapil, 2012).

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# Limitations

One of this study's primary limitations was insufficient protocol to limit the recruitment of participants who had features that could not be measured using the existing methods. The largest oversight was failing to exclude participants with facial hair, as this severely hampered the researcher's ability to place the LMs necessary for calculating facial FA. Additionally, participants who engage in a significant amount of weightlifting, or other activities that cause a large degree of hand abrasion and callusing should be excluded from future studies.

## **Future Direction**

This study suggests that the protocol for measuring skeletal FA adapted from Gangestad (Personal communication from Gangestad to Landers, in Landers, 2007), is sufficiently accurate to be used in future studies. However, for researchers doing studies in which data collection is largely conducted by one individual, the collection of measurements using a digital caliper may prove prohibitively time consuming, limiting the potential sample size to an insufficiently large number of participants. This study's results suggest that skeletal measurements made from photographing the hands (index-little finger) of participants are as accurate and sensitive measures of FA, as those measured via caliper. Focusing on this method and excluding the use of the calipers in future studies could allow for the collection of larger samples in the same time span. Additionally, unlike the measures taken by caliper, the handedness of the researcher is unlikely to introduce a directional bias when measuring from digital photographs.

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### References

- Alibert, P., & Auffray, J.-C. (2002). Genomic coadaptation, outbreeding depression and developmental instability. In M. Polak (Ed.), *Developmental instability: Causes and consequences* (pp. 116–134). Oxford University Press.
- Andersen, D. H., Pertoldi, C., Loeschcke, V., & Scali, V. (2006). Developmental instability, hybridization and heterozygosity in stick insects of the genus Bacillus (Insecta; Phasmatodea) with different modes of reproduction. *Biological Journal of the Linnean Society*, 87(2), 249–259.
- Babler, W. J. (1991). Embryologic development of epidermal ridges and their configurations. Birth Defects Original Article Series, 27(2), 95–112. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/1786361
- Borrell, Y., Pineda, H., & McCarthy, I. (2004). Correlations between fitness and heterozygosity at allozyme and microsatellite loci in the Atlantic salmon, Salmo salar L. *Heredity*, 585– 593. doi:10.1038/sj.hdy.6800477
- Byrne, M., Agerbo, E., Ewald, H., Eaton, W. W., & Mortensen, P. B. (2003). Parental age and risk of schizophrenia: A case-control study. *Archives of General Psychiatry*, *60*(7), 673–8. doi:10.1001/archpsyc.60.7.673
- Cannon, T. D., Cadenhead, K., Cornblatt, B., Woods, S. W., Addington, J., Walker, E.,
  Heinssen, R. (2008). Prediction of psychosis in youth at high clinical risk: A multisite
  longitudinal study in North America. *Archives of General Psychiatry*, 65(1), 28–37.
  doi:10.1001/archgenpsychiatry.2007.3
- Cannon T.D., van Erp T.G., Bearden C.E., Loewy R, Thompson P, Toga AW, Huttunen M.O., Keshavan M.S., Seidman L.J. (2003). Early and late neurodevelopmental influences in

the prodrome to schizophrenia: Contributions of genes, environment, and their interactions. Retrieved October 14, 2014, from http://schizophreniabulletin.oxfordjournals.org/content/29/4/653.full.pdf

- Cardno, A. G., & Gottesman, I. I. (2000). Twin studies of schizophrenia: From bow-and-arrow concordances to star wars Mx and functional genomics. *American Journal of Medical Genetics*, 97(1), 12–7. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10813800
- Chapman, L. J., Chapman, J. P., Kwapil, T. R., Eckblad, M., & Zinser, M. C. (1994). Putatively psychosis-prone subjects 10 years later. *Journal of Abnormal Psychology*, *103*(2), 171–83. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8040487
- Chapman, L. J., Chapman, J. P., & Miller, E. N. (1982). Reliabilities and intercorrelations of eight measures of proneness to psychosis. *Journal of Consulting and Clinical Psychology*, 50(2), 187–95. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7069026
- Chapman, L. J., Chapman, J. P., & Raulin, M. L. (1976). Scales for physical and social anhedonia. *Journal of Abnormal Psychology*, 85(4), 374–82. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/956504
- Claridge, G., McCreery, C., Mason, O., Bentall, R., Boyle, G., Slade, P., & Popplewell, D. (1996). The factor structure of 'schizotypal' traits: A large replication study. *British Journal of Clinical Psychology*, *35*(1), 103–115. doi:10.1111/j.2044-8260.1996.tb01166.x
- Cohen, J. (1977). *Statistical power analysis for the behavioral sciences (rev.* Lawrence Erlbaum Associates, Inc.

- Erlenmeyer-Kimling, L., & Cornblatt, B. (1987). The New York High- Risk Project: A followup report. *Schizophrenia Bulletin*, 13, 451- 461
- Faul, F., Erdfelder, E., Buchner, A., & Lang, A.-G. (2009). Statistical power analyses using
   G\*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods*, 41, 1149-1160
- Fonseca-Pedrero, E., Paíno, M., Lemos-Giráldez, S., García-Cueto, E., Campillo-Álvarez, Á.,
  Villazón-García, Ú., & Muñiz, J. (2008). Schizotypy assessment: State of the art and
  future prospects. *International Journal of Clinical and Health Psychology*, 8(2), 577–
  593. Retrieved from http://www.redalyc.org/resumen.oa?id=33712001015
- Gejman, P., Sanders, A., & Duan, J. (2010). The role of genetics in the etiology of schizophrenia. *Psychiatric Clinics of North America*, *33*(1), 1–30. doi:10.1016/j.psc.2009.12.003.The
- Golembo-Smith, S., Walder, D. J., Daly, M. P., Mittal, V. a, Kline, E., Reeves, G., & Schiffman, J. (2012). The presentation of dermatoglyphic abnormalities in schizophrenia: a meta-analytic review. *Schizophrenia Research*, *142*(1-3), 1–11. doi:10.1016/j.schres.2012.10.002
- Graham, J. H., Raz, S., Hel-Or, H., & Nevo, E. (2010). Fluctuating Asymmetry: Methods, Theory, and Applications. *Symmetry*, *2*(2), 466–540. doi:10.3390/sym2020466
- Grammer, K., & Thornhill, R. (1994). Human (Homo sapiens) facial attractiveness and sexual selection: The role of symmetry and averageness. *Journal of Comparative Psychology* (Washington, D.C. : 1983), 108(3), 233–42. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7924253

- Gross, G. M., Silvia, P. J., Barrantes-Vidal, N., & Kwapil, T. R. (2012). Psychometric properties and validity of short forms of the Wisconsin Schizotypy Scales in two large samples. *Schizophrenia Research*, 134(2-3), 267–72. doi:10.1016/j.schres.2011.11.032
- Gruzelier, J., Burgess, A., Stygall, J., Irving, G., & Raine, A. (1995). Patterns of cognitive asymmetry and syndromes of schizotypal personality. *Psychiatry Research*, 56(1), 71–79. doi:10.1016/0165-1781(94)02564-Y
- Gruzelier, J. H. (1994). Syndromes of schizophrenia and schizotypy, hemispheric imbalance and sex differences: Implications for developmental psychopathology. *International Journal* of Psychophysiology, 18(3), 167–178. doi:10.1016/0167-8760(94)90002-7
- Gupta, R. K., & Gupta, A. K. (2013). New, Easy and Effective Method to Take Dermatoglyphic Prints. *National Journal of Medical Research*, 3(1), 45–47. Retrieved from http://www.scopemed.org/?mno=36119
- Heim, S., Kissler, J., Elbert, T., & Rockstroh, B. (2004). Cerebral lateralization in schizophrenia and dyslexia: Neuromagnetic responses to auditory stimuli. *Neuropsychologia*, 42(5), 692–697. doi:10.1016/j.neuropsychologia.2003.09.007
- Hennessy, R. J., Lane, A., Kinsella, A., Larkin, C., O'Callaghan, E., & Waddington, J. L. (2004).
  3D morphometrics of craniofacial dysmorphology reveals sex-specific asymmetries in schizophrenia. *Schizophrenia Research*, 67(2-3), 261–8.
  doi:10.1016/j.schres.2003.08.003

Higgins, J., Gore, R., Gutkind, D., Mednick, S. A., Parnas, J., Schulsinger, F., & Cannon, T. D.
(1997). Effects of child-rearing by schizophrenic mothers: A 25-year follow-up. *Acta Psychiatrica Scandinavica*, *96*(5), 402–404. doi:10.1111/j.1600-0447.1997.tb09936.x

- Kendler, K. S. (1984). An independent analysis of the Danish adoption study of schizophrenia. *Archives of General Psychiatry*, *41*(6), 555. doi:10.1001/archpsyc.1984.01790170029004
- Klingenberg, C., & McIntyre, G. (1998). Geometric morphometrics of developmental instability: Analyzing patterns of fluctuating asymmetry with Procrustes methods. *Evolution*. Retrieved from http://www.jstor.org/stable/2411306
- Klingenberg, C. P., Barluenga, M., & Meyer, A. (2002). Shape analysis of symmetric structures: Quantifying variation among individuals and asymmetry. *Evolution; International Journal of Organic Evolution*, 56(10), 1909–20. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12449478
- Kwapil, T. R. (1998). Social anhedonia as a predictor of the development of schizophreniaspectrum disorders. *Journal of abnormal psychology* (Vol. 107, pp. 558–565).
- Kwapil, T. R., Miller, M. B., Zinser, M. C., Chapman, J., & Chapman, L. J. (1997). Magical ideation and social anhedonia as predictors of psychosis proneness : A partial replication. *Journal of Abnormal Psychology*, *106*(3), 491–495.
- Landers, D. (2007). Developmental instability and psychological fitness: Can morphological asymmetry predict psychopathology? Retrieved from http://books.google.com/books?hl=en&lr=&id=qytcU3aXYVMC&oi=fnd&pg=PA4&dq =DEVELOPMENTAL+INSTABILITY+AND+PSYCHOLOGICAL+FITNESS:+CAN+ MORPHOLOGICAL+ASYMMETRY+PREDICT+PSYCHOPATHOLOGY%3F&ots=0 sPOm9o0kM&sig=CashRtdvdBdJyqIX3BrJqJM9Ck8
- Malaspina, D., Harlap, S., Fennig, S., Heiman, D., Nahon, D., Feldman, D., & Susser, E. S. (2001). Advancing paternal age and the risk of schizophrenia. *Archives of General*

Psychiatry, 58(4), 361-7. Retrieved from

http://www.ncbi.nlm.nih.gov/pubmed/11296097

- Mazzi, D., Largiadèr, C. R., & Bakker, T. C. M. (2002). Inbreeding and developmental stability in three-spined sticklebacks (Gasterosteus aculeatus L.). *Heredity*, *89*(4), 293–9. doi:10.1038/sj.hdy.6800138
- McGrath, J., Saha, S., Chant, D., & Welham, J. (2008). Schizophrenia: A concise overview of incidence, prevalence, and mortality. *Epidemiologic Reviews*, 30(1), 67–76. doi:10.1093/epirev/mxn001
- McGrath, J., Saha, S., Welham, J., El Saadi, O., MacCauley, C., & Chant, D. (2004). A systematic review of the incidence of schizophrenia: The distribution of rates and the influence of sex, urbanicity, migrant status and methodology. *BMC Medicine*, *2*, 13. doi:10.1186/1741-7015-2-13
- Meehl, P. E. (1990). Toward an Integrated Theory of Schizotaxia, Schizotypy, and Schizophrenia. *Journal of Personality Disorders*, *4*(1), 1–99. doi:10.1521/pedi.1990.4.1.1
- Messias, E., Chen, C., & Eaton, W. (2007). Epidemiology of schizophrenia: Review of findings and myths. *Psychiatric Clinics of North America*, 30(3), 323–338.
  doi:10.1016/j.psc.2007.04.007.Epidemiology
- Møller, A. P. (1997). The University of Chicago developmental stability and fitness : A review. *The American Naturalist*, *149*(5), 916–932.

Møller, A. P. (2006). A review of developmental instability, parasitism and disease. Infection, genetics and evolution. *Infection, Genetics and Evolution : Journal of Molecular Epidemiology and Evolutionary Genetics in Infectious Diseases*, 6(2), 133–40. doi:10.1016/j.meegid.2005.03.005

- Møller, A. P., & Swaddle, J. P. (1997). *Asymmetry, developmental stability, and evolution*. (p. 291). Oxford, England: Oxford University Press.
- Palmer, A. (1994). Fluctuating asymmetry analyses: A primer. *Developmental Instability: Its* Origins and Evolutionary ..., 93, 335–364. Retrieved from http://medcontent.metapress.com/index/A65RM03P4874243N.pdf

Palmer, A., & Strobeck, C. (1986). Fluctuating asymmetry: Measurement, analysis, patterns. Annual Review of Ecology and Systematics. Retrieved from http://www.jstor.org/stable/2097002

- Palmer, A., & Strobeck, C. (2003). CH 17. Fluctuating asymmetry analyses revisited. Developmental Instability: Causes and ..., 2001, 279–319. Retrieved from http://www.biology.ualberta.ca/palmer/pubs/03BookChapt/PalmerStrobeckChapt.pdf
- Petavy, G., David, J., & Debat, V. (2006). Phenotypic and genetic variability of sternopleural bristle number in Drosophila melanogaster under daily thermal stress: Developmental instability and anti-. *Evolutionary Ecology* ..., 149–167. Retrieved from http://evomorpho.org/Petavy et al evol ecol res 2006.pdf
- Pravosudov, V. V., & Kitaysky, A. S. (2006). Effects of nutritional restrictions during posthatching development on adrenocortical function in Western Scrub-Jays (Aphelocoma californica). *General and Comparative Endocrinology*, *145*(1), 25–31. doi:10.1016/j.ygcen.2005.06.011
- Reilly, J. L., Murphy, P. T., Byrne, M., Larkin, C., Gill, M., O'Callaghan, E., & Lane, A. (2001).
   Dermatoglyphic fluctuating asymmetry and atypical handedness in schizophrenia.
   *Schizophrenia Research*, 50(3), 159–168. doi:10.1016/S0920-9964(00)00044-X

Rosa, A., van Os, J., Fañanás, L., Barrantes, N., Caparrós, B., Gutiérrez, B., & Obiols, J. (2000).
Developmental instability and schizotypy. *Schizophrenia Research*, *43*(2-3), 125–34.
Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10858631

Saha, S., Loesch, D., Chant, D., Welham, J., El-Saadi, O., Fañanás, L., ... McGrath, J. (2003).
Directional and fluctuating asymmetry in finger and a-b ridge counts in psychosis: a case-control study. *BMC Psychiatry*, *3*, 3. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=154091&tool=pmcentrez&re ndertype=abstract

- Salem, J. E., & Kring, A. M. (1998). The role of gender differences in the reduction of etiologic heterogeneity in schizophrenia. *Clinical Psychology Review*, 18(7), 795–819. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9827322
- Sanchez-Pages, S., & Turiegano, E. (2010). Testosterone, facial symmetry and cooperation in the prisoners' dilemma. *Physiology & Behavior*, 99(3), 355–61. doi:10.1016/j.physbeh.2009.11.013
- Sommer, I. (2001). Handedness, language lateralisation and anatomical asymmetry in schizophrenia: Meta-analysis. *The British Journal of Psychiatry*, 178(4), 344–351. doi:10.1192/bjp.178.4.344
- Swaddle, J.P., Witter, M.S., & Cuthill, I.C.(1994). The analysis of fluctuating asymmetry. *Animal Behavior.* 48, 986-989.
- Thoma, R., Gangestad, S., & Euler, M. (2008). Developmental instability and markers of schizotypy in university students. *Evol. Psychol*, 6(4), 586–594. Retrieved from http://search.ebscohost.com/login.aspx?direct=true&profile=ehost&scope=site&authtype =crawler&jrnl=14747049&AN=36007220&h=hePch3KaNKKtvid3Fvb1d%2FttOplmub

m4dxTNnS%2FwOIugAuvcV2rjgw0lIxwDO8sptBhBCtTlqHlGRUPuc3O%2BQg%3D %3D&crl=c

- Thornhill, R., & Gangestad, S. W. (2006). Facial sexual dimorphism, developmental stability, and susceptibility to disease in men and women. *Evolution and Human Behavior*, 27(2), 131–144. doi:10.1016/j.evolhumbehav.2005.06.001
- Thornhill, R., & MOeLLER, A. (1997). Developmental stability, disease and medicine. Biological Reviews of the Cambridge Philosophical Society, 72(04), 497–548. Retrieved from http://journals.cambridge.org/abstract\_S0006323197005082
- Tomkins, J. L., & Kotiaho, J. S. (2002). Fluctuating asymmetry. doi:10.1038/npg.els.0003741
- Valen, L. Van. (1962). A study of fluctuating asymmetry. *Evolution*, 16(2), 125–142. Retrieved from http://www.jstor.org/stable/2406192
- Verdoux, H., & van Os, J. (2002). Psychotic symptoms in non-clinical populations and the continuum of psychosis. *Schizophrenia Research*, 54(1-2), 59–65. doi:10.1016/S0920-9964(01)00352-8
- Waddington, J., Lane, A., Larkin, C., & O'Callaghan, E. (1999). The neurodevelopmental basis of schizophrenia: Clinical clues from cerebro-craniofacial dysmorphogenesis, and the roots of a lifetime trajectory of disease. *Biological Psychiatry*, 3223(99). Retrieved from http://www.sciencedirect.com/science/article/pii/S0006322399000554
- Wilson, J., & Manning, J. (1996). Fluctuating asymmetry and age in children: Evolutionary implications for the control of developmental stability. *Journal of Human Evolution*, (1995). Retrieved from

http://www.sciencedirect.com/science/article/pii/S004724849690041X

- Winterstein, B., & Silvia, P. (2011). Brief assessment of schizotypy: Developing short forms of the Wisconsin Schizotypy Scales. *Personality and ..., 51*(8), 920–924. doi:10.1016/j.paid.2011.07.027
- Yeo, R. A., Gangestad, S. W., & Thoma, R. J. (2007). Science developmental instability and individual variation in brain development: Implications for the origin of neurodevelopmental disorders. doi:10.1111/j.1467-8721.2007.00513.x
- Yeo, R., Gangestad, S., Edgar, C., & Thoma, R. (1999). The evolutionary genetic underpinnings of schizophrenia: The developmental instability model. *Schizophrenia Research*, *39*, 197–206. Retrieved from

http://www.sciencedirect.com/science/article/pii/S0920996499000742

Zakharov, Y., Demin, D., & Baranov, A. (1997). Developmental stability and population dynamics of shrews Sorex in central Siberia. *Acta Theriologica*, (1), 41–48. Retrieved from http://rcin.org.pl/Content/12733/BI002\_2613\_Cz-40-2\_Acta-T42-Supp4-41-48\_0.pdf

# **Appendix A: SONA Recruitment Statement**

This study will look at the relationship between individuals' perceptions about the world and themselves, and certain physical characteristics. If you choose to participate, you will be asked to answer a set of 60 yes or no questions about yourself. You will also have several measurements taken. These will include the width of elbows, wrists, ankles and feet. Additionally a set of photographs of your face and hands will be taken. Participation is entirely voluntary, and you may discontinue your involvement with the experiment at any point without negative repercussions. To be eligible for this study, participants must be males between the ages of 18 and 23. Participation will take up to 1 hour. Two SONA credits are awarded for participation.

## **Appendix B: Consent Information Sheet**

#### College of Humanities and Behavioral Sciences Department of Psychology

#### **Consent Information Sheet**

Title of Research: Fluctuating Asymmetry and Developmental Markers Researcher(s): Dr. Donald M. Hall, Ivan Zuidhoek,

We request that you participate in a study that will examine the link between individuals' perceptions about themselves and the world and certain physical characteristics. Approximately 115 Radford University students will be asked to participate in this study. Participants will receive up to two SONA credits for participating.

If you decide to participate, you will be asked to answer a set of yes or no questions about yourself. Additionally, you will be asked to allow the researcher to take several bodily measurements. These will include measuring the width of your wrists, elbows, ankles and feet. You will also have a set of photographs taken of your face and hands. Photographs shall be kept for no longer than three weeks before they are destroyed. Photographs will not be used in any presentations or publications without obtaining additional permission from the participants.

This study has no more risk than you may find in daily life. There are no direct benefits to you for being in the study.

You can choose not to be in this study. If you decide to be in this study, you may choose not to answer certain questions or not to be in certain parts of this study. You may end your participation at any time without being penalized. It is your choice whether or not to be in this study. What you choose will not affect any current or future relationship with Radford University.

There is no financial incentive or cost for participating in this research.

If you choose to participate your responses will be kept confidential, unless the experimenter is required by law to tell. When this study's results are presented, no personal information will be disclosed.

If you have any questions later, you may contact Dr. Hall (dhall@radford.edu) Phone: (540) 831-5514, or Ivan Zuidhoek (izuidhoek@radford.edu).

This study was approved by the Radford University Committee for the Review of Human Subjects Research. If you have questions or concerns about your rights as a research subject or have complaints about this study, you should contact Dr. Dennis Grady, Dean, College of Graduate and Professional Studies, Radford University, dgrady4@radford.edu, 1-540-831-7163.

If you have questions now about this study, ask before you sign this form. If all of your questions have been answered and you would like to take part in this study, then please sign below.

Date

Signature

\

Printed Name(s)



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Appalachian Studies Communication Criminal Justice English Foreign Languages and Literatures Geography Media Studies Military Science Philosophy and Religious Studies Political Science Sociology and Anthropology I/We have explained the study to the person signing above, have allowed an opportunity for questions, and have answered all of his/her questions. I/We believe that the subject understands this information.

Signature of Researcher(s)

Printed Name(s)

Date

Note: A signed copy of this form will be provided for your records.

# **Appendix C: Demographic Questions**

# What year were you born?

What is your ethnicity?

African American \_\_\_\_\_ European American (Caucasian) \_\_\_\_\_ Asian/Pacific-Islander American \_\_\_\_\_ Hispanic/Latino American \_\_\_\_\_ Native American/American Indian \_\_\_\_\_ Other \_\_\_\_\_

# What is your relationship status?

Single
Dating but not cohabitating (living together)
Dating and cohabitating (living together)
Married
Separated
Divorced
Widowed
What is your current GPA?

Have you ever fractured or broken one or more bones? If so please list which bones below.

	11		
Date		_	
Participant #_			
		Measurement 1	
	LEFT		RIGHT
Ear Width		_	
Earl Length		-	
Elbow Width		_	
Wrist Width		_	
Ankle Width		_	
Foot Width		_	

# Appendix D: Skeletal Trait Sheet

Measurement 2

	LEFT	RIGHT
Ear Width		
Earl Length		
Elbow Width		
Wrist Width		
Ankle Width		
Foot Width		