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Yosefa A. Modiano

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DERMATOGLYPHIC MEASURES IN RELATION TO DEPRESSIVE SYMPTOMS AMONG NON-CLINICAL ADOLESCENTS AND YOUNG ADULTS

by

YOSEFA ALLEGRA MODIANO, M.A., M.PHIL.

A dissertation submitted to the Graduate Faculty in the Psychology in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City University of New York

2019
DERMATOGLYPHICS AND DEPRESSIVE SYMPTOMS

Dermatoglyphic Measures in Relation to Depressive Symptoms Among Non-Clinical Adolescents and Young Adults

by

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This manuscript has been read and accepted for the Graduate Faculty in Psychology in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

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ABSTRACT
Depressive disorders are highly prevalent and can be devastating. Increasingly, depressive symptomatology is understood from a dimensional perspective such that non- or sub-clinical presentations may share a similar etiology. Depression etiology is believed to include genetic and environmental factors that may contribute to underlying vulnerability (diathesis) by way of neurodevelopment. Birth cohort studies have provided empirical evidence of the relationship between prenatal insult and later experience of adverse outcomes, including increased risk for depressive disorders. Retrospective investigation of the possible influence of prenatal disturbance on later experience of depressive symptoms has methodological limitations. Dermatoglyphic measurements offer a more methodologically viable (albeit indirect) proxy for estimating prenatal insult. Digit dermatoglyphics refer to fingerprint symmetry and patterns. Fingerprints develop concurrently with brain structures implicated in risk for depression. Thus, dermatoglyphic abnormalities such as fluctuating asymmetry (FA), or deviations in dermatoglyphic symmetry, and low ridge counts may illuminate the potential contribution of prenatal insult to later expression of depressive symptoms. Prior research has demonstrated relationships among dermatoglyphic measures (FA and ridge counts) and psychological symptoms across a wide range of non-clinical, mixed, and clinical populations. The current investigation primarily aimed to expand this body of literature by investigating the predictive
relationships among dermatoglyphic measures and depressive symptom endorsement in a sample of non-clinical adolescents and young adults from the general population. The secondary aim was to examine sex as a potential moderator of the relationships among dermatoglyphic measures and depressive symptoms. Participants were a subsample of individuals recruited as part of a larger study assessing factors implicated in depression risk and included $n = 53$ (22 M / 31 F) adolescents and young adults ($M_{age} = 20.04$, $SD_{age} = 1.05$). For the current report, measures included fingerprints from which four indices of FA and two finger ridge count measures were derived, as well as the total score from the Beck Depression Inventory II (BDI-II), a self-report measure of depressive symptoms. In line with hypotheses, one index of FA significantly positively predicted depressive symptoms after correcting for multiple comparisons, suggesting that neurodevelopmental factors may contribute to depressive symptoms. Findings did not hold, however, after taking multicollinearity into account. Sex did not moderate any relationships among dermatoglyphic measures and depressive symptoms. This research has potential implications for understanding risk for depression. Future work may benefit from assessing additional dermatoglyphic measures corresponding to different gestational periods, using larger samples, and alternative methods to address multicollinearity.
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CHAPTER I.

INTRODUCTION

Depression describes a transient mood state as well as a group of clinical biobehavioral syndromes comprised of a cluster of mood, cognitive and neurovegetative symptoms. Due to early age at onset (Hagan et al., 2015; Kessler et al., 2003), high prevalence rates ranging from 2-7% across studies (Kessler et al., 2003; Nestler et al., 2002; APA, 2013), disease chronicity (Kessler et al., 2005), and hefty disease burden in terms of societal, monetary and personal costs (Ferrari et al., 2013; Greenberg et al., 2003; Whiteford et al., 2013), depressive disorders are the focus of much investigation. Depressive symptomatology is understood to exist along a continuum with symptoms ranging from non-clinical to subthreshold to clinically significant (Cuijpers, De Graaf, & Van Dorsselaer, 2004; Fergusson, Horwood, Ridder, & Beautrais, 2005; Lewinsohn, Solomon, Seeley, & Zeiss, 2000). Individuals (often adolescents) with depressive symptoms in the non-clinical and subthreshold ranges are at heightened risk for negative outcomes, including conversion to clinical depression (Cuijpers et al., 2004; Fergusson et al., 2005), and are therefore an important population to assess towards better understanding depression etiology. The diathesis-stress model holds that depressive disorders develop as a function of the interaction between a stressor and underlying vulnerabilities (e.g., genetic, dispositional) (Rosenthal, 1963; Zubin & Spring, 1977). Within the diathesis-stress framework, evidence of heightened risk for depressive disorders following perturbations that impact aspects of neurological development (e.g., heightened maternal stress (Glover, 2014; O’Connor, Heron, Golding, & Glover, 2003; Van Den Bergh, Mulder, Mennes, & Glover, 2005), maternal mood factors (O’Donnell, Glover, Barker, & O’Connor, 2014; Pearson et al., 2013), and teratogen exposure (Huizink et al., 2007)) has led to investigation of neurodevelopment as one such
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vulnerability that may increase the risk for depressive disorders. Understanding the nature and timing of developmental events may be helpful in clarifying the impact of early acquired insults. Ultimately, that may illuminate the role of neurodevelopment in later expression of depressive disorders.

However, retrospective examination of factors influencing neurodevelopment has methodological limitations including lack of randomized design and biased recall. In order to circumvent these limitations, dermatoglyphic measurements of fluctuating asymmetry (FA) and ridge counts offer a proxy for retrospective assessment of neurodevelopment. These morphologic features, which are relatively stable over the course of development (Babler, 1978), are easily measured and understood to reflect genetic and environmentally mediated disruptions in early neurodevelopment. Dermatoglyphic measures have been associated with a host of psychological (Shackelford & Larsen, 1997), cognitive (Banks, Batchelor, & McDaniel, 2010) and physiological (Manning, Koukourakis, & Brodie, 1997) outcomes associated with irregular brain development, including subclinical depression (Martin, Manning, & Dowrick, 1999; Shackelford & Larsen, 1997) in both cross-sectional and longitudinal studies.

The current investigation intended to expand the body of literature regarding the role of aberrant neurodevelopment in sub-clinical experience of depressive symptoms. The primary aim was to investigate the cross-sectional relationship of self-reported depressive symptoms with indicators of neurodevelopmental insult (i.e., dermatoglyphic indices) in a non-clinical sample of adolescents and young adults recruited from the general population. While dermatoglyphics do not by themselves have the specificity or sensitivity for MDD diagnostic purposes, better
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understanding neurodevelopmental pathophysiology holds implications for improving prevention and diagnosis of depression-related disorders.

1. Depressive Disorders

Depression refers to a mood state recognized as early as classical times. Hippocrates described melancholia as a distinct disorder in his 400 C.E. writing, *Aphorisms*, and recognized that expressions of sorrow, grief and despondency spring from the brain (see Francis, 1886 for translation). Drawing on Emil Kraepelin’s symptomatic classification system, the field currently understands clinical depression as a group of biobehavioral illnesses comprised of a cluster of symptoms, including abnormalities of mood and affect, neurovegetative functions, and dysfunctional cognitions and psychomotor activity. Key features in a diagnosis of Major Depressive Disorder (MDD) include depressed mood, anhedonia, weight loss or gain, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue, negative cognitions, reduced concentration, and thoughts of death (see DSM-5; APA, 2013). Among the various diagnostic criteria are factors such as symptom intensity, duration, and functional impact. The DSM-5 diagnostic classification system includes qualifiers of ‘mild’, ‘moderate’ and ‘severe’ to reflect the varying dimensions of disorder severity.

1.1 Epidemiology of Depressive Disorders

MDD is a common and devastating neuropsychiatric disorder. Among the depressive disorders listed in the DSM-5, MDD is the most prevalent; twelve-month prevalence figures of MDD in the United States vary between 2-7% across studies (Kessler et al., 2003; Nestler et al., 2002; APA, 2013). Estimates suggest that 16.6% of adults have experienced a depressive disorder at
some point in their life (Kessler et al., 2003). Women are 1.5 to 2 times more likely to develop mood disorders compared to men (Galambos, Leadbeater, & Barker, 2004; Kessler et al., 2003; Leach, Christensen, Mackinnon, Windsor, & Butterworth, 2008); White and Hispanic populations are also more susceptible relative to other ethnic groups (Kessler et al., 2005). A global burden of disease study (Whiteford et al., 2013) showed that mental and substance use disorders contribute to 7.4% of the global burden of disease worldwide, ranking fifth in the list of leading causes. MDD alone ranked 11th as a leading cause of global disease burden and was the second leading cause of years lived with a disability (Ferrari et al., 2013). Direct medical costs associated with depressive disorders have been estimated at $26.1 billion and rising (Greenberg et al., 2003). Projections for annual loss to the US labor force due to MDD range between $36.6-51.5 billion (Greenberg et al., 2003; Kessler et al., 2006). Depression is also associated with a host of negative health problems, including obesity (Pratt & Brody, 2014), poor immune function (Kiecolt-Glaser & Glaser, 2002), heart disease (Whang et al., 2009), and suicide (Whiteford et al., 2013). A global estimate demonstrated that MDD accounted for 46.1% of suicides in a given year (Whiteford et al., 2013).

1.2 Illness Course

MDD is often considered a life-long disorder with multiple episodic recurrences. While the average age of onset for MDD is 30 years (Kessler et al., 2005), symptoms are often apparent in childhood or adolescence (Jones, 2013). Risk for developing a depressive disorder increases dramatically in adolescence (Kessler et al., 2003; APA, 2013), suggesting that aspects of adolescence (e.g., developmental, hormonal, neurophysiological, and social changes) may be implicated in the emergence of the disorder. The fact that illness onset often occurs in early
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Adulthood, in combination with a dimensional approach (presented below), has refocused some research efforts towards investigation of individuals in the pre-clinical phase of disorder onset in an effort to elucidate underlying etiological factors.

1.3 The Dimensional Theory of Depressive Disorders

Increasingly, depressive symptomatology is understood to exist along a continuum rather than in discrete categories (Cuijpers et al., 2004; Goldberg, 2000; Hankin, Fraley, Lahey & Waldman, 2005; Lewinsohn et al., 2000; Solomon, Haaga & Arnow, 2001). On the less severe end of the spectrum are mildly affected, non-clinical individuals from the general public who may endorse a variety of depressive symptoms below the range associated with impairment. Compared with the non-clinical cohort, individuals with subthreshold depression experience greater frequency or severity of symptoms, although they do not meet full criteria for a diagnosis. At the clinical end of the spectrum are individuals who meet full DSM-5 criteria for a major depressive episode, which is in and of itself then further subdivided into ranges of severity (as discussed above).

There are many clinical correlates shared by individuals across the depression spectrum, including overlap between age of onset, symptom course, family history, and comorbid diagnoses in both major and subthreshold depression (for review, see Solomon et al., 2001). Individuals with subclinical symptoms also share elevations in risk of future major depressive episodes (Cuijpers et al., 2004; Fergusson et al., 2005), substance use disorders (Lewinsohn et al., 2000), and suicidality (Fergusson et al., 2005) with those who meet full criteria for MDD. Impairments to physical, psychological, and social functioning are similar regardless of whether full criteria for MDD are met (Cuijpers et al., 2004). Imaging research has shown that individuals
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with subclinical MDD have neural organization that is more similar to those with MDD than healthy controls, suggesting that the neural correlates of depression do not map onto discrete diagnostic categories (Gao et al., 2016; Li et al., 2015). A variety of statistical methods have also lent support for the dimensional theory by demonstrating continuous distribution of depressive symptoms (see Hankin et al., 2005).

In sum, the dimensional framework of depression is gaining increasing empirical support. It suggests that shared etiological and developmental courses underlie a range of depressive presentations. Accordingly, subclinical depression is an important diagnostic population from a research perspective, as it may indicate factors related to timing and disorder severity that are important for prevention and treatment strategies.

1.4 General Theoretical Models of Depressive Disorder Etiology

A multitude of frameworks (psychological and biological) have been proposed to understand depressive disorder etiology. Among the psychological theories are Freud’s ideas of loss and inward aggression (Freud, 1924) and Beck’s theory of cognitive distortions (Beck, 1967). Other psychological factors implicated in depression include personality traits of neuroticism and negative emotionality (Klein, Kotov, & Bufferd, 2011), as well as aspects of self-esteem (Orth, Robins, Widaman, & Conger, 2014). From a biological perspective, depressive disorders are associated with a range of genetic, neurochemical, neurophysiological, and neuroanatomical abnormalities. Twin studies and heritability tests indicate a genetic component to the illness (Flint & Kendler, 2014), and genome-wide associations are increasing efforts to identify single-nucleotide polymorphisms associated with aspects of mood disorders, such as the serotonin
transporter gene (5-HTTLPR) (Levinson, 2006). Neurochemical theories draw from the effectiveness of psychopharmacological treatment in minimizing mood symptoms and posit that an imbalance of neurochemicals (via underproduction, excessive reuptake or degradation of neurochemicals, and up- or down-regulation of neuronal receptors) may be responsible for the illness (for review see Ferrari & Villa, 2016).

Neuroanatomically, several key areas implicated in depression are limbic and frontal structures and their connective pathways (for reviews see Bracht, Linden, & Keedwell, 2015; Brand, Moller, & Harvey, 2015; Mayberg, 2003; Zhang et al., 2016), as well as components of the hypothalamic-pituitary adrenal (HPA) axis (for reviews see Nemeroff & Vale, 2004; Pariante & Lightman, 2008). In particular, hippocampal volume loss up to 15% (Campbell & MacQueen, 2004), including reductions in both hippocampal grey-matter and glial density, is found in individuals with depression. Reduced hippocampal volume correlates with the duration of depressive symptoms (MacQueen et al., 2003). Volume reductions are also identified in the prefrontal cortex (Sheline, 2003), ventral and medial frontal cortices (Rajkowska, 2000), and other medial temporal structures (e.g., entorhinal cortex) (Gerritsen et al., 2011) of depressed individuals. Furthermore, individuals with depression have disruptions in fronto-limbic connectivity (Seminowicz et al., 2004), and aspects of dysfunctional cortical-limbic pathways are predictive of depression course (Mayberg, 2003). As far as dysregulated HPA axis functioning, depressed patients have elevated levels of cortisol, as well as increased pituitary and adrenal glands (Nemeroff & Vale, 2004) and impaired glucocorticoid-mediated feedback inhibition (Pariante, 2006).
Early etiological models of depressive disorders typically focused on one aspect thought to underlie the illness process (e.g., irrational beliefs, monoamine depletion). Research is increasingly, however, drawing upon theories that integrate multiple factors to understand depression. The diathesis-stress model integrates both genetic and environmental factors, as well as their interactions, in understanding depression etiology (Rosenthal, 1963). Within the framework of this well-established integrated model, neurodevelopment is one domain that jointly reflects aspects of genetic heritability and early environmental exposure, and thus may contribute to illness diathesis.

1.5 Integrated Models of Depressive Disorder Etiology: Diathesis-Stress

The diathesis-stress theory of psychopathology (Rosenthal, 1963; Zubin & Spring, 1977) takes into account a range of psychological and biological factors that increase risk. According to this model, major life stressors (defined as significant events that disrupt one’s physiological and psychological homeostasis) interact with latent genetic, biological, and psychological vulnerability factors (i.e., diathesis) to increase likelihood of psychopathology emergence (for review see Ingram & Luxton, 2005). Within the sphere of depressive disorders, this theory draws on the notion that stressful life events increase risk for depressive symptomatology (for review see Hammen, 2005). Incidence of significant stressors is 2.5 times higher in individuals with MDD compared with controls (Mazure, 1998). At the same time, it is clear that not all individuals exposed to stress experience pathological reactions. Accordingly, endogenous factors that contribute to risk are also considered. Such factors include genetic, neurochemical, neurophysiological, and neuroanatomical contributions to depression risk. Each of these biological domains may reflect, in part, aberrant neurodevelopment that increases vulnerability.
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to the disorder. Ultimately, the emergence of a depressive disorder is determined by an interaction between the extent and impact of stressors and aspects of underlying vulnerability. Better understanding of neurodevelopmental factors is therefore significant in illuminating these vulnerabilities.

1.6 Neurodevelopmental Model of Depressive Disorder Etiology

The process of human ontogeny is jointly determined by genetic code and the environment in which an organism develops. As depressive disorders are understood to have complex polygenic origins, a growing body of literature focuses on the role of early (e.g., prenatal) developmental factors that may impact neurodevelopment in understanding depression etiology. In the following section, we briefly review aspects of brain development focusing on structures implicated in depressive disorders. We then present evidence for a neurodevelopmental contribution to depression.

1.6.1 Central Nervous System Development. The central nervous system (CNS) undergoes crucial development throughout the prenatal period. However, primates, including humans, are unique in that nearly all cortical neurons are acquired during the first half of gestation (see Rakic, 1998), rendering the early prenatal period a critical window for neurodevelopment. The neural plate from which the CNS develops is evident approximately 17 days after conception (Rakic, 1998). And while neural cell migration, proliferation, and differentiation occur at different time points, the hippocampus is one of the first cortical areas to become apparent at week 9 of gestation (Arnold & Trojanowski, 1996; Kier, Kim, Fulbright, & Bronen, 1997). The hippocampus undergoes rapid growth during gestational weeks 14-22 (Ge et
al., 2015), and by approximately week 21, the hippocampal sulcus is related to surrounding structures in similar proportions to the adult brain (Jacob et al., 2011). Entorhinal and parahippocampal differentiation begin around gestational week 13, and their connections to the hippocampus, as well as other frontal association areas, are established by week 19 (Kostović, Petanjek, & Judas, 1993). With regards to aberrant structural formation, the timing of prenatal insults is crucial. Perturbations (e.g., viral infection, malnourishment, teratogen exposure) during the second trimester may adversely impact limbic structures, which undergo rapid neuronal proliferation, migration and differentiation during this period (Nowakowski & Hayes, 1999; Welberg & Seckl, 2001).

1.6.2 Role of the Limbic System in Depressive Disorders. Limbic system structures and their connective pathways, established during weeks 9-22 of gestational life, are highly susceptible to inputs during prenatal periods (Qiu et al., 2013, 2015) and are widely implicated in depressive disorders (Bracht et al., 2015; Brand et al., 2015; Hoogenboom et al., 2014; Jalbrzikowski et al., 2017; Oakes, Loukas, Oskouian & Tubbs, 2016; Zhang et al., 2016). As reviewed earlier, individuals with depressive disorders are commonly shown to have reduced gray matter volume in limbic structures (Brand et al., 2015; Zhang et al., 2016) and decreased white matter connectivity between limbic and frontal structures (Bracht et al., 2015; Hoogenboom et al., 2014; Jalbrzikowski et al., 2017). One explanation for the frequent observation of reduced hippocampal volume in depressed patients involves HPA axis dysregulation. The hippocampus contains glucocorticoid receptors that induce feedback inhibition to decrease HPA activity. Reduced HPA inhibition in individuals with depressive disorders may be a result of hippocampal cell death and atrophy due to chronic glucocorticoid
exposure from repeated depressive episodes and/or early abnormal programming (Pariante & Lightman, 2008). Conversely, abnormal hippocampal development may also be responsible for reduced HPA inhibition (Knoops, Gerritsen, van der Graaf, Willem, & Geerlings, 2010).

1.6.3 Evidence of the Relationship Between Neurodevelopment and Depressive Disorders.

Much evidence for the link between aberrant neurodevelopment and depression risk is drawn from birth cohort studies. A host of prenatal conditions associated with increased risk of affective disorders have been identified. Such factors include maternal experience of: heightened stress (Huizink et al., 2007; Huizink, Robles de Medina, Mulder, Visser, & Buitelaar, 2003; O’Connor, Heron, Golding, Beveridge, & Glover, 2002; O’Connor et al., 2003; Van Den Bergh et al., 2005), aberrant mood states (i.e., depression, anxiety) (O’Donnell et al., 2014; Pearson et al., 2013), maladaptive immune response (Bale, 2009; Goel & Bale, 2009), viral infections (Machón, Mednick, & Huttunen, 1997), heightened exposure to teratogens (such as radioactive material) (Huizink et al., 2007), and malnourishment (Brown, Susser, Lin, Neugebauer, & Gorman, 1995; Brown, van Os, Driessens, Hoek, & Susser, 2000). Although these varied prenatal insults likely differentially impact fetal development, it is possible they are all, in part, acting on shared mechanisms (i.e., limbic structure connectivity, HPA programming) (Bale et al., 2011).

2. Dermatoglyphics

As demonstrated, there is a compelling body of evidence suggesting that the neurobiological characteristics of depression reflect, in part, neurodevelopmental disturbance. However, outside of animal models, resource-demanding longitudinal designs, and cohort studies capturing unique
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circumstances (e.g., natural disasters including Chernobyl (Huizink et al., 2007), ice storms (King, Dancause, Turcotte-Tremblay, Veru, & Laplante, 2012) and famines (Brown et al., 1995, 2000)), the impact of neurodevelopment on later outcomes is difficult to study. Limitations include high costs associated with longitudinal research, lack of randomized experimental design, and inherent difficulties in retroactive measurement (e.g., recall bias). Dermatoglyphics, or measurement of skin ridges, offer an easier method for retroactive investigation into the nature and timing of early developmental disturbance that may impact brain structure and function.

The term dermatoglyphics was proposed in 1926 to refer to the epidermal skin ridge formations on the tips of fingers, palms of hands and soles of feet. Derived from Greek (derma = skin + glyphe = carve), the term dermatoglyphics also refers to the field of its study (Cummins & Midlo, 1926). Fascination with the unique patterns of hands and feet dates back to ancient times and has captured the attention of doctors, scholars, artists, and others for centuries (Cummins & Midlo, 1961). The varieties of finger patterns were first classified by J. E. Purkinje, the Czech anatomist who most famously discovered Purkinje cells of the cerebellum (Purkinje, 1823). Sir Francis Galton, who contributed immensely to statistical measurement and psychological theory, was among the first scholars to observe that ridge configurations remain unchanged, as he tracked his own patterns across his lifetime (Galton, 1892). However, it was not until 1939 when H. Cummins published his seminal findings on unusual dermatoglyphic patterns in individuals with Down syndrome (Cummins, 1939) that the field of dermatoglyphic study launched to clinical significance. Since that time, dermatoglyphics have been investigated in relation to multiple congenital defects originating from both genetic and environmental aberrations as well as physical, psychological, and cognitive sequelae (discussed below).
2.1 Dermatoglyphic Development

The process and determinants of dermatoglyphic development are timing specific. During weeks 5 to 6 post-fertilization, the volar (finger) pads of the hand become evident (Babler, 1991). By the second month of gestational life, individual fingers become elongated and separated with protruded finger pads. Between weeks 10 to 17 of development, the finger pads begin to regress, and the precise configuration of the epidermal ridges is determined. Secondary development continues until week 24. Thus, weeks 10 to 24 of gestation reflect the critical window of ridge differentiation. By the end of this gestational period, the ridges are comparable to adult morphology (for further discussion, see Babler, 1991) and only under very rare circumstances undergo further change (Cummins & Midlo, 1961).

The exact influences on ridge configuration are not entirely understood; however, it is evident that genetic and environmental factors each play a role in fingerprint pattern development. Ontogenetic factors indirectly influence ridge alignment through pad topography (Babler, 1991). The height of the volar pad during the period of epidermal differentiation determines the ridge pattern. A high, round pad results in a whorl pattern, which is typically characterized by multiple triradii (discussed further below; see Figure 1), resulting in high ridge counts. An intermediate pad is associated with development of a loop pattern with correspondingly intermediate ridge counts, while a low pad results in an arch, a pattern defined by a ridge count of zero (Babler, 1991). However, this process is further mediated by growth rates and epidermis stress, each of which is only partly determined by genetics. Dermatoglyphic features are shown to reflect embryonic stress due to various environmental factors, including hypoxia (Mulvihill & Smith, 1991).
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1969), fetal alcohol syndrome (Qazi, Masakawa, McGann, & Woods, 1980), rubella (Purvis-Smith & Menser, 1973), and cytomegalovirus (Wright, Parker, & Mavalwala, 1972). In particular, the intensity, duration, and timing of the insult, as well as genetic resistance, determine dermatoglyphic patterns (King et al., 2009). In sum, the ridge configurations of the fingertips emerge between gestational weeks 10-24, are determined by ontogenetic and environmental factors, and remain “fossilized” throughout one’s lifetime.

2.1.1 Fluctuating Asymmetry. Symmetry is a commonly investigated aspect of dermatoglyphic measurements based on the concept of developmental stability. Developmental stability refers to an individual’s ability to develop in accordance with his or her ontogenic plan despite disruptive environmental factors (Livshits & Kobyliansky, 1991). In other words, developmental stability describes an organism’s ability to buffer against such disruption via employment of homeostatic mechanisms (Clarke & McKenzie, 1992). The inverse, developmental instability, is the degree to which the developmental trajectory is influenced by exogenous insult. The construct of developmental instability (decreased buffering capacity) is measured by assessing deviations from symmetry of bilateral traits, referred to as fluctuating asymmetry (FA) (Kowner, 2001). Bilateral development is under the control of the same genome; thus, deviations from symmetry in finger ridge counts, for example, offers a quantifiable measurement of the degree to which development was influenced by exogenous factors. Increased FA variability is associated with a host of intrinsic and extrinsic factors that influence gestational development as well as later expression of cognitive, psychological, and behavioral differences (further discussed below) (for review see: Livshits & Kobyliansky, 1991).
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2.1.2 Ridge Counts. While FA refers to deviations in any bilateral physical trait (e.g., finger ridge counts, digit length, wrist diameter, ear size, eye distance), ridge counts more specifically refer to the dermatoglyphics of digits, palms and soles (Cummins & Midlo, 1961). Digit dermatoglyphics are of particular interest, as they are easily measured, quantifiable, and subject to minimal alteration after birth. Ridge counts serve as a proxy for measuring the size and intensity of a fingerprint pattern (Cummins & Midlo, 1961; Schaumann & Alter, 1976).

The three primary fingerprint patterns (whorl, loop and arch) were first defined by Galton and contain morphologically distinct features (see Cummins & Midlo, 1961 for complete discussion), as shown in Figure 1. A whorl, the most elaborate pattern, is distinguished by a concentric design where the ridges form circuits around a core, interior feature. It is usually comprised of multiple triradii, the meeting point of three opposing ridge systems. A loop pattern is simpler, containing a single triradius and ridges curving in one direction towards the margins of the digit. An arch is the simplest detectable pattern and is typically described as “patternless” in that the ridges pass the range of the digit without forming a distinguishable triradial or central point. Ridges are counted along a straight line drawn between a triradial and central point of the pattern. There are two types of ridge counts that can be computed: total and absolute. Total finger ridge counts (TFRC) refer to the number of ridges crossing the longest straight line drawn between the triradius and the core. In loop patterns there is only one available line from which TFRCs can be measured. However, whorl patterns are characterized by multiple triradii. Absolute finger ridge counts (AFRC) include ridges counted from all lines drawn between a pattern’s triradius and core and are therefore expected to be larger than TFRCs.
Figure 1. Depictions of the most common dermatoglyphic patterns: loops, whorls, and arches. Image from Jatti, Kantraj, & Nagaraju (2014)

Figure 2. (Left) Example of a loop dermatoglyphic pattern. The triradius, or meeting point of three opposing ridge systems, is outlined in red. The core, or center of the pattern is outlined in blue. (Right) Ridges are counted by drawing a straight line from the triradius to the core (yellow) and counting the number of ridges crossing the line. This print has a total finger ridge count (TFRC) of 8 (the ridges comprising the triradius and core are not counted). Characteristic of the loop pattern, there is a single triradius; accordingly, the absolute finger ridge count (AFRC) is also 8.
Image from Golembo-Smith et al. (2012)
Finger ridge counts are understood to represent the rate of fetal growth such that higher counts reflect more rapid cell division during the first and second trimesters of prenatal development (Cohen-Bendahan, Van de Beek, & Berenbaum, 2005; Newell-Morris, Fahrenbruch, & Sackett, 1989). As ridge patterns become more elaborate during gestational weeks 10-19, fetal susceptibility to developmental insult (both genetic and environmental) during that period may result in lower counts (Babler, 1978). Research shows that a higher incidence of arch patterns (lowest ridge counts) is associated with a host of congenital and environmental defects (i.e., chromosomal defects, congenital heart disease, and prenatal rubella (see Babler, 1978 for further discussion)). A higher frequency of arch patterns has also been detected in spontaneous fetal death (Babler, 1978), suggesting that factors adversely impacting ontogeny may be detected in fingerprint patterns. Aside from reflecting differences in patterns (e.g., whorl v. loop v. arch), the distinction between TFRCs and AFRCs is not entirely clear; however, it has been posited that AFRCs may be more sensitive to non-genetic influences, such as environmental prenatal insults (Schaumann & Alter, 1976).

Dermatoglyphic FA can be captured by assessing differences in TFRC and/or AFRC. While greater FA using TFRCs captures subtle differences in counts (bidirectional asymmetry), greater FA using AFRCs often reflects differences in overall fingerprint patterns, which is understood to indicate more significant environmental contributions to asymmetric development. Previous studies have assessed for pattern matches in a categorical manner (for review see Golembo-Smith et al., 2012); however, using FA counts reduces subjectivity by capturing these changes in more statistically versatile variables (continuous, ratio data).
3. Dermatoglyphics and Neurodevelopment

Epidermis cell proliferation and differentiation undergo major development during the same critical period as the CNS. Furthermore, the nervous system and the skin both derive from migration of the same fetal ectoderm (Cummins & Midlo, 1961). As discussed earlier, the majority of neural cell proliferation occurs during the first half of gestation in primates, marking the early prenatal period a critical window for neurodevelopment (Rakic, 1998). Of significance, many of the brain regions (e.g., limbic structures (Ge et al., 2015; Jacob et al., 2011)) and their connective pathways (e.g., fronto-limbic connections (Kostović & Judaš, 2010; Kostović, Petanjek & Judaš, 1993) that are established during weeks 9-22 of gestational life are implicated in depressive disorders (Bracht et al., 2015; Brand et al., 2015; Zhang et al., 2016) and overlap with epidermal ridge formation (gestational weeks 10-24).

3.1 Evidence of the Relationship among FA, Dermatoglyphics, Neurodevelopment, and Clinical Outcome

Fluctuating asymmetry (FA) of dermatoglyphics and other morphologic traits is associated with a range of genetic and prenatal environmental insults impacting neural ontogeny (for review see Kowner, 2001; Thornhill & Møller, 1997). Genetic contributions to FA are seen in the form of heightened asymmetry of morphologic traits due to inbreeding (Markow & Martin, 1993), homozygosity (Mitton, 1995; Mitton & Grant, 1984), deleterious recessives (Parsons, 1990), and chromosomal abnormalities (Fraser, 1994; Shapiro, 1992). Environmental perturbations impacting FA include maternal stress (King et al., 2009), health factors (e.g., blood pressure, obesity and teratogens) (Kieser & Groeneveld, 1993; Kieser, 1992; Livshits et al., 1988),
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exposure to parasites, pathogens (Møller, 1996; Polak, 1997), and pollutants (Parsons, 1990, 1992), and adverse physical conditions, such as extreme temperatures, during gestation (Harris & Nweeia, 1980; Parsons, 1990, 1992). Dermatoglyphic abnormalities are also related to genetically-mediated disorders (e.g., Down syndrome and Fragile-X syndrome) (Langenbeck, Varga, & Hansmann, 1988; Shapiro, 1992), markers of physiological fitness (Manning et al., 1997), cognitive outcomes (Banks et al., 2010) and emotional and psychological health correlates (reviewed further below). Taken together, these findings provide evidence intimating that prenatal insult leaves indelible bodily marks that are related to later expression of clinical/functional symptoms.

Maternal prenatal stress is one particular insult that has been a focus of investigation in relation to expression of dermatoglyphic measurements (King et al., 2009) as well as risk for internalizing disorders (King et al., 2012). King et al. (2009) conducted a cohort study of mothers ($n = 97$) who were pregnant during a devastating ice storm in Canada in 1998 that knocked out power for as long as 40 days during the coldest months of the year. By studying the effects of the natural disaster on offspring digit asymmetry, these researchers were able to: (1) identify the overall impacts of stress on that proxy for neurodevelopment; and (2) assess the effect of stressor timing on dermatoglyphic outcomes. The research team found significant correlations between greater maternal objective stress (as measured by threat, scope, change and loss), higher maternal subjective stress, and increased digit asymmetry in offspring at age 5.5 years. Regarding timing, the offspring of women who were between gestational weeks 14-22 during the storm who reported high levels of objective stress had a greater degree of asymmetry relative to offspring affected during other gestational periods. These findings offer direct
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evidence of the relationship between maternal stress and a measure of dermatoglyphic FA, which, in turn, indirectly informs us about the possibility of disturbance to neural structures that develop contiguously. Using this same cohort of mothers, King et al. (2012) also showed that greater subjective maternal stress during the storm was significantly related to offspring internalizing problems (anxiety, depression, social withdrawal) at ages 4-11 years. Moreover, the severity of the internalizing problems was shown to increase over time relative to a group of unaffected peers. As of yet, there is no known published study from this research group that collapses across topics by investigating maternal stress, stressor timing, dermatoglyphic asymmetry and later outcome of internalizing disorder altogether.

In sum, the temporal overlap between dermatoglyphic formation and neuroanatomical development suggests that these markings may elucidate aspects of neurodevelopment associated with later expression of psychopathology, including depression (Martin et al., 1999; Shackelford & Larsen, 1997). As dermatoglyphic markings generally remain fixed across one’s lifetime, they demarcate a relatively stable and narrow time stamp of gestation. Furthermore, the FA measurements and ridge counts reflect a combination of genetic and environmental factors. Although the genetic aspects are not entirely understood, endogenous events that are reflected in dermatoglyphic alterations (e.g., stress, infections, nutritional deficits) are also associated with depression (as discussed above). Finally, dermatoglyphics are easily measured in a standardized and non-invasive procedure that can be conducted at any age. Thus, these markings offer a methodologically viable proxy allowing for investigation into the relationships between aberrant early environmental conditions, neurodevelopment and later expression of depressive symptoms.
4. Dermatoglyphics and Psychopathology

There is a body of literature investigating the relationships between dermatoglyphic abnormalities and psychopathology in clinical and non-clinical samples (Benderlioglu, Sciulli, & Nelson, 2004; Burton et al., 2003; Campbell, Geller, Small, Petti & Ferris, 1978; Daly, Gooding, Jessen, & Auger, 2008; de Bruin, Graham, Louwerse, & Huizink, 2014; Glamuzina, Mihanovic, Milicic, Devcic, & Restek-Petrovic, 2009; Golembo-Smith et al., 2012; Jelovac et al., 1998; Jelovac, Milicic, Rudan, & Milas, 1995; Martin et al., 1999; Shackelford & Larsen, 1997; Shrivastava, Mathur, Dhaneria, & Goyal, 2006; Stevenson et al., 2006; Vonk et al., 2014). A large majority of the research investigates dermatoglyphic measures in relation to psychosis, and in particular, schizophrenia spectrum disorders (for review see Golembo-smith et al., 2012), as they are widely believed to have neurodevelopmental origins. The literature also includes investigations of clinical conditions with neurodevelopmental components such as autism spectrum disorders (ASD) (Campbell et al., 1978; de Bruin et al., 2014) and attention deficit hyperactivity disorder (ADHD) (Burton et al., 2003; Stevenson et al., 2006). Further research examines bipolar disorder (BD) (Shrivastava et al., 2016; Vonk et al., 2014), borderline personality disorder (BPD) (Jelovac et al., 1998; Jelovac et al., 1995), and post-traumatic stress disorder (PTSD) (Glamuzina et al., 2009). Measures of mood and emotional well-being (including depressive symptoms and reactive aggression) are explored in relation to dermatoglyphic measures in non-clinical samples, as well (Benderlioglu et al., 2004; Martin et al., 1999; Shackelford & Larsen, 1997). Despite theoretically compelling evidence (presented throughout), empirical evidence linking dermatoglyphics and clinical depression (MDD) is more limited, as detailed below.
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4.1 Dermatoglyphic/FA Measures in Non-Clinical Samples

Shackelford and Larsen (1997) investigated FA as it relates to depressive symptoms, as well as other measures of psychological wellbeing, in a non-clinical sample. This study included college students from the general population ($n = 101$; $34 \text{ M} / 67 \text{ F}; M_{\text{age}} = \sim 20$) who completed a battery of measures assessing psychological and personality variables. Depressive symptom endorsement was assessed using the Beck Depression Inventory, a validated measure providing an index of depressive symptoms (BDI; Beck, 1967). Various other validated self-report measures were administered to explore anxiety, personality features and behavioral tendencies. Each subject was photographed, and various measures of facial symmetry (eye height, nostril, cheekbone and jaw width) were derived as a proxy for developmental instability in keeping with the method proposed by Grammer & Thornhill (1994). A significant relationship was established between greater facial asymmetry and more depressive symptoms in men only. Likewise, men with greater FA also tended to report more anxiety, exhibit poorer impulse control, be less optimistic, feel inferior and anger more quickly compared to men with greater facial symmetry. These findings led the authors to suggest that facial asymmetry may be a more reliable signal of psychological factors in men than women citing differential evolutionarily-derived mating selection (Thornhill & Møller, 1997).

On the heels of the Shackelford and Larsen’s (1997) finding that high FA was associated with poor impulse control, Benderlioglu et al. (2004) investigated the relationship between FA and another behavioral trait, reactive aggression. In order to clarify that relationship, they exposed $n = 100$ ($51 \text{ M} / 49 \text{ F}; M_{\text{age}} = 20.1$) college students from the general population to experimental conditions designed to elicit aggressive reactions. FA of various morphologic features (e.g., digit
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length, palm height, wrist diameter) was measured. Results showed that males with high morphologic FA were more likely to display high aggression under a low provocation condition, whereas females with high FA were more prone to aggression under a high provocation condition. The authors concluded that these sexually dimorphic findings could likely be attributed to a variety of factors (differential arousal during conflict and responses to provocation). Nevertheless, the overall findings provide evidence that genetic and environmental insult during development (captured in morphologic FA) may adversely impact aspects of executive function and overt behavior that can be seen in the general population.

Relationships between dermatoglyphic measures and other mental disorders with putative neurodevelopmental origins (e.g., ADHD, schizophrenia spectrum) are also found in non-clinical samples. Burton et al., (2003) investigated ADHD symptoms in childhood in relation to facial FA (e.g., eye width, ear placement) to assess the relative contribution of genetic and environmental insults in ADHD emergence. Statistically significant trends were identified between an aggregated index of total FA and increases in “ADHDness” in males only. This not only points to a neurodevelopmental component of disorder pathophysiology but also suggests that females may be buffered against certain prenatal insults relative to males (for reviews see Walder, Ospina, Daly, Statucka, & Raparia, 2012; Walder, Yaffe, & Ehrlich, 2015). In another investigation, Daly et al. (2008) showed that a sample of high-endorsing schizotypes derived from a non-clinical population had significantly lower dermatoglyphic ridge counts compared to controls, for male subjects only. This lends support to the notion that the developmental insults associated with elevated risk for schizophrenia spectrum disorders (understood from a dimensional perspective) can be captured using dermatoglyphic measures. Both of these findings
further underscore the importance of sex differences in neurodevelopmental contribution to disorder pathophysiology.

4.2 Dermatoglyphic/FA Measures in Mixed Clinical and Non-Clinical Samples

In order to test the hypothesis that neural malfunction seen in depression may reflect developmental instability of the CNS, another investigation explored the relationship between FA and depressive symptoms in a mixed clinical and non-clinical sample (Martin et al., 1999). Subjects \( n = 102 \); 52M / 50F; \( M_{\text{age}} = 40.5 \) were recruited from the community who were not excluded on the basis of current or past DSM-IV Axis I disorders, thereby allowing for increased generalizability of results. A measure of FA was derived based on discrepancies between left and right (L – R asymmetry) morphological traits (i.e., digit length, wrist diameter and ear height). Depressive symptoms were measured using the revised BDI (BDI-R; Beck & Steer, 1984). Results showed a significant positive relationship between higher self-reported depressive symptoms and greater FA among males alone. Put differently, asymmetric men endorsed more depressive experiences relative to symmetric men, whereas there were no differences among women. Notably, this finding emerged in a mixed clinical and non-clinical sample, suggesting that FA may be a reliable marker of underlying processes regardless of disorder emergence. Further, the finding that FA was predictive of depressive symptoms in males (and not females) is consistent with other research and suggests unique etiological process for depression as a function of sex differences.

4.3 Dermatoglyphic/FA Measures in Clinical Samples
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A recent study explored the relationship between dermatoglyphic alterations and structural brain abnormalities in a sample of subjects with bipolar disorder (BD; Vonk et al., 2014). While BD is distinct from depression symptomatically and diagnostically, it nevertheless can be challenging to differentiate the two clinically (Wallace et al., 2016). Due to the high degree of overlap, there is increasing consideration of the existence of a mood spectrum between unipolar and bipolar depression (Cassano et al., 2004; Moreno et al., 2012; Strakowski, Fleck, & Maj, 2011), where MDD and BD reflect different stages of a similar disorder process. This theory is presented with a degree of caution, as imaging studies have shown unique neural pathophysiology (Cardoso De Almeida & Phillips, 2013) implicated in the two illness processes. Nevertheless, this study will be discussed, as the findings lend support to the value of dermatoglyphics in understanding neurodevelopmental disorder process.

Vonk et al. (2014) sampled 53 twin pairs concordant or discordant for BD and 51 matched healthy control twin pairs. Dermatoglyphic measures of various patterns were collected, including total finger ridge counts, FA, and palmar a-b ridge counts. Palmar a-b ridge counts refer to the number of ridges drawn between two triradii found on the palm approximately between digits 2 and 3 (Vonk et al., 2014). They are understood to develop over a wider range of the prenatal period and are therefore more susceptible to embryopathic influence throughout the second and third trimesters (Davis & Bracha, 1996), whereas digit dermatoglyphics are believed to reflect disruptions during the late first and early second trimesters. Brain volumetric data were acquired using structural magnetic resonance imaging (MRI). The authors found a negative correlation between a-b ridge counts and all measures of brain volume regardless of mental illness status, where higher counts were associated with decreased brain volume. In addition to
supporting the notion that dermatoglyphics and neurological development are partly mediated by similar genes, this finding specifically suggests that increased a-b ridge counts are related to stunted brain development. Analyses also showed that increased a-b ridge counts related to abnormal neural structures shared 70% of the covariance with risk for developing BD. This suggests that dermatoglyphics may indeed be indicative of both genetic and environmental factors adversely affecting neurodevelopment associated with increased risk of mental illness. A degree of caution is warranted though, as another investigation found that total finger ridge counts and a-b ridge counts were decreased in subjects with BD compared with healthy controls (Shrivastava et al., 2016). The discrepancies between these studies may be a function of differences in insult type and timing, as well as the dermatoglyphic measure used in these analyses. It has been proposed (Bracha, Torrey, Gottesman, Bigelow, & Cunniff, 1992) that while certain prenatal conditions (e.g., edema v. ischemia) may both adversely impact development, they may do so differentially. According to Babler (1978), the timing of extrinsic insult may also result in lower or higher ridge counts as a function of when the ridges differentiate relative to volar pad regression. For example, arch patterns typically have later differentiation relative to pad regression, while more complex patterns undergo ridge differentiation more proximally to pad regression. Therefore, higher frequency of arch patterns may reflect an earlier acquired insult that forestalled ridge differentiation. Finally, development of a-b ridge counts typically occurs over a more diffuse gestational period than digit dermatoglyphics (Golembo-Smith et al., 2012) such that this measure captures more variability.

In another investigation, Jelovac et al. (1998) examined dermatoglyphic abnormalities in a sample of subjects with BPD to investigate the potential role of prenatal influences in that
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psychiatric outcome. Results showed that males \( n = 33 \) and females \( n = 29 \) with BPD (ages 18-63 years) had significantly lower ridge counts compared with healthy controls. However, BPD ridge counts were significantly higher than a psychiatric control group of schizophrenic subjects. While this investigation possesses limitations (high likelihood of Type I error with no apparent statistical correction), it provides evidence that dermatoglyphic variables may discriminate between various psychiatric conditions (BPD and schizophrenia) and the general population.

Taken together, the findings across non-clinical, mixed, and clinical samples establish associations among dermatoglyphic measures and varied aspects of mental illness, including mood symptoms. FA may serve as a clearer dermatoglyphic indicator of aberrant neurodevelopment, as directional findings are generally consistent with this measure (greater FA related to greater symptom endorsement). Conversely, the direction of findings regarding ridge counts remains less clear, where counts may be either higher or lower in affected samples perhaps as a function of differences in type and timing of prenatal insult (Babler, 1978; Bracha et al., 1992; Green, Bracha, Satz, & Christenson, 1994). Moreover, FA measures offer the methodological advantage of minimizing noise through intraindividual comparisons.

5. Current Study

The current study aimed to expand the body of literature investigating the contribution of abnormal neurodevelopment to depressive disorder pathophysiology. This investigation included a sample of adolescents and young adults ages 18-21 years from the general population with varying degrees of depressive symptoms who did not meet diagnostic criteria for any DSM-IV-
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TR Axis I disorder. In keeping with the dimensional theory of depression that proposes shared underlying etiology across disorder severity, the current sample was drawn from the non-clinical population. This study differed from previous iterations in that dermatoglyphic measurements included various measures of both digit asymmetry (as opposed to asymmetry measures of other morphological features (e.g., facial features, digit ratios)) and ridge counts. The temporal overlap between epidermal ridge morphology and establishment of brain regions implicated in depression intimates that digit dermatoglyphic FA may be a more relevant measure (as opposed to other bodily symmetry) to assess markers of developmental instability associated with genetic and environmental insult. Both FA and ridge counts are understood to reflect deviations to ontogeny via developmental noise. However, the constructs vary slightly in that FA measures reflect reduced homeostatic buffering in relation to insult (Clarke & McKenzie, 1992) whereas ridge count abnormalities reflect deviations to genetically-mediated processes (Babler, 1978). Accordingly, they were both investigated separately in relation to depressive symptomatology.

Secondarily, this study examined the potential moderating effects of sex in the relationships among depressive symptoms and dermatoglyphic indices, consistent with the empirical support for differential sex effects in neurodevelopment.

Findings from this study may hold important theoretical and clinical implications. Relative to other mental health disorders (e.g., schizophrenia, ADHD), there is less emphasis on the importance of neurodevelopment in depression etiology (for discussion see Ansorge, Hen, & Gingrich, 2007). Although heterogeneity in illness precursors, onset, course, duration and treatment response supports the likelihood of multiple pathophysiologies in depression, there is compelling evidence to further investigate the role of neurodevelopment. The current study was
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part of an overarching project aimed at advancing the field’s understanding of the contribution of developmental processes to depression risk and pathophysiology. From a clinical perspective, findings showing a link between dermatoglyphics (one index of neurodevelopment) and depressive symptoms would hold potentially meaningful implications for disorder prevention from an individual and, possibly, societal perspective (via early screenings and prenatal programs for mothers at high risk). Identification of biomarkers, such as relatively altered patterns of dermatoglyphics, is not expected to be of great utility diagnostically at this point given the lack of sensitivity and specificity. However, better understanding the neurodevelopmental contribution to illness risk may aid early identification of individuals at-risk.

6. Specific Aims and Hypotheses

Aim 1: To indirectly assess the possible impact and timing of environmental stress and genetic factors during prenatal neurodevelopment on later experience of depressive symptoms among non-clinical adolescents and young adults in the general population. Specifically, we examined the relationships among dermatoglyphic FA measurements and self-reported depressive symptoms in a non-clinical sample of adolescents and young adults.

Aim 1 Hypothesis: Developmental instability refers to an organism’s decreased capacity to buffer against insult during ontogeny. Literature suggests that FA of dermatoglyphics (an indirect marker of early developmental instability) may be a sensitive measure of environmental perturbations during gestation, such as prenatal stress (King et al., 2009). Multiple other adverse prenatal conditions, including maternal poor health (Kieser & Groeneveld, 1993; Kieser, 1992; Livshits et al., 1988), exposure to parasites, pathogens and pollutants (Møller, 1996; Parsons,
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1990, 1992; Polak, 1997), and injurious living conditions (Harris & Nweeia, 1980; Parsons, 1990, 1992) are also captured by deviations in symmetry of bilateral morphological traits (e.g., digit ratio, facial features). As bilateral fingerprints, in particular, develop concurrently with certain neuroanatomical regions (e.g., limbic structures), deviations in fingerprint FA may reflect the impact of such insults on neurodevelopment. Moreover, increased FA may reflect decreased ability to employ homeostatic mechanisms that buffer against insults (developmental stability) and thereby increase vulnerability to adverse outcomes. Support for this can be inferred from evidence that prenatal insults are also implicated in child emotional and psychological outcomes such as increased risk for offspring depression (O’Connor et al., 2003; Van Den Bergh et al., 2005), which have known neuroanatomical correlates (i.e., limbic and HPA axis abnormalities).

Prior research demonstrates that FA may capture the timing of such insults. For example, King et al. (2009) showed that women who experienced a high level of stress during a particular gestational window (weeks 14-22) produced offspring with significantly greater digit FA than offspring of mothers who experienced stress during other periods of gestation.

Consistent with prior research showing significant positive associations of FA of morphological traits with depressive symptom endorsement in mixed clinical and non-clinical adult and adolescent samples (Martin et al., 1999; Shackelford & Larsen, 1997), we hypothesized that dermatoglyphic FA measurements would positively predict endorsement of subjective depressive symptoms in a non-clinical sample of adolescents and young adults.
Aim 2: In order to further establish the impact of possible prenatal environmental insults to genetically mediated development using another proxy index of developmental disturbance, we examined the relationship between ridge counts (total and absolute) and self-reported depressive symptoms in a non-clinical sample of adolescents and young adults.

Aim 2 Hypothesis: Unusual dermatoglyphic ridge counts (including high frequency of arches) are understood to reflect deviations from genetically mediated common developmental pathways that dictate aspects of pad/ridge development (Babler, 1978). Consistent with literature suggesting that total finger ridge counts reflect the rate of fetal growth (Babler, 1978; Cohen-Bendahan et al., 2005; Newell-Morris et al., 1989), lower ridge counts may reflect developmental stress during the primary period of ridge proliferation (prenatal weeks 10-17), which is also a crucial period for CNS structure development (limbic regions) implicated in depression (Ge et al., 2015; Jacob et al., 2011). As another proxy for rate of growth, absolute finger ridge counts also provide information about the size and intensity of fingerprint patterns (Schaumann & Alter, 1976). The literature is mixed regarding the direction of findings using these measures. Therefore, we hypothesized a non-directional relationship where ridge count measures (total and absolute) would predict depressive symptoms.

Aim 3: To investigate potential sex differences in the relationships of dermatoglyphic measures (FA and ridge count) with depressive symptom endorsement among non-clinical adolescents and young adults from the general population. This secondary, exploratory aim would potentially shed light on whether one aspect of neurodevelopment is differentially related to later emergence
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of depressive symptoms as a function of sex in a non-clinical sample of adolescents and young adults.

**Aim 3 Hypothesis:** Based on past findings of sex differences in depressive symptomatology as a function of dermatoglyphic indices (Martin et al., 1999; Shackelford & Larsen, 1997), we expected that sex would moderate the predictive relationships of dermatoglyphic measures with self-reported depressive symptoms. Despite the frequent finding in the literature suggesting that dermatoglyphics are a more sensitive measure of neurodevelopmental disturbance among males than females (Burton et al., 2003; Daly et al., 2008; Martin et al., 1999; Shackelford & Larsen, 1997), the underlying mechanisms responsible for these sex differences are not entirely clear. Therefore, we proposed a non-directional hypothesis with respect to hypothesized sex differences.
CHAPTER II.

METHOD

The current study was conducted as part of an overarching project (Walder, PI; e.g., Walder et al. 2014) examining environmental and biological predictors of depression risk among non-clinical adolescents and young adults from the general population. The current study involved a subset of the data collected as part of this larger protocol. The following chapter describes the subsample, select study procedures, select measures, and statistical analyses relevant to the current study hypotheses.

1. Sample

Participants included male and female individuals 18-21 years of age, recruited from an urban, public university and the broader community. This age range captures the developmental window that precedes typical age of onset of depressive disorders (Kessler et al., 2003) and may therefore illuminate aspects of pathophysiology prior to illness emergence (Hagan et al., 2015). This sample was intended to reflect the non-clinical general population of adolescents and young adults with varying degrees of depressive symptoms.

2. Study Procedures

The current study was conducted as part of an overarching project (Walder, PI; e.g., Walder et al., 2014) examining environmental and biological predictors of depression risk among non-clinical adolescents and young adults from the general population. The current study involved a subset of the data collected as part of this larger protocol. The study protocol received approval from the
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Human Subjects Institutional Review Board (IRB) in accordance with Brooklyn College/CUNY IRB policies.

2.1 Recruitment

Participants were recruited via self-referral. IRB-approved advertising materials (e.g., flyers, brochures) providing a brief study description, general inclusion criteria, and contact information were posted or presented in approved locations.

2.2 Inclusion/Exclusion Criteria

Prior to enrollment in the study, participants were screened over the phone to assess inclusion and exclusion criteria for purposes of the umbrella study. Here are described inclusion and exclusion criteria used for the subset of participants included in the current study (as part of the larger umbrella study).

2.2.1 Inclusion Criteria. (1) Age 18-21 years; (2) English speaking.

2.2.2 Exclusion Criteria. (1) Age > 21 or < 18 years; (2) Indication of possible presence of DSM-IV-TR psychiatric condition; (3) Past or current psychiatric illness; (4) Past or current treatment with medication or psychotherapy for emotional or psychological reasons; (5) Major/serious medical or neurological illness; (6) History of loss of consciousness; (7) Contraindications for MRI (e.g., metal implants, pregnancy, claustrophobia).
2.3 In-Office Visit

Data collection was completed in a laboratory setting at Brooklyn College. Participants provided informed consent in accordance with Brooklyn College IRB policies and procedures; they were reminded that participation was voluntary and that they were free to withdraw at any time without penalty. Demographic information was collected from participants (age, sex, race, and family household income). Select measures from the broader protocol (not described here) were used for purposes of the current study. Specifically, in order to meet inclusion/exclusion criteria, participants were interviewed by one of three trained doctoral-level graduate students using the Structured Clinical Interview for DSM-IV-TR Axis I disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1997, 2002) to assess for presence of past or current DSM-IV-TR Axis I disorders. Consensus diagnosis was achieved across two to three raters for a random sample of administered SCIDs \( n = 17 \) during consensus meetings. Inter-rater accuracy at the item-level for SCID-I administration was approximately 99.3\%, and consensus diagnosis was established at a rate of 100\% across two to three diagnostic raters for a random sample of \( n = 17 \) subjects. Participants who met criteria for any Axis I disorder were not included in the current investigation.

Participants were asked to complete a self-report mood questionnaire, the Beck Depression Inventory-II (BDI-II; Beck, Steer & Brown 1996). To derive dermatoglyphic measures, participants provided fingerprints (detailed further below). At the conclusion of study participation, all participants were debriefed about the study purposes and compensated for transportation and time/effort. The portion of the overarching protocol relevant to the proposed study lasted approximately 1 hour and 20 minutes.
3. Measures

3.1 Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First et al., 1997, 2002)

The SCID-I was developed by First, Spitzer, Gibbon & Williams in 1997 as a semi-structured assessment instrument for diagnosing past or current mental disorders based on DSM-IV criteria and later revised (First et al., 2002) to be consistent with DSM-IV-TR specifications. The instrument, intended for administrative use by a trained mental health professional, includes a series of modules designed to assess for mood, psychotic, substance use, anxiety, somatoform, eating, and adjustment disorders. Within each module, the assessor asks participants about individual criteria comprising particular disorders. Based on subject endorsement, each criterion is scored on a scale from 1 (absent) to 2 (sub-clinical experience) to 3 (clinically significant). Diagnoses are made on the basis of meeting the minimum number of criteria as detailed in the DSM-IV-TR (First et al., 2002).

The SCID-I is both reliable (Lobbestael, Leurgans, & Arntz, 2011; Zanarini et al., 2000; Zanarini & Frankenburg, 2001) and valid (Drill, Nakash, DeFife, & Westen, 2015). In one study, inter-rater reliability of SCID-I fluctuated between the fair to excellent ranges (kappa values between 0.61 to 0.83) across different diagnoses (Lobbestael et al., 2011). This is generally commensurate with findings from other investigations (Zanarini et al., 2000; Zanarini & Frankenburg, 2001). In terms of validity, Drill et al. (2015) found significant correlations between SCID-I and patient ratings across multiple adaptive functioning variables. Further, they found the SCID-I had a high “hit rate” in that it correctly matched interviewer- and patient-rated diagnoses. Measures of sensitivity and specificity were also generally high in that same study; interviewers correctly
identified problems that patients endorsed as well as problems that they did not directly endorse. In sum, the SCID-I has high inter-rater reliability and validly categorizes diagnoses with a sufficient degree of sensitivity and specificity.

3.2 Beck Depression Inventory-II (BDI-II; Beck et al., 1996)

The BDI-II is a widely used and well-validated measure that assesses depressive symptoms. The 21-item measure asks users to rate their experience of various depressive symptoms (e.g., depressed mood, loss of interest, changes in appetite, sleep) over the past two weeks on a scale from 0 (absent) to 3 (present, severe, and functionally impactful), such that higher scores reflect greater depressive symptom endorsement with a highest possible score of 63. The BDI-II reliably and validly measures depressive symptom endorsement in both clinical and non-clinical samples (Wang & Gorenstein, 2013), as well as among adults and adolescents (Steer & Clark, 1997; Whisman, Perez, & Ramel, 2000).

The BDI-II yields multiple indices that capture different aspects of depressive symptomatology. Summing all the responses yields a total score (BDI-Total) that ranges from 0 to 63. While this continuous scale reflects varying endorsement of depressive symptoms, the authors established clinical cut-offs (Beck et al., 1996). Scores between 0-13 are considered ‘minimal’, 14-19 comprise the ‘mild’ range, 20-28 fall in the ‘moderate’ range, and 29-63 reflect ‘severe’ depressive symptoms. Similar ranges are also established in adolescent populations (Steer, Kumar, Ranieri, & Beck, 1998; Uslu, Kapci, Oncu, Ugurlu, & Turkecapar, 2008; Whisman et al., 2000).
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As one of the most popular measures used to assess depression worldwide (McDowell, 2006), there is substantial evidence of the BDI-II’s psychometric properties (see Wang & Gorenstein, 2013). In a review of 178 studies using the BDI-II across clinical and non-clinical samples of adults and adolescents, Wang & Gorenstein (2013) found an average $\alpha$ coefficient of 0.9, suggesting high reliability. The overlap between scores on the BDI-II and other scales used to assess depression ranged from 0.66 to 0.86 across studies, showing strong convergent validity. Likewise, the authors found medium ($r = 0.37$) to large ($r = 0.83$) convergence between the BDI-II and scales tapping highly comorbid mood states (e.g., anxiety), as well as scales of general psychopathology. Investigations of discriminant validity showed relatively lower correlations ($r < 0.4$) with items measuring alcohol and drug use and chronic pain. The authors found evidence suggesting that the BDI-II can be used to effectively screen for probable cases of MDD, as the measure had a sensitivity of $< 0.70$ aggregated across studies. The content validity is also adequate in that the BDI-II continuously showed high specificity to the DSM-IV criteria of MDD (Wang & Gorenstein, 2013).

3.3 Dermatoglyphic Measures

Dermatoglyphic abnormalities are understood to reflect, in part, the impact, timing, and nature of prenatal insult (Cummins & Midlo, 1961). The current investigation included fingerprint dermatoglyphic measures only, as opposed to other morphological features (e.g., facial symmetry, digit ratios). Digit dermatoglyphics develop during gestational weeks 10-24 (Babler, 1991) in conjunction with neuroanatomical structures implicated in depression (i.e., limbic and endocrine structures) (Challis, Matthews, Gibb, & Lye, 2000; Ge et al., 2015; Kostović et al., 1993; Matthews, 2002; Qiu et al., 2013, 2015). Thus, these measurements may hold the greatest
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amount of available information (relative to palm or sole ridge counts, which develop later in gestation (Cummins & Midlo, 1961)) regarding neurodevelopment associated with depression risk. Digit dermatoglyphics are also uniquely relevant to the investigation of prenatal insult. Unlike other conventional neurodevelopmental markers (e.g., brain ventricle size), dermatoglyphics are not subject to postnatal insult to the brain or body (e.g., via psychotropic medication use, drug or alcohol abuse, aging) (Bracha et al., 1992) and remain relatively stable throughout development (Babler, 1978; Cummis & Midlo, 1961).

To assess dermatoglyphics, participants rolled each finger (bilaterally) on a Porta Palm Print Kit® colorless inkpad and then onto specialized paper to produce a clear print. Data ascertainment was conducted by a trained doctoral-level graduate student in keeping with the procedure proposed by Holt (1968). Ridge counts were determined by counting ridges that intersected a straight line drawn between the triradius (i.e., outermost meeting point of three ridges) and the core (i.e., center of the fingerprint pattern) for each finger on both hands (see Figure 2). The ridges comprising the triradius and the core were not included in the total count. In patterns with a single triradius (i.e., loops), ridges crossing the single line were counted as both total and absolute finger ridge counts. In patterns with multiple triradii (i.e., whorls), the number of ridges crossing the longest line were included in the total finger ridge count and the sum of all the ridges were included in the absolute finger ridge count. The following dermatoglyphic indices were computed (final terms are bolded):

3.3.1 Fluctuating Asymmetry (FA). Dermatoglyphic FA serves as a proxy for developmental instability, or susceptibility to developmental disruptions from environmental and
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genetic prenatal insults (Kowner, 2001). Two methodologies for calculating FA were used, both of which contain two indices derived from total and absolute finger ridge counts. There does not appear to be an objective difference between these methodologies noted in the literature and both appear with similar frequency.

**Total Fluctuating Asymmetry:** FA-Total was computed by measuring discrepancy in ridge counts between homologous fingers. Given that hypotheses did not specify the direction of asymmetry but rather its degree, measures included the absolute value of the difference scores (right side ridges minus left side ridges) for the total finger ridge counts (TFRC) from each digit (King et al., 2009; van Oel et al., 2001). These absolute values were summed across digits 1 through 3, as legible prints were not available for every digit for all participants; using digits 1 through 3 allowed for maximal data to be preserved while increasing power. The same procedure was repeated using absolute values of absolute finger ridge counts (AFRC) from each digit and summed across digits 1 through 3 to compute FA-Absolute (FA-Abs).

**Average Fluctuating Asymmetry:** FA-Average was computed by taking the quotient of the absolute value of the TFRC right-left difference of each finger ridge divided by the respective sum of right and left for each finger \([(R – L) / (R + L)]\) (Goleumbo-Smith et al., 2012; Green et al., 1994). In order to compute FA-Average indices, an intermediate term was first computed \([(R1 – L1) / (R1 + L1)]\) for each digit. To compute the final FA-Average terms, each of these intermediate computations was averaged across all available fingers. A constant value of .0001 was added to each intermediate term, such that the term was computed as \([(R1 + .0001) – (L1 + .0001)) / ((R1 + .0001) + (L1+ .0001))\). By eliminating the possibility of a ‘0’
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denominator, this allowed for all the data to be preserved in computing these indices. There were no significant group differences (measured via t-tests) between the original values and the adjusted values after the constant was added. This demonstrated that adding a constant did not significantly alter the data while allowing for inclusion of all relevant data and greater power. A ‘0’ value indicates a finger ridge count that equals zero, which was relevant information given hypotheses (for further discussion on calculating FA, see Palmer & Strobeck, 1986). This procedure was conducted for digits 1 through 3 in order to increase power due to variable print legibility. As above, the same procedure was repeated using AFRCs to compute FA-Absolute-Average (FA-Abs-Average).

3.3.2 Finger Ridge Counts. Abnormalities in finger ridge counts (e.g., higher frequency of low ridge count patterns) are understood to reflect deviations from genetically-mediated developmental processes (Babler, 1978, 1990, 1991).

Total Finger Ridge Counts: Total finger ridge count (TFRC) expresses the size of the fingerprint pattern (Cummins & Midlo, 1961) and may be sensitive to the nature of some prenatal insults (e.g., edema versus ischemia) (Green et al., 1994). TFRCs were measured by finding the sum of finger ridges crossing a line drawn from the triradius to the core (van Oel et al., 2001). Arch patterns lack triradii and had a ridge count of zero. In fingerprint patterns with multiple triradii, only the larger ridge count was used in calculating TFRCs. This measurement was conducted for digits 1 through 3 alone in order to increase power.
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Absolute Finger Ridge Counts: Absolute finger ridge count (AFRC) is an alternative measure of ridge counts that take into account fingerprint patterns with multiple triradii. Multiple triradii reflect the size and intensity of fingerprint patterns and are somewhat more sensitive to non-genetic influences, such as environmental prenatal insults (Schaumann & Alter, 1976). The same procedure as that used in TFRC was applied, except the calculation for AFRC included all ridge counts for fingerprints with multiple triradii (van Oel et al., 2001) from digits 1 through 3. Thus, the AFRCs were expected to be larger than TFRCs.

4. Statistical Analyses

A power analysis was conducted to determine sufficient sample size using GPower 3.1 (Erdfelder, Faul, & Buchner, 1996). With a medium effect size of $R^2 = .3$, $\alpha = .05$, and the number of tested predictors = 3 (for the three categories of digit dermatoglyphic measures: FA using TFRC, FA using AFRC, and finger ridge counts), a sample size of 49 was required to yield sufficient power ($1 - \beta = 0.95$) for one-tailed hypotheses. The sample size increased to 56 subjects for two-tailed hypotheses using the same metrics.

Analyses were performed using IBM SPSS (24). Descriptive statistics were generated for the sample. Outliers on all demographic, independent, and dependent variables, defined by three standard deviations above or below the mean, were excluded from all analyses such that total sample size was $n = 53$ subjects. Normality was assessed using the Kolmogorov-Smirnov analysis ($p < .05$ considered non-normal). Any variables that were not normally distributed were normalized using a Box-Cox transformation. Normality was assessed again using the criteria of skewness and kurtosis ($< 0.5$ and $> -0.5$) after transformations were conducted. Pearson’s
bivariate correlations, point biserial correlations, and between group analysis of variance (ANOVA) computations were used to assess for potential covariates among continuous and categorical demographic characteristics, respectively (age, sex, race, household income), in relation to all variables of interest (BDI-Total, dermatoglyphic measures; see Table 1 for all variables). Inter-rater reliability on SCID-I administration was assessed by calculating inter-rater accuracy at the item level.

All normalized independent and dependent variables (shown in Table 1) were transformed into z-scores to create standardized regression coefficients for analysis. ANOVAs for the overall regression models were interpreted using two-tailed tests, with $\alpha$ set at .05. All multiple regression analyses included six dermatoglyphic variables (see 4.1 and 4.2). Therefore, Bonferroni correction using $\alpha$ set at .008 was applied to all tests examining main effects and interaction effects, to adjust for multiple comparisons.

4.1 Hypotheses 1 & 2 Analyses

To assess the extent to which FA measures (FA-Total, FA-Average, FA-Abs, FA-Abs-Average; Hypothesis 1) and finger ridge count measures (TFRC, AFRC; Hypothesis 2) predicted BDI-Total, multiple regression analyses were conducted using the enter method. All six FA variables were entered into a single block to discern relative predictive contribution. Results pertaining to FA measures were interpreted using one-tailed tests, given directional hypotheses; whereas results pertaining to finger ridge count measures were interpreted using two-tailed tests, given non-directional hypotheses. For the applied Bonferroni correction, $\alpha$ was set at .008 for all analyses. Demographic covariates (i.e., demographic variables that significantly correlated with
FA and/or BDI-Total at the <.05 level) were entered into Block 1 of the model. All independent variables were entered into Block 2. The BDI-Total score was entered as the dependent variable. Analyses of $R^2$ change (two-tailed) and the overall regression value ($F$-statistic) for main effects were interpreted with $\alpha$ set at .05.

4.2 Hypothesis 3 Analyses

To examine the potential role of sex in moderating the relationships of dermatoglyphic measures with BDI-Total scores, a second multiple regression analysis was conducted using the enter method. This analysis was conducted separately given the exploratory nature of this hypothesis. Covariates were included in Block 1 of the model. All variables assessed for main effects (i.e., all FA and finger ridge count measures), as well as sex, were entered in Block 2. Interaction terms for each individual dermatoglyphic measure by sex, as well as all FA and finger ridge count measures and sex, were entered into Block 3. Including all predictor variables in a single model reduced likelihood of Type I error while allowing for assessment of overall $R^2$ change between the blocks, as well as relative contributions of each interaction term. For the applied Bonferroni correction, $\alpha$ was set at .008. All tests of sex effects were two-tailed given the non-directional nature of the hypotheses. Analyses of $R^2$ change (two-tailed) and the overall regression value ($F$-statistic) for main effects were interpreted with $\alpha$ set at .05.
CHAPTER III.

RESULTS

1. Demographics and Descriptive Statistics

All independent and dependent variables are shown in Table 1. Demographic characteristics for the entire sample are shown in Table 2 (\(n = 53; 22 \text{ M} / 31 \text{ F}; M_{age} = 20.04, SD_{age} = 1.05\)). Given sex-specific hypothesis 3, demographic data split by sex is also shown in Table 2.

Table 1. A list of all independent and dependent variable indices along with a summary of the underlying construct each index reflects, the particular measure(s) used to capture each construct, and the variable name that corresponds to each measure.

<table>
<thead>
<tr>
<th>Index</th>
<th>Underlying Construct</th>
<th>Measure</th>
<th>Variable Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressive Symptoms</td>
<td>State depressive symptomatology</td>
<td>BDI-II</td>
<td>BDI-Total</td>
</tr>
<tr>
<td>Finger Ridge Counts (FRC)</td>
<td>Deviations to genetically-mediated developmental processes. They may be sensitive to the nature and timing of prenatal insults.</td>
<td>Total Finger Ridge Counts</td>
<td>TFRC</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Absolute Finger Ridge Counts</td>
<td>AFRC</td>
</tr>
<tr>
<td>FA (with Total Finger Ridge Counts)</td>
<td>Reduced homeostatic buffering in relation to insult.</td>
<td>FA Total</td>
<td>FA-Total</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FA Average</td>
<td>FA-Average</td>
</tr>
<tr>
<td>FA (with Absolute Finger Ridge Counts)</td>
<td>Reduced homeostatic buffering in relation to insult that may be more sensitive to non-genetic influences.</td>
<td>FA Absolute</td>
<td>FA-Abs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FA Absolute Average</td>
<td>FA-Abs-Average</td>
</tr>
</tbody>
</table>

FA = Fluctuating Asymmetry; BDI-II = Beck Depression Inventory-II

Table 2. Demographic characteristics for the total sample as well as separately for each sex.

<table>
<thead>
<tr>
<th>Sex Distribution:</th>
<th>Total Sample</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>(n = 22) (41.5%)</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Female</td>
<td>(n = 31) (58.5%)</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Total</td>
<td>(n = 53)</td>
<td>--</td>
<td>--</td>
</tr>
</tbody>
</table>
**DERMATOGlyphICS AND DEPRESSIVE SYMPTOMS**

<table>
<thead>
<tr>
<th>Age (years):</th>
<th>18-21</th>
<th>18-21</th>
<th>18-21</th>
</tr>
</thead>
<tbody>
<tr>
<td>Range</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>20.04</td>
<td>19.84</td>
<td>20.18</td>
</tr>
<tr>
<td>SD</td>
<td>1.05</td>
<td>1.15</td>
<td>0.96</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Race/Ethnicity:</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>White/Caucasian</td>
<td>$n = 11 (20.8%)$</td>
<td>$n = 3$</td>
<td>$n = 8$</td>
</tr>
<tr>
<td>Black/African American</td>
<td>$n = 20 (37.7%)$</td>
<td>$n = 8$</td>
<td>$n = 12$</td>
</tr>
<tr>
<td>Asian</td>
<td>$n = 11 (20.8%)$</td>
<td>$n = 5$</td>
<td>$n = 6$</td>
</tr>
<tr>
<td>Hispanic</td>
<td>$n = 4 (7.6%)$</td>
<td>$n = 2$</td>
<td>$n = 2$</td>
</tr>
<tr>
<td>Middle Eastern</td>
<td>$n = 2 (3.8%)$</td>
<td>$n = 1$</td>
<td>$n = 1$</td>
</tr>
<tr>
<td>Other / Mixed</td>
<td>$n = 5 (9.4%)$</td>
<td>$n = 3$</td>
<td>$n = 2$</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Household Income:</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;$10,000</td>
<td>$n = 5 (9.4%)$</td>
<td>$n = 2$</td>
<td>$n = 3$</td>
</tr>
<tr>
<td>$10,000 – 24,000</td>
<td>$n = 9 (17.0%)$</td>
<td>$n = 4$</td>
<td>$n = 5$</td>
</tr>
<tr>
<td>$25,000 – 39,000</td>
<td>$n = 11 (20.8%)$</td>
<td>$n = 4$</td>
<td>$n = 7$</td>
</tr>
<tr>
<td>$40,000 – 69,000</td>
<td>$n = 8 (15.1%)$</td>
<td>$n = 4$</td>
<td>$n = 4$</td>
</tr>
<tr>
<td>$70,000 – 100,000</td>
<td>$n = 10 (18.9%)$</td>
<td>$n = 4$</td>
<td>$n = 6$</td>
</tr>
<tr>
<td>&gt;$100,000</td>
<td>$n = 9 (17.05%)$</td>
<td>$n = 3$</td>
<td>$n = 6$</td>
</tr>
<tr>
<td>Missing</td>
<td>$n = 1 (1.9%)$</td>
<td>$n = 1$</td>
<td>$n = 0$</td>
</tr>
<tr>
<td>Median</td>
<td>$40,000-69,999$</td>
<td>--</td>
<td>--</td>
</tr>
</tbody>
</table>

Complete data for dermatoglyphic indices derived from TFRCs were available for 42 subjects; complete data for AFRC were available for 38 subjects. We excluded from analyses subjects for whom prints were not legible.
Dermatoglyphics and Depressive Symptoms

Descriptive statistics for symptoms and dermatoglyphic measures for the current sample are shown in Table 3. There were no outliers on these measures as defined by mean ± 3 SDs. BDI-Total, FA-Total, FA-Average, and FA-Abs-Average were not normally distributed, though normality (as measured by skewness and kurtosis > -0.5 and < 0.5) was achieved after Box-Cox transformation. FA-Abs, TFRC, and AFRC were normally distributed.

Table 3. Descriptive characteristics for measures of symptoms and dermatoglyphics.

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Depressive Symptoms</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Total Symptoms (BDI-Total)</td>
<td>53</td>
<td>8.21</td>
<td>±7.87</td>
</tr>
<tr>
<td><strong>Dermatoglyphic Indices</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Fluctuating Asymmetry Total (FA-Total)</td>
<td>42</td>
<td>9.07</td>
<td>±5.99</td>
</tr>
<tr>
<td>*Fluctuating Asymmetry Average (FA-Average)</td>
<td>42</td>
<td>0.19</td>
<td>±0.17</td>
</tr>
<tr>
<td>Absolute Fluctuating Asymmetry Total (FA-Abs)</td>
<td>38</td>
<td>13.16</td>
<td>±9.07</td>
</tr>
<tr>
<td>*Absolute Fluctuating Asymmetry Average (FA-Abs-Average)</td>
<td>38</td>
<td>0.22</td>
<td>±0.17</td>
</tr>
<tr>
<td>Total Finger Ridge Count (TFRC)</td>
<td>42</td>
<td>68.50</td>
<td>±32.72</td>
</tr>
<tr>
<td>Absolute Finger Ridge Counter (AFRC)</td>
<td>38</td>
<td>90.37</td>
<td>±57.61</td>
</tr>
</tbody>
</table>

* Adjusted using Box-Cox transformation to achieve normality of distribution.

As shown in Table 4, BDI-Total scores ranged from 0 to 30, with the majority of scores concentrated below a score of 7. Nearly one quarter of the sample (22.6%) endorsed a score of zero. The distribution of BDI-Total scores in the current sample fell into the following clinical categories: Minimal (0-13): 75.5%; Mild (14-19): 17.0%; Moderate (20-28): 3.8%; Severe (29-63): 3.8%.
Table 4. *Frequency distribution for BDI-Total scores organized by clinical categories depicting relative and cumulative percentages.*

<table>
<thead>
<tr>
<th>Clinical Category</th>
<th>BDI-Total Score</th>
<th>Frequency</th>
<th>Relative %</th>
<th>Cumulative %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimal</td>
<td>0</td>
<td>12</td>
<td>22.6</td>
<td>22.6</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>3</td>
<td>5.7</td>
<td>28.3</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1</td>
<td>1.9</td>
<td>30.2</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>2</td>
<td>3.8</td>
<td>34.0</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>3</td>
<td>5.7</td>
<td>39.6</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>3</td>
<td>5.7</td>
<td>45.3</td>
</tr>
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<td></td>
<td>6</td>
<td>3</td>
<td>5.7</td>
<td>50.9</td>
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<td>7</td>
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<td>1.9</td>
<td>52.8</td>
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<td></td>
<td>8</td>
<td>2</td>
<td>3.8</td>
<td>56.6</td>
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<td></td>
<td>9</td>
<td>2</td>
<td>3.8</td>
<td>60.4</td>
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<td></td>
<td>10</td>
<td>1</td>
<td>1.9</td>
<td>62.3</td>
</tr>
<tr>
<td></td>
<td>11</td>
<td>6</td>
<td>11.3</td>
<td>73.6</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>1</td>
<td>1.9</td>
<td>75.5</td>
</tr>
<tr>
<td>Mild</td>
<td>14</td>
<td>2</td>
<td>3.8</td>
<td>79.2</td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>2</td>
<td>3.8</td>
<td>83.0</td>
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<td>3</td>
<td>5.7</td>
<td>88.7</td>
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<td>90.6</td>
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<td>18</td>
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<td>1.9</td>
<td>92.5</td>
</tr>
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<td>Moderate</td>
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<td>1.9</td>
<td>94.3</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>1</td>
<td>1.9</td>
<td>96.2</td>
</tr>
<tr>
<td>Severe</td>
<td>30</td>
<td>2</td>
<td>3.8</td>
<td>100.0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>n = 53</strong></td>
<td></td>
<td><strong>100.0</strong></td>
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</tr>
</tbody>
</table>

In terms of demographic covariates, the normalized BDI-Total score was significantly negatively associated with age and household income \( (r = -.36, p = .01; r = -.33, p = .02, \text{ respectively}) \), as shown in Table 5. Accordingly, age and household income were included as covariates in all analyses that included BDI-Total. No other demographic variables were significantly associated with BDI- Total. Demographic variables were not significantly associated with any dermatoglyphic indices. There were no group differences among ethnic groups across any independent or dependent variables.
Table 5. Correlation matrix of all demographic variables with all dependent and independent variables to assess for covariates.

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Sex</th>
<th>Ethnicity</th>
<th>SES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>.16</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>.25</td>
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<td></td>
<td></td>
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<tr>
<td>Ethnicity</td>
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<td>.01</td>
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<td>.17</td>
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<tr>
<td></td>
<td>.97</td>
<td>.04</td>
<td></td>
<td>.23</td>
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<td></td>
<td>.58</td>
<td>.79</td>
<td>.10</td>
<td>.48</td>
</tr>
<tr>
<td>SES</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>.08</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>.58</td>
<td></td>
<td>.04</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>.10</td>
<td>.48</td>
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<tr>
<td>BDI-T</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>-.36</td>
<td></td>
<td>.08</td>
<td>-.33</td>
</tr>
<tr>
<td></td>
<td>.01</td>
<td>-.12</td>
<td></td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td></td>
<td>.41</td>
<td>.59</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.02*</td>
</tr>
<tr>
<td>FA-Total</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>-.22</td>
<td></td>
<td>.26</td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td>.16</td>
<td></td>
<td>.06</td>
<td>.90</td>
</tr>
<tr>
<td>FA-Average</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>-.13</td>
<td></td>
<td>.17</td>
<td>-.07</td>
</tr>
<tr>
<td></td>
<td>.40</td>
<td>-.14</td>
<td></td>
<td>.07</td>
</tr>
<tr>
<td></td>
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<td>.39</td>
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<td>.66</td>
</tr>
<tr>
<td>FA-Abs-Total</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>-.15</td>
<td></td>
<td>.03</td>
<td>-.06</td>
</tr>
<tr>
<td></td>
<td>.36</td>
<td>-.10</td>
<td></td>
<td>.72</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.72</td>
</tr>
<tr>
<td>FA-Abs-Average</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>-.18</td>
<td></td>
<td>.07</td>
<td>-.14</td>
</tr>
<tr>
<td></td>
<td>.28</td>
<td>-.07</td>
<td></td>
<td>.39</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.39</td>
</tr>
<tr>
<td>TFRC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>.15</td>
<td></td>
<td>.02</td>
<td>-.06</td>
</tr>
<tr>
<td></td>
<td>.34</td>
<td>.16</td>
<td></td>
<td>.06</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.69</td>
</tr>
<tr>
<td>AFRC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>.28</td>
<td></td>
<td>.08</td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td>.09</td>
<td>.17</td>
<td></td>
<td>.91</td>
</tr>
</tbody>
</table>

* Significant finding (p < .05); all tests are two-tailed

2. Dermatoglyphics in Relation to Depressive Symptoms

Correlations among all independent and dependent variables are shown in Table 6. Several of the dermatoglyphic measures were significantly related. Multicollinearity was examined by assessing for a variance inflation factor (VIF) of greater than 10 (Alin, 2010). Using this threshold, all dermatoglyphic variables were multicollinear. See Table 7 for VIF figures for all assessed variables.
Table 6. Correlation matrix of all independent and dependent variables.

<table>
<thead>
<tr>
<th></th>
<th>BDI-Total</th>
<th>FA-Total</th>
<th>FA-Average</th>
<th>FA-Abs-Total</th>
<th>FA-Abs-Average</th>
<th>TFRC</th>
</tr>
</thead>
<tbody>
<tr>
<td>FA-Total</td>
<td>$r = -.00$</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>FA-Average</td>
<td>$r = -.03$</td>
<td>$r = .55$</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>FA-Abs-Total</td>
<td>$r = .05$</td>
<td>$r = .73$</td>
<td>$r = .22$</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>FA-Abs-Average</td>
<td>$r = .10$</td>
<td>$r = .54$</td>
<td>$r = .97$</td>
<td>$r = .38$</td>
<td>$p = .02^*$</td>
<td>-</td>
</tr>
<tr>
<td>TFRC</td>
<td>$r = .02$</td>
<td>$r = .16$</td>
<td>$r = -.50$</td>
<td>$r = .41$</td>
<td>$r = -.42$</td>
<td>$p = .01^*$</td>
</tr>
<tr>
<td>AFRC</td>
<td>$r = -.26$</td>
<td>$r = .13$</td>
<td>$r = -.44$</td>
<td>$r = .34$</td>
<td>$r = -.41$</td>
<td>$r = .94$</td>
</tr>
</tbody>
</table>

* Significant finding ($p < .05$); all tests are two-tailed

Table 7. Variance inflation factors (VIF) for covariates and dermatoglyphic variables

<table>
<thead>
<tr>
<th></th>
<th>VIF</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE</td>
<td>1.46</td>
</tr>
<tr>
<td>SES</td>
<td>1.25</td>
</tr>
<tr>
<td>FA-Total</td>
<td>13.87</td>
</tr>
<tr>
<td>FA-Average</td>
<td>47.50</td>
</tr>
<tr>
<td>FA-Abs-Total</td>
<td>13.75</td>
</tr>
<tr>
<td>FA-Abs-Average</td>
<td>42.01</td>
</tr>
<tr>
<td>TFRC</td>
<td>17.32</td>
</tr>
<tr>
<td>AFRC</td>
<td>19.59</td>
</tr>
</tbody>
</table>

Demographic covariates (age, household income) significantly predicted changes in BDI-Total scores ($F(2, 32) = 6.70, p = .004$). Adding the main effects (all dermatoglyphic variables) into the multiple regression model also significantly predicted BDI-Total ($F(8, 26) = 3.28, p = .01$), although it did not add more predictive variance to the model beyond that of demographic
DERMATOGLYPHICS AND DEPRESSIVE SYMPTOMS

covariates alone \( F_{\text{Change}}(6, 26) = 1.81, p = 0.14 \). Results for the overall regression model are shown in Table 8.

Table 8. *Multiple regression values for R, R^2 change, overall F statistics, and F change statistics for covariates and dermatoglyphic main effects predicting BDI-Total*

<table>
<thead>
<tr>
<th>Block 1</th>
<th>( R )</th>
<th>( R^2 ) Adjusted</th>
<th>Standard Error of Estimate</th>
<th>( F )</th>
<th>( df_{(1, 2)} )</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Covariates</td>
<td>0.54</td>
<td>0.25</td>
<td>0.78</td>
<td>6.70</td>
<td>(2, 32)</td>
<td>*0.004</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Block 2</th>
<th>( R )</th>
<th>( R^2 ) Adjusted</th>
<th>Standard Error of Estimate</th>
<th>( \Delta R^2 )</th>
<th>( \Delta F )</th>
<th>( df_{(1, 2)} )</th>
<th>( \Delta F ) ( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main Effects</td>
<td>0.71</td>
<td>0.35</td>
<td>0.72</td>
<td>0.21</td>
<td>1.81</td>
<td>6, 26</td>
<td>0.14</td>
</tr>
</tbody>
</table>

\* *Significant finding (p < .05); all tests are two-tailed*

In terms of individual variables, multiple regression analyses revealed that FA-Total significantly positively predicted BDI-Total \( (\beta = 1.44, p = .005; \) one-tailed test) after Bonferroni correction (i.e., \( \alpha \) level set at .008). Likewise, FA-Average \( (\beta = -2.18, p = .015; \) one-tailed test) and FA-Abs \( (\beta = -1.31, p = .01; \) one-tailed test) both significantly negatively predicted BDI-Total at the trend level after Bonferroni correction (i.e., \( \alpha \) level set at .008). For two-tailed hypotheses, neither TFRC nor AFRC significantly predicted BDI-Total. No other findings were significant. Beta coefficients are shown in Table 9.
Table 9. Beta coefficients for covariates and dermatoglyphic main effects predicting BDI-Total

<table>
<thead>
<tr>
<th>Block 1</th>
<th>Variable</th>
<th>Unstandardized $B$</th>
<th>Standard Error</th>
<th>Standardized $β$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Covariates</td>
<td>²Age</td>
<td>-0.27</td>
<td>0.16</td>
<td>-0.28</td>
<td>0.10</td>
</tr>
<tr>
<td></td>
<td>²Household Income</td>
<td>-0.25</td>
<td>0.08</td>
<td>-0.46</td>
<td>*0.01</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Block 2</th>
<th>Variable</th>
<th>Unstandardized $B$</th>
<th>Standard Error</th>
<th>Standardized $β$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main Effects</td>
<td>¹FA-Total</td>
<td>1.29</td>
<td>0.46</td>
<td>1.44</td>
<td>*0.01</td>
</tr>
<tr>
<td></td>
<td>¹FA-Average</td>
<td>-2.1</td>
<td>0.89</td>
<td>-2.18</td>
<td>+0.03</td>
</tr>
<tr>
<td></td>
<td>¹FA-Abs-Average</td>
<td>-1.2</td>
<td>0.46</td>
<td>-1.31</td>
<td>+0.02</td>
</tr>
<tr>
<td></td>
<td>²TFRC</td>
<td>-0.18</td>
<td>0.50</td>
<td>-0.21</td>
<td>0.72</td>
</tr>
<tr>
<td></td>
<td>²AFRC</td>
<td>0.23</td>
<td>0.54</td>
<td>0.26</td>
<td>0.68</td>
</tr>
</tbody>
</table>

¹ One-tailed test (divide the reported $p$-value by 2)
² Two-tailed test
* Significant finding ($p < .05$)
† Significant finding after Bonferroni correction (using $α$ set at .008); one-tailed test
†† Trend level finding after Bonferroni correction (using $α$ set at .017); one-tailed test

Note: Bonferroni correction (i.e., divide .05 by 6 comparisons = .008) was applied to analysis of all variables. Findings were significant at trend level by dividing .05 by 3 comparisons = .017)

3. Dermatoglyphics in Relation to Depressive Symptoms and Potential Moderating Sex Effects

The overall ANOVA showed that sex interaction terms (i.e., each dermatoglyphic measure by sex) did not significantly contribute to the overall multiple regression model ($F(15, 19) = 1.70, p = 0.14$). Likewise, sex interaction terms did not significantly contribute additional explanatory variance to the multiple regression model above and beyond the contribution of main effects ($F_{Change}(7, 25) = 1.74, p = 0.87$). Results are shown in Table 10.
Table 10. *Multiple regression values for R, R² change, overall F statistics, and F change statistics for covariates, dermatoglyphic main effects, and sex interaction terms predicting BDI-Total.*

<table>
<thead>
<tr>
<th></th>
<th>R</th>
<th>R² Adjusted</th>
<th>Standard Error of Estimate</th>
<th>Δ R²</th>
<th>Δ F</th>
<th>df(1, 2)</th>
<th>Δ F p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Block 1</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Covariates</td>
<td>0.54</td>
<td>0.25</td>
<td>0.78</td>
<td></td>
<td></td>
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</tr>
<tr>
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<td>F</td>
<td>df(1, 2)</td>
<td>p</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>6.70</td>
<td>(2, 32)</td>
<td>*0.004</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Block 2</strong></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Main Effects</td>
<td>0.72</td>
<td>0.34</td>
<td>0.73</td>
<td>0.22</td>
<td>1.64</td>
<td>7, 25</td>
<td>0.17</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>df(1, 2)</td>
<td>p</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2.97</td>
<td>(9, 25)</td>
<td>*0.02</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Block 3</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interaction Terms</td>
<td>0.76</td>
<td>0.23</td>
<td>0.78</td>
<td>0.06</td>
<td>0.41</td>
<td>6, 19</td>
<td>0.87</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>df(1, 2)</td>
<td>p</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.70</td>
<td>(15, 19)</td>
<td>0.14</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Significant finding (p < .05); all tests are two-tailed.

Sex did not significantly moderate the relationships of any dermatoglyphic variables (FA or finger ridge counts) with BDI-Total. Results are shown in Table 11.
## Table 11. Beta coefficients for covariates, dermatoglyphic main effects, and sex interaction terms predicting BDI-Total

<table>
<thead>
<tr>
<th>Block 1</th>
<th>Variable</th>
<th>Unstandardized B</th>
<th>Standard Error</th>
<th>Standardized β</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Covariates</td>
<td>²Age</td>
<td>-0.20</td>
<td>0.19</td>
<td>-0.21</td>
<td>0.30</td>
</tr>
<tr>
<td></td>
<td>²Household Income</td>
<td>-0.22</td>
<td>0.11</td>
<td>-0.41</td>
<td>0.07</td>
</tr>
<tr>
<td>Block 2</td>
<td>Variable</td>
<td>Unstandardized B</td>
<td>Standard Error</td>
<td>Standardized β</td>
<td>p</td>
</tr>
<tr>
<td>Main Effects</td>
<td>¹FA-Total</td>
<td>1.31</td>
<td>0.60</td>
<td>1.47</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>¹FA-Average</td>
<td>-2.47</td>
<td>1.19</td>
<td>-2.63</td>
<td>0.05</td>
</tr>
<tr>
<td></td>
<td>¹FA-Abs</td>
<td>-1.30</td>
<td>0.59</td>
<td>-1.44</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>¹FA-Abs-Average</td>
<td>2.29</td>
<td>1.12</td>
<td>2.35</td>
<td>0.06</td>
</tr>
<tr>
<td></td>
<td>²TFRC</td>
<td>-0.21</td>
<td>0.66</td>
<td>-0.24</td>
<td>0.76</td>
</tr>
<tr>
<td></td>
<td>²AFRC</td>
<td>0.30</td>
<td>0.69</td>
<td>0.34</td>
<td>0.67</td>
</tr>
<tr>
<td></td>
<td>²Sex</td>
<td>-0.35</td>
<td>0.31</td>
<td>-0.19</td>
<td>0.28</td>
</tr>
<tr>
<td>Block 3</td>
<td>Variable</td>
<td>Unstandardized B</td>
<td>Standard Error</td>
<td>Standardized β</td>
<td>p</td>
</tr>
<tr>
<td>Interaction Terms</td>
<td>²FA-Total x Sex</td>
<td>0.21</td>
<td>1.17</td>
<td>0.12</td>
<td>0.86</td>
</tr>
<tr>
<td></td>
<td>²FA-Average x Sex</td>
<td>0.57</td>
<td>2.42</td>
<td>0.31</td>
<td>0.82</td>
</tr>
<tr>
<td></td>
<td>²FA-Abs x Sex</td>
<td>0.49</td>
<td>1.33</td>
<td>0.26</td>
<td>0.72</td>
</tr>
<tr>
<td></td>
<td>²FA-Abs-Average x Sex</td>
<td>-1.28</td>
<td>2.41</td>
<td>-0.66</td>
<td>0.60</td>
</tr>
<tr>
<td></td>
<td>²TFRC x Sex</td>
<td>-0.98</td>
<td>1.34</td>
<td>-0.53</td>
<td>0.47</td>
</tr>
<tr>
<td></td>
<td>²AFRC x Sex</td>
<td>0.27</td>
<td>1.58</td>
<td>0.14</td>
<td>0.86</td>
</tr>
</tbody>
</table>

1. One-tailed test (divide the reported p-value by 2)
2. Two-tailed test

* Significant finding (p < .05)
* Significant finding after Bonferroni correction (using α set at .008); one-tailed test
† Trend level finding after Bonferroni correction (using α set at .017); one-tailed test

Note: Bonferroni correction (i.e., divide .05 by 6 comparisons = .008) was applied to analysis of all variables. Findings were significant at trend level by dividing .05 by 3 comparisons = .017)
CHAPTER IV.

DISCUSSION

1. Present Findings

The current study had the following three goals towards understanding expression of depressive symptomatology among non-clinical adolescents and young adults: (1) to examine the potential impact and timing of environmental stress and genetic factors during prenatal development by measuring the relationship of indirect measures of developmental instability (i.e., indices of dermatoglyphic FA) with depressive symptoms; (2) to consider the differential impact of other indirect measures of prenatal insult that are believed to more heavily reflect genetically-mediated processes (i.e., indices of dermatoglyphic ridge counts); and (3) to investigate the potential moderating effect of sex in the relationship of dermatoglyphic indices with depressive symptoms. Towards these ends, individuals without any current or past psychiatric diagnoses were recruited from the general population and asked to provide fingerprints and self-reported depressive symptoms. Multiple regression analyses were conducted to assess for relative contributions of various digit dermatoglyphic measures to depressive symptomatology.

Depressive symptom endorsement in the current non-clinical sample of adolescents and young adults was generally consistent with prior investigations (Osman, Barrios, Gutierrez, Williams, & Bailey, 2008; Steer & Clark, 1997; Whisman et al., 2000). The BDI-Total score for the current sample of $M = 8.21$ ($SD = 7.87$) is commensurate with, albeit somewhat lower than, other studies of non-clinical adolescents that found $M = 12.50$ ($SD = 10.50$) (Osman et al., 2008), $M = 12.75$ ($SD = 9.07$) (Steer & Clark, 1997), and $M = 7.16$ ($SD = 7.16$) (Whisman et al., 2000). Further, the percentage of subjects falling into the minimal (75.5%), mild (17.0%), moderate (3.8%), and
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severe (3.8%) ranges in the current sample are similar to those found by Whisman et al. (2000) (80.2%; 12.5%; 5.7%; 1.6%, respectively). Taken together, this suggests that the BDI-II validly captured depressive symptoms in the current non-clinical sample of adolescents and young adults.

In line with predictions, findings initially revealed that one measure of dermatoglyphic fluctuating asymmetry (FA-Total) significantly predicted endorsement of depressive symptoms among a non-clinical sample of adolescents and young adults, after applying statistical corrections for multiple comparisons. Specifically, greater total finger ridge count (TFRC) fluctuating asymmetry (FA) significantly predicted greater depressive symptomatology. Notably, this finding did not hold after accounting for multicollinearity, and results are accordingly interpreted with caution. In this light, the initial finding lends potential (albeit tentative) support to the hypothesis that reduced buffering capacity to environmental noise during the prenatal period, as measured by heightened dermatoglyphic FA (Kowner, 2001), may in fact increase risk of depressive experiences during adolescence and young adulthood (King et al., 2009; Martin, Manning, Dowrick, 1999; Shackelford & Larsen, 1997). Although speculative, this finding may indirectly suggest that development of neural limbic structures (i.e., hippocampus, entorhinal cortex, parahippocampal cortex) and their connective pathways (i.e., with frontal systems) that are implicated in depression (for reviews see Bracht, Linden, & Keedwell, 2015; Brand, Moller, & Harvey, 2015; Mayberg, 2003; Zhang et al., 2016) are affected by prenatal disturbance during the late first and early second trimester, as captured by dermatoglyphic FA. In line with the diathesis-stress model of psychopathology, the tentative finding of greater FA predicting greater depressive symptoms lends potential evidence that aberrant neurodevelopment may serve as a putative underlying vulnerability that warrants further study in depression emergence. Further, in
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line with previous findings in non-clinical and mixed-clinical samples (Martin et al., 1999; Shackelford & Larsen, 1997), the current study demonstrates that this relationship is potentially detectable within a non-clinical sample, further supporting the dimensional approach in understanding depression etiology.

Contrary to hypotheses, however, the other three FA measures did not significantly predict depressive symptoms after statistical correction for multiple comparisons. This lack of findings is likely due to, in part, issues with multicollinearity, limited sample size, and other considerations discussed in more detail in the ‘Limitations’ section below. Moreover, this is the first study to our knowledge investigating the relationship of mood factors such as depressive symptoms with dermatoglyphic FA, as opposed to indices of FA using other morphologic features. It is possible that the total FA measure, compared with absolute FA and average FA measures, is more sensitive to such symptoms in a non-clinical sample. In terms of timing, FA derived from TFRCs is believed to reflect disturbance during weeks 6 to 10.5 of gestational age, while FA from AFRCs is understood to reflect the slightly later period of gestational weeks 10.5 to 13 (Davis & Bracha, 1996). The current findings may suggest that, among non-clinical individuals, risk for depressive symptoms may be related to environmental disturbance during the early prenatal period. In terms of methodology, previous studies looking at dermatoglyphic FA have used both total FA (King et al., 2009; van Oel et al., 2001) and average FA (Golembo-Smith et al., 2012; Green et al., 1994) with no seeming difference. However, the average FA measures yielded high variability in the current sample (see Table 3), which may have obscured findings via increased statistical noise in the analysis. There are alternate methodologies for calculating asymmetry, such as qualitative assessment of whether patterns (e.g., whirls, loops,
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arches) match across homologous fingers (Davis & Bracha, 1996), that may be used in future studies that might reduce noise and potentially yield clearer results. Other studies investigating FA also found mixed or null findings until analyses were split by sex or sex was included as a moderator (Burton et al., 2003; Daly et al., 2008; Martin et al., 1999; Shackelford & Larsen, 1997). Given the small sample size in this study (discussed below), analyses looking at sex-effects were limited.

Finger ridge counts (both total and absolute) also did not significantly predict depressive symptoms. These measures are conceptualized as reflecting both the size (TFRC) and intensity (AFRC) of fingerprint patterns (Cummins & Midlo, 1961; Schaumann & Alter, 1976). Based on Babler’s (1978) study of spontaneous versus selective abortions, higher frequency of low TFRCs is understood to reflect disruptions to genetically-mediated development. Lower AFRCs are considered to be somewhat more sensitive to environmental insults (Schaumann & Alter, 1976). FRCs have traditionally been investigated in relation to schizophrenia spectrum disorders (Golembo-Smith et al., 2012) and may be less clearly related to subtler psychological symptoms with putatively dissimilar pathophysiologies, such as depressive symptoms. These indices are also computationally more susceptible to high error than FA measures, as there is no intra-individual comparison.

Unlike prior investigations in non-clinical and mixed-clinical samples (Burton et al., 2003; Daly et al., 2008; Martin et al., 1999; Shackelford & Larsen, 1997), in the current study, sex did not moderate relationships among dermatoglyphic variables and depressive symptoms. Specifically, taking sex into consideration accounted for an insignificant portion of variance in the overall
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regression model ($R^2$ change = .06, $p > .05$; see Table 10). The absence of a moderating effect by sex may be due to relatively low statistical power given the small sample size and the somewhat uneven distribution of the sample by sex (41.5% M / 58.5% F). Nevertheless, considering the current sample was primarily derived from college students, a population that typically skews more towards females (Harton & Lyons, 2003), the current distribution more closely resembled national averages than may have been expected. Accordingly, the lack of interaction effects by sex most likely reflects limited sample size.

2. Limitations

There were multiple limitations to the current investigation. First, there were issues with multicollinearity among the dermatoglyphic variables that may have affected findings. Accordingly, findings are reported and interpreted with caution. Second, the relatively small sample size limited statistical power and may have increased the likelihood of Type II error. Third, some researchers have raised theoretical concerns when investigating relationships between fixed trait markers such as dermatoglyphics and transient, state-like symptoms of depression (Gabaldon & Compton, 2010). Such relationships may emerge with a degree of inconsistency and/or be difficult to replicate. This consideration is particularly relevant in the current study of adolescents and young adults, as these participants represented the developmental stage around which depression onset often emerges and were therefore more likely to endorse early state-like symptoms. Nevertheless, state measures of depression were appropriate to assess given the goals of the overarching project using a non-clinical sample. Fourth, the current sample had unique racial/ethnic demographic characteristics that were more consistent with an urban setting than the general US population. This population is important to
include in investigations; however, findings should be generalized to non-urban populations with caution. Fifth, the current investigation did not include dermatoglyphic measures that may reflect disruptions during different developmental periods (e.g., a-b palmar ridge counts capture second to third trimesters, ATD angles capture change from the prenatal period throughout adolescence (Davis & Bracha, 1996) (see Golemo-Smith et al., 2012)). Future investigations should assess the extent to which different dermatoglyphic measures relate to specific symptom domains across conditions with putative neurodevelopmental considerations (e.g., psychosis, ADHD, ASD, depression) (Russak, Ives, Mittal, & Dean, 2016). Sixth, measurement error in detecting non-directional asymmetry has been estimated to range from 10-76% across various traits of non-human species (Palmer & Strobeck, 1986). Measurement error in human traits, and particularly digit dermatoglyphics is considerably lower. For example, certain morphologic traits are more likely to change over time due to exogenous events (e.g., wrist diameter changing as a function of a sport or previous injury), while digit dermatoglyphics are quite stable over time (Cummins & Midlo, 1961). However, print clarity was varied across subjects and may have contributed to measurement error in this study. In the current study, TFRCs were not legible for 11 subjects due to poor print quality; AFRCs were not available for an additional 4 subjects due to unreadable prints. However, there were no significant differences on demographic variables between subjects with and without legible prints, suggesting randomly distributed error. Seventh, dermatoglyphic measures serve as indirect, distal indices of potential prenatal disruptions. Birth record data or maternal retrospective report of the prenatal period would help validate dermatoglyphic measures as a proxy for prenatal insult.

3. Conclusions, Implications and Future Directions
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In conclusion, consistent with prior literature (King et al., 2009) and current study hypotheses, one measure of dermatoglyphic FA significantly predicted greater endorsement of depressive symptoms in a sample of non-clinical adolescents and young adults recruited from the general population. Importantly, however, this finding did not hold when considering multicollinearity, and results are therefore interpreted conservatively and with caution. The tentative finding that dermatoglyphic FA predicts depressive symptoms supports prior literature demonstrating the utility of digit dermatoglyphics as a potentially viable proxy for estimating decreased buffering capacity to environmental disturbance during the prenatal period (King et al., 2009; Kowner, 2001). Likewise, this finding (albeit limited in the current study) is consistent with previous research that suggests aberrant neurodevelopment is an underlying vulnerability that may contribute to risk for depression (King et al., 2009, 2012; Martin et al., 1999; Shackelford & Larsen, 1997). The emergence of this finding in a non-clinical sample of individuals with a range of depressive symptoms, and during the period that precedes the typical age of onset of depression, fits with, though does not directly lend further evidence to, the dimensional model of depressive experiences (Cuijpers et al., 2004; Goldberg, 2000; Hankin et al., 2005; Lewinsohn et al., 2000; Solomon et al., 2001). Viewing depression pathophysiology through the lens of aberrant neurodevelopment holds potential implications for an improved understanding of illness etiology and prevention through early identification of individuals who may be at risk.

There is a growing body of research investigating potential diagnostic applications for dermatoglyphic measurements across varied medical conditions (Ahmed-Popova, Mantarkov, Sivkov, & Akabaliev, 2014; Dangore-Khasbage, Degwekar, Bhowate, Lohe, & Tamgire, 2012; Domany et al., 2018; Lakshmana, Nayyar, Pavani, Ratnam, & Upendra, 2017; Shivaleela, Hanji,
& Kumar, 2013), though findings are more limited with psychiatric conditions, and MDD in particular. To date, dermatoglyphic deviations have been detected in a range of metabolic, neurologic, dermatologic, oncologic, cardiac, autoimmune, developmental, and oral conditions, including type II Diabetes Mellitus (Shivaleela et al., 2013), aphthous stomatitis (Dangore-Khasbage et al., 2012), sickle cell anemia, psoriasis, epilepsy, tumors, congenital heart disease, cervical cancer, and lupus erthematodes (for reviews see Ahmed-Popova et al., 2014; Lakshmana et al., 2017). Multiple investigations have demonstrated that dermatoglyphics can be diagnostically sensitive in identifying Down syndrome (Kiran, Rai, & Hegde, 2010; Langenbeck et al., 1988). As far as psychiatric conditions, a recent study demonstrated that a combination of biomarkers pertaining to the hand, including palmar dermatoglyphics, was able to effectively distinguish patients with schizophrenia from individuals with other mental disorders at a rate of nearly 80% (Domany et al., 2018). That said, the results from studies investigating dermatoglyphics alone in schizophrenia are mixed (for review see Golembo-Smith et al., 2012), and there is no demonstration as of yet that dermatoglyphics have adequate sensitivity or specificity in diagnosing schizophrenia or other psychiatric conditions, such as MDD. Rather, investigation of dermatoglyphics in relation to depressive disorders (and risk for depressive disorders) hold implications primarily via heightened understanding of disease pathogenesis. Recognition of the potential effects of neurodevelopment in increasing risk towards depressive experiences may help with identification of individuals who are at risk due to deleterious prenatal conditions.

Future investigations may build on the current study by repeating analyses with larger sample sizes, more even sex distributions, and individuals with a range of racial/ethnic characteristics.
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representative of both urban and non-urban environments. Future work may also assess additional dermatoglyphic measures such as a-b palmar ridge counts, ATD angles, and qualitative assessment of patterns, to capture their relative predictive contributions. It would be helpful to include corroborative markers in future analyses to confirm the validity of dermatoglyphic indicators. Finally, it would be important to conduct large-scale follow-up assessments to determine whether dermatoglyphic markers continue to remain predictive of depressive symptoms in individuals who eventually convert to clinically significant levels of MDD.
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