SEX DIFFERENCES IN ANXIETY:

TESTING A PRENATAL ANDROGEN HYPOTHESIS USING BEHAVIORAL AND PHYSIOLOGICAL MARKERS

A Thesis

by

MILAGROS EVARDONE

Submitted to the Office of Graduate Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

May 2006

Major Subject: Psychology

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Approved by:

Chair of Committee,	Gerianne M. Alexander
Committee Members,	Leslie C. Morey
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ABSTRACT

Sex Differences in Anxiety:

Testing a Prenatal Androgen Hypothesis Using Behavioral

and Physiological Markers. (May 2006)

Milagros Evardone, B.S., University of Florida

Chair of Advisory Committee: Dr. Gerianne M. Alexander

The majority of studies examining the role of prenatal androgens on abnormal behavior have focused on developmental disorders showing large male to female ratios (i.e., autism and Tourette's Syndrome). There is a scarcity of research examining the role of prenatal sex hormones on female-linked disorders or disorders showing adult onset. This study is the first to evaluate the organizational and activational influences of sex hormones on adult levels of anxiety, while simultaneously examining previously reported hormone-behavior associations. In addition, this study explores the relation between prenatal and postnatal sex hormones and two other female-linked disorders, depression and borderline personality. As part of this study, participants (n = 110) completed a battery of psychopathology questionnaires, gender role measures, and spatial/cognitive tasks. Prenatal androgen levels were indirectly measured by means of the index to ring finger ratio (2D:4D), and testosterone and estrogen levels were obtained from saliva samples. Results replicate previously reported sex differences in anxiety and gendered behavior and confirm various hormone-behavior associations. More importantly, results provide preliminary evidence for the organizational role of

prenatal androgens in two female-linked conditions, anxiety and borderline personality. Individuals with a higher (i.e., more feminine) 2D:4D reported greater symptoms of trait anxiety and borderline personality (i.e., affective instability), and this effect appeared to be strongest in males.

DEDICATION

I would like to dedicate this thesis to my family, and especially to my grandmother. Though you are no longer with me, you continue to be my role model and source of strength. You are my motivation to pursue graduate work, and I can only hope that this modest accomplishment brings you pride. Through difficult times, your voice pushes me to succeed.

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INTRODUCTION

National epidemiological surveys in various countries consistently demonstrate a higher prevalence of anxiety disorders in women than in men (Horwath & Weissman, 1997). According to reviews, females are two times more likely than males to develop an anxiety disorder during their lifetime (Bijl, De Graaf, Ravelli, Smit, & Vollebergh, 2002; Halbreich, 2003; Pigott, 1999; Pigott, 2003; Reich, 1986; Shear, Feske, & Greeno, 2000; Weissman, 1988). Panic disorder (PD) with and without agoraphobia, simple phobia, post-traumatic stress disorder (PTSD), and generalized anxiety disorder (GAD) show the strongest female vulnerability (Bijl et al., 2002; Carter, Wittchen, Pfister, & Kessler, 2001; Fredrikson, Annas, Fischer, & Wik, 1996; Gavranidou & Rosner, 2003; Pigott, 1999; Pigott, 2003;Wittchen & Hoyer, 2001; Wittchen, Zhao, Kessler, & Eaton, 1994). Obsessive-compulsive disorder (OCD) and social phobia show less of a sex difference, although these two conditions continue to affect more females at a ratio of 1.5 to 1 (Furmark et al., 1999; Pigott, 1999; Pigott, 2003). Children and adolescents display a similar pattern, with girls having a higher risk for separation anxiety, social phobia, simple phobia, and panic disorder, and boys showing a greater risk for obsessive-compulsive disorder (Weiss & Last, 2001; Weissman, 1988). Females also report higher levels of non-clinical anxiety, and a trend for higher anxiety sensitivity than males (Armstrong & Khawaja, 2002).

In addition to sex differences in the prevalence of anxiety disorders, sex and/or

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This thesis follows the style of *Archives of Sexual Behavior*.

gender also play an influential role in the individual's symptoms and clinical course. Table 1 presents a summary of previous work reviewing the gender-divergent presentation of anxiety disorders. With the exception of OCD, anxiety disorders appear to be not only more prevalent, but also more severe in females than in males. As a rule of thumb, females are more functionally impaired, report a greater number and degree of symptoms, and have added comorbid diagnoses. Strong support is, thus, available for a sex difference in anxiety. The question left to answer is why women seem to be at a greater risk. It is this perpetual and enigmatic question that is at the core of the present study.

Table 1. Gender-divergent Presentation of Anxiety Disorders

	Female to Male Ratio	Symptoms	Comorbidity	Severity	Onset/Age Distribution
GAD (Carter et al., 2001; Pigott, 1999; Pigott, 2003; Wittchen et al., 1994; Wittchen & Hoyer, 2001; Yonkers, Bruce, Dyck, & Keller, 2003)	2:1	----------	- Greater comorbid dysthymia in females	- More chronic course in females - Higher rate of remission in males, followed by greater relapse at 8-year follow-up	- Increased rates in females over 45 years old
PD (Pigott, 1999; Pigott, 2003; Sheikh, Leskin, & Klein, 2002; Turgeon, Marchand, & Dupuis, 1998)	2:1 – 3:1	- Greater number of individual symptoms reported by females -Females report greater agoraphobic avoidance, catastrophic cognitions, and fear of bodily sensations - Females report greater shortness of breath, faintness, and smothered feeling - Males report more GI complaints	- Females have increased risk of comorbid agoraphobia, depression, GAD, simple phobia, alcohol abuse, social phobia, PTSD, and somatization disorder - Males have increased risk of alcohol abuse	- Greater severity, functional impairment, and recurrence in females	----------
PTSD (Gavranidou & Rosner, 2003; Pigott, 1999; Pigott, 2003; Yonkers & Kidner, 2002)	2:1	- Most common trauma in females: sexual assault - Most common trauma in males: combat or physical attack	- Greater comorbid somatoform pain disorder in females - Equal rates of GAD and major depression in males and females	- Longer, more chronic course expected in females	- Males report a greater number of traumatic events, but women exposed to trauma are more susceptible to PTSD
Simple Phobia (Fredrikson et al., 1996; Pigott, 1999; Pigott, 2003)	Animal phobias 2:1 Situational phobias 4:1 Mutilational/Health-related 1:1	- Females report higher fear ratings in general - Most common phobia in females: animal - Most common phobia in males: fear of heights	----------	----------	----------

Table 1. Continued

	Female to Male Ratio	Symptoms	Comorbidity	Severity	Onset/Age Distribution
Social Phobia (Furmark et al., 1999; Pigott, 1999; Pigott, 2003; Turk et al., 1998)	1.5:1	- Generalized subtype more common in females - Females report greater fears when: 1) speaking before an audience 2) speaking to an authority figure 3) being observed as they work 4) entering the room after others are seated 5) becoming the center of attention 6) having to speak up at a meeting 7) disagreeing with others 8) reporting to a group 9) throwing a party - Males report greater fears when: 1) using public bathrooms 2) returning purchases	- Greater comorbid agoraphobia in females - Greater substance abuse in men	- Greater severity of symptoms in females	----------
OCD (Fontenelle, Marques, & Versiani, 2002; Lochner et al., 2004; Pigott, 1999; Pigott, 2003; Yonkers & Kidner, 2002)	1.5:1	- Males display more aggressive obsessions and compulsions and checking rituals - Females display more compulsive cleaning and washing rituals	- Greater comorbid depression and eating disorders in females - Greater history of tic and bipolar disorders in males	- Nonsignificant trends for longer course, decreased global functioning, and worsened outcome in males	- Earlier onset in males - Increased rate of onset in females after menarche - Acute onset and episodic course in females

Note. GAD = Generalized Anxiety Disorder, PD = Panic Disorder, PTSD = Post-traumatic Stress Disorder, and OCD = Obsessive-Compulsive Disorder

REVIEW OF LITERATURE

Several theories aim to account for the robust sex difference in anxiety. This research falls generally under three major categories: social factors/stressors, gender role, and biological explanations.

Social Factors/Stressors

Some researchers argue that women encounter more aversive and stress-producing events during the course of their daily lives (Davis, Matthews, and Twamley, 1999). For instance, females are more likely to experience sexual, physical, and emotional abuse in comparison to males (Finkelhor, Hotaling, Lewis, & Smith, 1990; Young, Abelson, Curtis, & Nesse, 1997), while an increased history of childhood abuse can be found among individuals with anxiety disorders (Stein et al., 1996, Young et al., 1997). Logically then, if females have an increased risk for childhood abuse, and abused individuals are especially vulnerable to developing an anxiety disorder, then it follows that females should have an increased risk for anxiety.

Structural gender inequalities favoring men are another explanation offered for increased rates of anxiety in females. Controlling for education level, women continue to be paid less than men (Gibelman, 2003; O'Campo, Eaton, & Muntaner, 2004), enter less prestigious occupations (Cohen, 2004), and are perceived as having less power (Diekman, Goodfriend, & Goodwin, 2004). This inferior social status is, according to some researchers, a constant and potent source of stress in the lives of women.

Other researchers seek to explain the sex difference in affective disorders by enumerating the multiple roles or "double burden" faced by women. Females appear to

be in a constant struggle between their desire to be occupationally successful and their sense of duty as the lead caretakers of the family. Although according to the role-accumulation hypothesis, men and women with an increased number of social roles should experience less mental health problems (Sachs-Ericsson & Ciarlo, 2000), women who have multiple roles report feeling more anxious, distressed, and describe more somatic complaints (Simon, 1995). These same women also describe more negative self-evaluations and feelings of inadequacy (Simon, 1995). Distress is particularly evident in women who view work as independent of their family, with wife and mother as their primary roles and their job as an added responsibility (Simon, 1995). Nevertheless, other studies fail to support the association between multiple roles and higher rates of psychological disorders in women (Klumb & Lampert, 2004; Sachs-Ericsson & Ciarlo, 2000; Thornton & Leo, 1992).

While it may be a likely contributor, an increased number of social stressors in women cannot sufficiently account for the gender difference in anxiety. First, there is no specific link between stressors and anxiety. Stressful events, such as childhood abuse, are associated with many forms of psychopathology, including depression, substance abuse, dissociative, and eating disorders (Chu, Frey, Ganzel, & Matthews, 1999; Léonard, Steiger, & Kao, 2003; MacMillan et al., 2001). Second, if women experience more stress, then one might expect them to be more vulnerable to other stress-related disorders, such as substance abuse. Yet, the latter is more common among males (Doherty & Szalay, 1996). The issue of why, in females, some disorders (i.e., anxiety) are favored over others (i.e., substance abuse) remains unresolved.

Gender-role Orientation

In an attempt to explain the gender difference in anxiety, the gender role socialization theory has received the most extensive support. According to Bem (1981b), society prescribes a gender dichotomy of behaviors that children internalize and develop into a network of cognitive associations known as a gender schema. Then, this cognitive schema serves as a funnel through which children filter all environmental stimuli, retaining information pertaining to their own gender and rejecting gender-inconsistent information. Through this process, individuals become sex-typed, with the more extensively sex-typed persons displaying an overactive gender schema and recalling more gender-congruent stimuli than androgynous individuals (Bem, 1981b).

Fodor (1974) was one of the early proponents of a link between gender socialization and female affluent disorders. She theorized that women's socially acquired roles as dependent, passive, and fearful creatures fostered a higher prevalence of phobias, particularly agoraphobia, within this gender. In her view, phobias are an expression of hyper-feminine sex typing. A similar perspective states that women are socialized to be interpersonally mindful and worry about others to the point of neglecting themselves. This increased concern for others is, in turn, thought to increase women's risk for psychological distress (Katz, Joiner, & Kwon, 2002). Some researchers, in fact, argue that individuals high on femininity report higher levels of anxiety and that those adhering to the more traditional female role are especially susceptible (Perez Blasco & Serra Desfilis, 1997).

Other researchers state that it is not femininity that is relevant. Rather, it is a lack

of masculinity that is associated with anxiety. Individuals scoring high on feminine characteristics (i.e., dependence, passiveness) do not show greater agoraphobia, fear, or state anxiety (Arrindell, Kolk, Pickersgill, & Hageman, 1993; Chambless & Mason, 1986; Eisler, Skidmore, & Ward, 1988; Ginsburg & Silverman, 2000). Instead, adults and children reporting lower levels of masculine characteristics (i.e., assertiveness, aggression, dominance) also report the highest levels of state anxiety, agoraphobic symptoms, and fear, including social, bodily, and animal fears (Arrindell et al., 1993; Chambless & Mason, 1986; Eisler et al., 1988; Ginsburg & Silverman, 2000). Only fear of sexual and aggressive scenes appears unrelated to decreased masculinity (Arrindell et al., 1993). Given the above results, researchers theorize that masculinity, or the possession of positive male characteristics (i.e., instrumentality), is protective of female disorders.

Still other researchers find both masculinity and femininity to relate to anxiety. While feminine-typed individuals report the highest levels of fear, masculine-typed individuals report the lowest levels of fear both on the Wolpe Fear Inventory and the Fear Survey Schedule (FSS) (Carey, Dusek, & Spector, 1988; Dillon, Wolf, & Katz, 1985). Similarly, females who are more feminine or undifferentiated (i.e., low scores on masculinity and femininity) report higher levels of anxiety on the Clinical Anxiety Scale (CAS) than women who are more masculine or androgynous (i.e., high scores on masculinity and femininity) (Thornton & Leo, 1992). It is possible, however, that feminine individuals score high on fear and anxiety measures because they are also more willing to express their emotions than masculine individuals (Gallacher & Klieger,

2001). In one study, although individuals with a female gender role orientation reported higher levels of fear than masculine individuals, no behavioral difference in fear emerged in the Behavioral Avoidance Task (BAT) (Gallacher & Klieger, 2001). Incidentally, androgynous individuals reported lower levels of fear than masculine individuals on the FSS, perhaps suggesting that androgyny is somewhat protective of psychopathology (Gallacher & Klieger, 2001).

Excessive adherence to gender roles may also increase risk for anxiety. For instance, one line of research argues that masculinity is not always favorable to psychological consequences. Men with increased gender-role stress, those who show a strict commitment to the masculine gender role (Eisler & Blalock, 1991), are more likely to experience higher state and trait anxiety than men with a more flexible behavioral repertoire (Eisler & Skidmore, 1987). Generally speaking, both males and females scoring high on masculine gender-role stress (MGRS) report increased levels of anger, fear, anxiety, depression, and poor health habits (Arrindell et al., 1993; Eisler et al., 1988; McCreary et al., 1996). Nevertheless, there may be a differential trend in the expression of emotional distress. One study found that men who score high on MGRS are more likely to display anger, while females who score high on MGRS are more likely to display anxiety (Eisler et al., 1988). However, McCreary et al. (1996) failed to replicate this sex effect.

Feminine Gender-role Stress (FGRS) may also prove as detrimental to mental health as MGRS. Women who score high on FGRS, those who adhere strictly to the female gender role, also report increased levels of depression (Gillespie & Eisler, 1992).

Given that depression occurs more frequently in women and is highly comorbid with anxiety disorders, these results may prove relevant in the study of anxiety. One should note, however, that the association between FGRS and depression dissolves after controlling for gender-neutral stressors (Gillespie & Eisler, 1992). The latter may perhaps serve as another example that it is not increased feminine characteristics (i.e., expressive traits) but decreased masculine characteristics (i.e., instrumental traits) that elicit the female disorders.

Although the relative contributions of femininity, masculinity, and gender-role stress require further clarification, gender socialization clearly exerts an effect on psychological health consistent with the observed sex differences in anxiety. Even so, learned gender roles cannot fully account for the 2 to 1 female to male ratio in anxiety disorders. For example, only 5% of the variance in fearfulness can be explained by gender-role orientation, suggesting that other factors are involved (Ginsburg & Silverman, 2000). A more comprehensive theory considers environmental influences, such as gender-role and social stressors, in conjunction with biological predispositions (Yonkers & Kidner, 2002).

Biological Explanations

Genetics. Researchers and clinicians have long observed the tendency for anxiety disorders to aggregate in families. A meta-analysis of family studies shows a significant association between probands and first-degree relatives with PD, GAD, phobias (including simple, social, and agoraphobia), and OCD (Hettema, Neale, & Kendler, 2001). For probands with PD, 10% of first-degree relatives developed the disorder in

comparison to 2.1% of control relatives (Hettema et al., 2001). Similarly, for probands with OCD, 8.2% of first-degree relatives developed the disorder in comparison to 2.0% of control relatives (Hettema et al., 2001). This pattern of familial inheritance, in turn, lends support for a genetic component.

Twin studies consistently demonstrate a modest to moderate genetic heritability for anxiety disorders, with the remaining variance largely explained by non-shared (individual) environmental factors and little or no role for shared (familial) environmental factors (Hettema et al., 2001; Kendler et al., 1995). Among the anxiety disorders, PD appears to have the highest genetic heritability, with 43% of variance attributed to shared genes and the rest attributed to non-shared experience (Hettema et al., 2001). For GAD, the genetic contribution is smaller at approximately 32%. A differential distribution by sex occurs for the remaining factors contributing to GAD. Only non-shared environment is important for males, while familial factors play an added role in female etiology (Hettema et al., 2001). In addition, the genetic contribution estimate for phobias is 20-40% (Hettema et al., 2001; Kendler et al., 1995). There is also evidence for two genetic clusters within the anxiety disorders classification. PD and phobia form one genetic grouping, while GAD and depression share the second genetic factor (Kendler et al., 1995). In fact, researchers argue that GAD and major depressive disorder are rooted in the same genetic locus but develop disorder-specific symptoms based on individual, or non-shared environmental factors (Kendler et al., 1995; Kendler, Neale, Kessler, Heath, & Eaves, 1992).

Genetic heritability is not limited to the disorders themselves but also to what

researchers view as their precursors. Childhood separation anxiety, which some believe leads to later PD or agoraphobia, has a 41% genetic heritability in females but no genetic history in males. Non-shared factors and, to a lesser extent, familial factors determine the onset of separation anxiety in boys (Silove, Manicavasagar, O'Connell, & Morris-Yates, 1995). Likewise, genetics account for 45% and 30% of anxiety sensitivity and trait anxiety, respectively (Silove et al., 1995; Stein, Jang, & Livesley, 1999). Genetic factors may, therefore, play a role in the development of the anxious or "neurotic" personality, which may then render the individual susceptible to clinical levels of anxiety.

With the advances in genomic technology and the apparent effectiveness of anxiolytic and antidepressant drugs, research has recently shifted to a search for specific genes involved in the etiology of anxiety. Because monoamine oxidase inhibitor (MAO –A) antidepressants are often used for the treatment of anxiety disorders, the MAO-A gene on chromosome X is one possible culprit. The MAO-A gene has been linked to OCD and PD in particular (Camarena et al., 2001; Deckert et al., 1999). Interestingly, several sex differences emerge at the gene level. For example, females with PD have a greater number of MAO-A high activity alleles than males (Deckert et al., 1999). The opposite is true for OCD, where females have a higher frequency of low activity MAO-A alleles than males (Camarena et al., 2001). Sex differences also exist for the catechol-O-methyltransferase (COMT) gene. Females with OCD have a higher frequency of the low activity allele (Alsobrook et al., 2002). Several other genes seem to affect the course of anxiety, but generating a list is beyond the scope of this research. The MAO-

A and COMT genes are listed here as an illustration of a possible sex-specific genetic contribution to the differences in anxiety.

Neurobiology. While genetics do in fact seem to contribute a significant portion to the heritability of anxiety, the expression of a genotype requires the aid of biologic mechanisms. Genes simply provide a groundwork, encoding information translated into neurochemical processes. Research in this field primarily focuses on the actions of neurotransmitters and neurohormones, which show a link to anxiety in pharmacologic studies. The most commonly implicated systems in the pathogenesis of anxiety include the noradrenergic, dopaminergic, serotonergic, opiate, glutamatergic, and GABA pathways (Brawman-Mintzer & Lydiard, 1997; Charney, Deutch, Krystal, Southwick, & Davis, 1993; Nutt, 2000; Nutt, Bell, & Malizia, 1998; Pigott 2003.) In addition, other neurotransmitters, such as chatecholamine and cholecystokinin (CCK), appear to be involved (Brawman-Mintzer & Lydiard, 1997). Evidence also points to a neuroendocrine dysregulation (Brawman-Mintzer & Lydiard, 1997; Charney et al., 1993; Nutt, 2000; Pigott, 2003). The hypothalamic-pituitary-adrenal (HPA) axis is responsible for the body's normal stress response. In the presence of threat, neurochemicals stimulate the production of corticotropin releasing factor (CRF), which then encourages the pituitary gland to secrete ACTH and the adrenal gland to produce cortisol, leading to autonomic arousal (Nutt, 2000). However, a disruption in this chain of chemical events occurs in anxious individuals and yields an abnormal stress response (Brawman-Mintzer & Lydiard, 1997; Charney et al., 1993; Nutt, 2000.) For example, individuals with PTSD have high glucorticoid and CRF levels (Charney et al., 1993).

HORMONAL EFFECTS ON ANXIETY

Sex Hormones

Neurotransmitter and neuroendocrine systems also appear to interact with sex hormones to influence anxiety symptoms. Estrogen and progesterone, in particular, seem to play a modulatory role in anxiety-linked systems, including the GABAergic, noradrenergic, dopaminergic, and serotonergic complexes, as well as the HPA axis (Chrousos, Torpy, & Gold, 1998; Pigott, 1999; Seeman, 1997). According to researchers, progesterone is both an anxiolytic and a mood destabilizer (Pigott, 1999). The anxiolytic effect of progesterone proceeds from its ability to increase GABA's inhibitory function (Seeman, 1997). Estrogen, on the other hand, is generally considered an anxiolytic (Pigott, 1999). By enhancing dopamine, serotonin, and norepinephrine transmission, decreasing monoamine oxidase activity, exciting glutamate function, and upregulating the GABA/benzodiazepine receptor, estrogen helps decrease levels of anxiety (Pigott, 1999; Seeman, 1997; Smith, Adams, Schmidt, Rubinow, & Wasserman, 2002). It, nevertheless, remains to be seen exactly through what pathways estrogen and progesterone interact with neurotransmitters and neuropeptides to influence the pathogenesis of anxiety, and whether other sex hormones may be involved (i.e. androgens). At the very least, estrogen and progesterone's modulation of anxiety illustrates the ability of sex hormones to directly alter brain structures and influence behavior.

In general, sex steroids affect behavior in two ways: prenatal organization and postnatal activation. A human fetus is phenotypically female until approximately the 6th

week of gestation (for review, see Alexander and Peterson, 2001). At this time, if a Y chromosome is present, testes develop and begin to secrete androgens, causing a masculinization and defeminization of the body, including the brain. If a Y chromosome is not present, then the fetus continues normal development as a female, forming ovaries and female genitalia (Alexander & Peterson, 2001, Breedlove, 1994). The sexual differentiation of brain structures, through androgen levels in prenatal life, results in sex differences in a variety of behaviors (i.e., organizational effects). (Alexander & Peterson, 2001; Breedlove, 1994; Rubinow & Schmidt, 1996). In many instances, the expression of sex-linked behaviors depends on their "activation" by adult circulating levels of gonadal hormones (Alexander & Peterson, 2001; Breedlove, 1994; Rubinow & Schmidt, 1996). In fact, one sign of prenatal hormone involvement in behavior is the sensitivity of that behavior to postnatal hormone levels.

Activational Effects of Sex Hormones

Research examining the effects of gonadal hormones on anxiety concentrates heavily on the cognitive and behavioral effects exerted by post-pubertal sex hormone fluctuations (i.e., activational effects). At puberty, there is an increase in sex hormone production, which catalyzes further sexual differentiation in the individual and establishes adult circulating levels. Coincidentally, anxiety disorders typically onset in adolescence and young adulthood, periods falling on or shortly after puberty (Reich, 1986; Yonkers & Kidner, 2002). The onset of OCD in females, for instance, increases following menarche and surpasses the rate for males (Pigott, 2003). These converging points provide preliminary evidence for the role of sex hormones in anxiety and warrant

their further study.

Menstrual cycle studies offer one possible avenue for the study of gonadal hormones. In retrospective reports, females consistently describe a worsening of anxiety symptoms in the premenstrual phase, when both estrogen and progesterone levels are elevated. The above is true for several conditions, including individuals with panic attacks (Cameron, Kuttesch, McPhee, & Curtis, 1988), PD (Cook et al., 1990), OCD (Williams & Koran, 1997), and GAD in conjunction with premenstrual syndrome (PMS) (McLeod, Hoehn-Saric, Foster, & Hipsley, 1993). However, prospective monitoring of anxiety symptoms across the menstrual cycle shows no significant premenstrual worsening of anxiety (Cameron et al., 1988; Cook et al., 1990). Despite the null findings in prospective studies, individuals with PD viewing anxiety-provoking scenes show increased skin conductance during the premenstrual phase (Sigmon et al., 2000). Furthermore, lactate infusion in individuals with PMS produces panic attack frequencies similar to that of individuals with PD (Facchinetti, Romano, Fava, & Gernazzani, 1992). This lactate sensitivity illustrates a link between the premenstrual period and anxiety. Therefore, the true effect of the premenstrual period on anxiety continues to be an object of debate.

Contrary to a premenstrual increase in anxiety (i.e., a time of increased estrogen and progesterone levels), a second perspective attributes fluctuations in anxiety to the periodic drops of estrogen and progesterone (Seeman, 1997; Williams & Koran, 1997). Support for this theory comes from several sources. A study of rats found that metestrus (i.e., low estrogen/low progesterone) females displayed more anxious behavior (i.e.,

decreased time in the open arms of the plus-maze task) than females in the proestrus phase (i.e., high estrogen/ high progesterone) (Fernandez-Guasti & Picazo, 1990). Metestrus females were also less sensitive than proestrus females to the actions of anxiolytics (Fernandez-Guasti & Picazo, 1990). Similarly, in people with PD, anxiety and panic attacks were more frequent following administration of 35% CO_2 during the early follicular phase, when female hormones are fairly stable and minimal, than during the high estrogen/high progesterone mid-luteal phase (Perna, Brambilla, Arancio, & Bellodi, 1995).

If low levels of female gonadal hormones indeed increase anxiety responses (Seeman, 1997; Williams & Koran, 1997), then one would expect anxiety to decrease during pregnancy, when estrogen and progesterone increase dramatically, and to increase during the postpartum, when high levels of these hormones drop abruptly. The pregnancy and postpartum literature partially supports this theory. A general finding of retrospective studies that anxiety symptoms are reduced during pregnancy was not supported by the results of the only prospective study available (Altshuler, Hendrick, & Cohen, 1998). A second review of PD and pregnancy reveals that an improvement in PD symptoms occurs in 41% of the pregnancies (Hertzberg & Wahlbeck, 1999). Although a mixed picture emerges from the literature, it may be that increased estrogen and progesterone levels during pregnancy may exert an anxiolytic effect on panic disorder (Altshuler et al., 1998). This proposed anxiolytic effect, however, appears to be absent in OCD. Instead, pregnancy seems to confer an increased risk for the onset and worsening of OCD, at least as reported in retrospective studies (for review, see Altshuler

et al., 1998). One particular retrospective study found that 13% of female participants experienced an onset of OCD during pregnancy, and of those who already had OCD prior to pregnancy, 17% described a worsening at this time (Williams & Koran, 1997). Based on results for OCD and PD, no clear conclusion can be drawn on the effect of pregnancy hormones on anxiety. Elevations in female hormones during this period appear to yield adverse effects for some disorders and protective effects for others, and the effects appear specific to the individual, with some people being more sensitive to hormones than others.

In contrast to studies of pregnancy and anxiety, results of postpartum studies generally support the hypothesis that anxiety increases with abrupt decreases in levels of female sex hormones (Seeman, 1997; Williams & Koran, 1997). Panic disorder symptoms are consistently found to increase during the postpartum period (Altshuler et al., 1998). For instance, a review of pregnancy studies found that 38% of pregnancies in these studies were associated with a worsening of PD during the postpartum period (Hertzberg & Wahlbeck, 1999). Similarly, retrospective studies support an onset and increase in OCD symptoms during the postpartum period (Altshuler et al., 1998). In one sample study, 29% of females with OCD prior to pregnancy reported worsening of symptoms during the post-partum (Williams & Koran, 1997). Miscarriage, like the postpartum, also results in a dramatic drop in sex hormones (i.e., progesterone and estrogen). Accordingly, it appears to heighten the risk for an initial or recurrent OCD episode. Of women who miscarried, 3.5% developed OCD as compared to 0.4% of controls (Geller, Klier, & Neugebauer, 2001). A similar trend emerges for PD following

miscarriage, although not statistically significant (Geller et al., 2001). Overall, the postpartum literature and, to some extent, the pregnancy literature suggest that high levels of progesterone and estrogen may together prove protective against anxiety. Conversely, low levels of these hormones may have anxiogenic effects, likely through an interaction with neurotransmitters and the HPA axis (Chrousos et al., 1998; Pigott, 1999; Seeman, 1997).

Organizational Effects of Sex Hormones

From the earlier discussion, it appears that adult sex hormones, in combination with gender role socialization, varying social stressors, and genetics, account for a good portion of the sex difference in anxiety. However, as activational effects are largely dependent on the prenatal sexual differentiation of the brain (Alexander & Peterson, 2001; Breedlove, 1994; Rubinow & Schmidt, 1996), it is possible that prenatal levels of androgens, or the lack thereof, represent another significant risk factor in the female vulnerability to anxiety. Relatively little research has been undertaken in this area, but what has been done suggests a contribution by prenatal hormones to sex differences in psychopathology (Alexander & Peterson, 2001).

In animal research, a sex difference in anxiety has been found for rats, but in the opposite direction as humans. Male rats tend to display more anxious behavior than females, as measured by the amount of time spent in the open arms of the plusmaze task (Lucion, Charchat, Pereira, & Rasia-Filho, 1996; Zimmerberg & Farley, 1993). This sex difference, in turn, appears to be mediated by the organizational and activational effects of gonadal hormones. Male rats castrated at birth, resulting in low androgen levels and

abolishing a male organization of the brain, showed less anxious and more feminized behavior in adulthood (Lucion et al., 1996). Because in humans, females are more at risk for anxiety than males, results based on animal research may be interpreted in a reverse manner. One may hypothesize that males feminized by low levels of prenatal androgens will be more susceptible to anxiety.

Due to ethical constraints, manipulations of prenatal hormone environment cannot be undertaken in human research. Knowledge of prenatal gonadal hormone effects on human behavior is primarily based on studies of individuals with hormonal abnormalities and studies examining physiological markers of prenatal androgen levels, such as the 2D:4D finger-length ratio (Sanders, Sjodin, & de Chastelaine, 2002), which is smaller in males than in females (Brown, Hines, Fane, & Breedlove, 2002; Rahman & Wilson, 2003). In view of these constraints, few studies assessing the organizational role of androgens on sex differences in psychopathology exist. Of the studies available, one examined the relation between the 2D:4D ratio and the development of abnormal behavior in preschool children (Williams, Greenhalgh, & Manning, 2003). Girls with a low 2D:4D ratio (i.e., a more masculine (high) level of androgens in utero) expressed more hyperactivity and increased difficulties with social cognition, prosocial ability, and peer relationships (Williams et al., 2003). Boys with a higher 2D:4D ratio (i.e., a feminine (low) level of prenatal androgens) displayed more anxious behavior than boys with a masculine ratio (Williams et al., 2003). Similar results might be expected in adult men and women, suggesting a vulnerability to anxiety for feminine males. In support of this hypothesis, the 2D:4D ratio also shows a positive correlation with neuroticism, as

measured by the Big Five personality test. Individuals with a feminine (higher) finger-length ratio score higher on the neuroticism factor (Fink, Manning, & Neave, 2004). In as much as neuroticism is considered a dimensional precursor to anxiety, results suggest that individuals with a higher digit ratio will also report greater levels of anxiety. Additionally, though no relation was found between depression and 2D:4D by previous researchers (Martin, Manning, & Dowrick, 1999), a recent study found a positive association between digit ratio and trait depression in males (Bailey & Hurd, 2005). As depression is a female-linked disorder that is highly comorbid with anxiety, a similar association might be expected between 2D:4D and anxiety. Males with a feminine (higher) digit ratio would be predicted to be more anxious than males with a masculine (lower) digit ratio.

The 2D:4D ratio also shows a correlation with autism, a developmental disorder primarily affecting males. Children with autism, their parents, and siblings all seem to exhibit a low 2D:4D ratio indicative of high androgen levels prenatally (Manning, Baron-Cohen, Wheelwright, & Sanders, 2001). The association between high prenatal androgen levels and a male-linked disorder, such as autism, is relevant here in that it suggests a possible association between low prenatal androgen levels and female-linked disorders (i.e., anxiety disorders).

Despite the scarcity of research linking prenatal sex steroids and abnormal behavior directly, pertinent knowledge can be drawn from studies assessing sex differences in normative behavior. Visible differences in gendered behavior, including childhood toy and play preferences, have been repeatedly associated with prenatal

androgen levels. The greater the androgen exposure, the more male-typical the behavior, and the lower the androgen levels, the more female-typical the behavior (Berenbaum & Hines, 1992; Leveroni & Berenbaum, 1998; Meyer-Bahlburg et al., 2004; Servin, Nordenström, Larsson, & Bohlin, 2003; Udry, 2000; Udry, Morris, & Kovenock, 1995). In particular, girls with Congenital Adrenal Hyperplasia (CAH), who have masculine levels of androgens prenatally, prefer boys' toys (e.g. a ball or a car) over girls' toys (e.g. a doll or tea set), report more male playmates, less maternal interest in infants, and aspire to more masculine professions (Berenbaum & Hines, 1992; Leveroni & Berenbaum, 1998; Meyer-Bahlburg et al., 2004; Servin et al., 2003). Females with a low 2D:4D ratio (high prenatal androgens) are also more likely to conform to a masculine gender role (Csathó et al., 2003).

Recall from the gender role discussion that a masculine sex-type is related to a lower risk for anxiety (Arrindell et al., 1993; Carey et al., 1988, Chambless & Mason, 1986; Dillon et al., 1985; Eisler et al., 1988; Thornton & Leo, 1992), unless it is accompanied by a rigid adherence to the male gender role characteristics (Eisler & Blalock, 1991; Eisler & Skidmore, 1987; Eisler et al., 1988). Also, recall that a feminine sex-type is associated with an increased level of fear and anxiety (Perez Blasco & Desfilis, 1997; Carey et al., 1988; Dillon et al., 1985; Gallacher & Klieger, 2001; Thornton & Leo, 1992), although this association is not as strongly supported. Now, if prenatal androgens predict gender role behavior and this, in turn, predicts vulnerability to anxiety, it is possible to think of prenatal sex steroids as indirect determinants of later anxious behavior. It is also possible for prenatal sex hormones to exert direct effects on

brain structures, which are involved in the pathogenesis of anxiety disorders.

Prenatal androgens may also account for large sex differences in cognitive abilities, and this association could theoretically provide an avenue for the indirect measurement of prenatal androgen levels across and within the sexes. Males, who have an increased amount of androgens in utero, (Breedlove, 1994; Rubinow & Schmidt, 1996), demonstrate a consistent advantage on visuospatial (e.g. mental rotation) and targeting (e.g. throwing a ball at a target) tasks (Linn & Petersen, 1985; Watson & Kimura, 1991). Females, who have lower levels of androgens than males in prenatal life (Breedlove, 1994; Rubinow & Schmidt, 1996) exhibit a small advantage on verbal fluency (Hyde & Linn, 1988) and a moderate advantage on memory tasks (e.g. spatial working memory and memory for object locations) (Alexander, Packard, & Peterson, 2002; Duff & Hampson, 2001; Eals & Silverman, 1994). Sex-linked tasks could, thus, serve as behavioral markers of prenatal androgen levels, such that individuals performing better on male-linked tasks would be expected to have high levels of androgens prenatally (e.g. CAH females) (Hampson, Rovet, & Altmann, 1998), and individuals performing better on female-linked tasks would be expected to have low levels of androgens.

As certain psychological disorders show a sexually dimorphic pattern, also theorized to emerge from differing levels of prenatal sex hormones, performance on sex-linked tasks is believed to vary systematically across these disorders. Recently, a correlation was established between several behavioral markers and Tourette's Syndrome, a developmental disorder characterized by abnormal tic behavior and

showing a strong loading in males (Alexander & Peterson, 2004). To test the hypothesis that a prenatal masculinization of the brain via high levels of androgens increased risks for tic disorders, this study found that affected females displayed a more masculine pattern of behavior. They reported more gender dysphoria and masculine play preferences and showed poor performance on location memory, but enhanced performance on mental rotation. Results imply that females masculinized by high levels of androgens during the organizational stage of neural development are at a heightened risk for tic-related disorders (Alexander & Peterson, 2004).

PURPOSE AND HYPOTHESIS

If prenatal sex steroids are involved in the pathogenesis of male-linked psychological disorders (Alexander & Peterson, 2004; Manning et al., 2001), then there is reason to believe that female-typical psychopathologies are also influenced by prenatal androgens. In fact, recent findings suggest a link between depression and prenatal sex hormones (Bailey & Hurd, 2005). This one study, however, is insufficient to justify an organizational effect on female-linked disorders. As a further test of this hypothesis, this study examined the relation between prenatal androgens and another common female disorder, anxiety. In the hopes of accumulating preliminary evidence that would later warrant the study of specific diagnoses, the present study examined the relation between non-clinical anxiety and phenotypic markers of prenatal androgen levels (i.e., 2D:4D digit ratio), as well as several behavioral correlates, including gender identity, gender-type, play preferences, and spatial abilities.

Results were expected to replicate previous findings of greater anxiety in women compared to men and demonstrate the typical sex differences in play preferences, gender role bias, spatial abilities, and the 2D:4D ratio. In view of the organizational effects of androgens on anxiety in rats and the indirect support of this association in humans, the primary hypothesis was that prenatal androgen levels, as measured by the finger-length ratio and indicated by the direction of performance on androgen-sensitive tasks, would be associated with a sexually-dimorphic vulnerability to anxiety. Specifically, it was hypothesized that measures of anxiety would differ between groups of individuals with smaller and larger digit ratios. It was also expected that within-sex performance on

gender-linked tasks would correlate with levels of anxiety.

METHODS

Participants

As part of a larger study of sex differences in memory and spatial abilities, 110 participants (58 male and 52 female) were recruited from Introductory Psychology courses at Texas A&M University and the community. Those recruited through Introductory Psychology courses received two credits applied to their course work, and those recruited from the community were paid $15 for their participation.

Materials

Salivary Samples Questionnaire. To ensure participants' compliance to sample collection restrictions, a short questionnaire was given asking participants about medication use, use of alcohol within 24 hours, consumption of a major meal within 60 minutes, avoidance of highly acidic and sugar foods, brushing teeth within 3 hours, and consumption of dairy products within 30 minutes of sample collection.

Handedness Questionnaire. Because handedness is associated with brain lateralization and may influence performance on sex-linked tasks (i.e., left handedness is more frequent among males), handedness was assessed by hand preference for writing.

Menstrual Cycle Questionnaire. Female hormone fluctuations across the menstrual cycle may affect mood (Cameron et al, 1988; Cook et al., 1990; McLeod et al., 1993; Williams & Koran, 1997) and spatial task performance (Kimura & Hampson, 1994). To account for these possible activational effects, female participants completed a short questionnaire assessing the date of onset of their last menstrual period, average length of cycle, menstrual irregularities, any accompanying symptoms in the

premenstrual period, and current or prior oral contraceptive/hormone use.

Vocabulary. A verbal test (Ekstrom & Stone, 1976) was given as a proxy measure of intelligence. Participants were presented with a stimulus word followed by several response words. They were asked to circle the word whose meaning was closest to the first and were given three minutes to complete the test.

Digit Ratio. Consistent with previous research (Manning et al., 2001), digit ratios were derived by measuring (on scanned copies of right and left hands) the lengths of fingers (index and ring finger) in millimeters from the basal crease to the tip of the finger. Two judges measured each hand copy using digital calipers, and an average measure was obtained across judges. As right hand digit ratio is believed to be a more sensitive measure of prenatal androgens, only right hand 2D:4D was examined in the analyses.

Salivary Samples. Research suggests that circulating levels of testosterone and estrogen may influence performance on sex-linked, spatial tasks (Kimura & Hampson, 1994; Silverman, Kastuk, Choi, & Phillips, 1999) and contribute to anxiety symptoms (Cameron et al, 1988; Cook et al., 1990; Granger et al., 2003; McLeod et al., 1993; Williams & Koran, 1997). Adult concentrations of testosterone also appear to have a negative correlation with the 2D:4D finger-length ratio, whereas estrogen appears to have a positive correlation (Manning, Scutt, Wilson, & Lewis-Jones, 1998). Given the above findings, this study obtained two saliva samples from each participant, one at the beginning of the session and one at the end. Time of sample collection was recorded to account for possible diurnal effects. Saliva samples were then collected in small vials,

stored at – 80° C, and shipped to Salimetrics LLC for testosterone (both males and females) and estrogen (only females) assays.

Gender Identity and Gender-Role Measures. Measures of gender identity and gender-role behavior included the Bem Sex-Role Inventory (BSRI; Bem, 1981a), the Pre-School Activities Inventory (PSAI; Golombok & Rust, 1993), the Occupation, Activities, and Traits – Attitudes and Personal Measures (OAT-AM & OAT-PM; Liben & Bigler, 2002), and the Draw-A-Person task (Zucker, Finegan, Doering, & Bradley, 1983). The BSRI consists of 60 items assessing masculinity and femininity as separate dimensions. Scores on these two scales may be used to classify individuals into one of four gender role categories: masculine-typed, feminine-typed, androgynous, and undifferentiated.

The PSAI is a 24-item measure assessing play preferences. Individuals describe how frequently they play with certain toys (i.e., "guns"), engage in specific activities (i.e., "playing house"), and possess several characteristics (i.e., "enjoys rough and tumble play"). Responses are given on a 5-point likert scale: N = "never", HE = "hardly ever", S = "sometimes", O = "often", and VO = "very often." Items are scored on a range from 1-5, with a higher score indicating male-typical play preferences and a lower score indicating female-typical play preferences. Although this questionnaire is designed for parents of children ages 2-5, it has been used in previous research with older populations to recall childhood play preferences. Participants were thus instructed to respond as they would have when they were 3-7 years old.

The OAT-AM is a 75-item questionnaire measuring gender attitudes towards

others. It consists of three sections, each with 25 items, asking participants to describe whether men, women, or both sexes should do certain jobs (i.e., "secretary", "plumber", "florist"), activities (i.e., "fix a car", "bake cookies", "go to the beach") or possess certain traits (i.e., "be emotional", "be cruel", "enjoy math"). The OAT-PM, which measures gender typing of the self, also has 75 total items and three 25-item subscales. Participants are asked to describe their own occupational interests, involvement in activities, and personality characteristics.

Finally, the Draw-A-Person task asks individuals to draw a person and then identify the sex of the figure. In previous research, individuals with gender dysphoria (Zucker et al., 1983) and individuals exposed to atypical hormone levels in prenatal life (Zucker, Bradley, Oliver, & Blake, 1996) are less likely to depict same-sex figures.

Psychopathology Measures. For a thorough assessment of anxiety symptoms, participants completed a battery of questionnaires including the Beck Anxiety Inventory (BAI; Beck & Steer, 1993), the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983), and the Anxiety Subscales of the Personality Assessment Inventory (PAI; Morey, 1991). The BAI consists of 21 items assessing somatic and affective symptoms of anxiety (i.e., "feeling hot," "fear of dying", "scared", "dizzy or lightheaded"). Responses are given on a scale from 0-3, with 0 representing no symptom presence and 1-3 representing increasing symptom levels. Scores on individual items are summed to provide an overall anxiety score.

The STAI is composed of 40 items and 2 scales, 20 items per scale. For the State Scale, participants are asked to describe how they are feeling "right now, that is, at this

moment." Responses are based on a 4-point scale ranging from 1 = "Not at all" to 4 = "Very Much So." On the Trait Scale, participants are prompted to describe how they "generally feel." Responses are based on a 4-point scale ranging from 1 = "Almost never" to 4 = "Almost Always." Half the items on each scale are scored in the positive direction and half in the opposite direction. Scores on the 20 items are summed to provide an overall scale score.

The PAI has three Anxiety Subscales (24 items total) measuring cognitive (ANX-C), affective (ANX-A), and physiological (ANX-P) components. Test-takers must choose whether symptoms are "Totally False," "Slightly True," "Mainly True," or "Very True" of them. Scores on each item are weighted on a scale from 0-3. Items in the ANX-C scale focus on ruminative worry and impaired concentration, whereas items in the ANX-A scale measure tension and fatigue caused by perceived stress. In addition, the ANX-P scale evaluates somatic symptoms of anxiety (i.e., "shortness of breath" and "trembling of hands"). The PAI also includes three Anxiety-related Disorders Scales (24 items total) measuring obsessive-compulsiveness (ARD-O), phobias (ARD-P), and traumatic stress (ARD-T). Responses are given on the same 4-point, weighted categories used in the Anxiety Subscales. The ARD-O scale contains items assessing inflexibility, perfectionism, and the presence of intrusive thoughts and behaviors, while the ARD-P scale evaluates fear for common objects and situations (i.e., "fear of heights" and "enclosed spaces"). Items on the ARD-T scale probe for a history of trauma and determine whether these events have reasonably altered the respondent's life, or are presently causing distress.

As a secondary interest, this study also examined two additional female-typical disorders: depression and borderline personality. Symptoms of depression were assessed through the Beck Depression Inventory (BDI; Beck & Steer, 1987) and the Depression Subscales of the PAI (PAI; Morey, 1991). Like the BAI, the BDI contains 21 items (i.e., "sadness", "self-dislike", "tiredness or fatigue") with a response scale from 0 to 3. Zero represents absence of symptom and 1-3 indicates increasing levels of the symptom. Scores on individual items are summed to provide a total depression score.

The PAI contains three depression subscales (24 items total) measuring cognitive (DEP-C), affective (DEP-A), and physiological (DEP-P) components. Items on the DEP-C scale focus on the individual's beliefs about worthlessness, inadequacy, and helplessness, whereas items on the DEP-A scale provide a general measure of life satisfaction and assess feelings of sadness and distress. Lastly, the DEP-P scale is concerned with the vegetative symptoms of depression, such as the loss or gain in appetite and sleep. Responses are provided using the same 4-point, weighted categories used for the anxiety scales (ANX and ARD).

Borderline Personality may represent an extreme form of normative gendered behavior (Skodol, 2000), which has previously been linked to prenatal sex hormones. The disorder's symptoms appear to worsen with oral contraceptive use and during the high-estrogen phase of the menstrual cycle (De Soto, Geary, Hoard, Sheldon, & Cooper, 2002). This activational effect is also suggestive of a prenatal sex hormone involvement. Accordingly, this study examined borderline symptoms using the Borderline Features Scale of the PAI (PAI-BOR). The PAI-BOR (24 items total) is sub-divided into four

subscales measuring affective instability (BOR-A), identity problems (BOR-I), negative relationships (BOR-N), and self-harm (BOR-S). Response scales consist of four weighted alternatives similar to the anxiety and depression scales, (ANX, ARD, and DEP), with scores on each item ranging from 0-3. The BOR-A scale measures emotional lability, whereas the BOR-I scale focuses on the individual's lack of purpose and self-concept. The BOR-N scale probes for a history of intense but unstable relationships, and the BOR-S scale examines impulsive behavior that could potentially result in self-destructive consequences.

It is also possible for mood state to be affected by completion of psychopathology measures and/or performance on spatial tasks. For a quick assessment of mood, the Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1992) was administered at the start and end of each experimental session. The POMS consists of 64 mood-related adjectives. Participants rate the applicability of each adjective to themselves using a 5-point scale ranging from 0 = "Not at all" to 4 = "Extremely." The first time the POMS-SF was administered, participants were asked to indicate how they had "been feeling during the past week including today." The second time it was given, participants were asked to rate how they "currently feel."

Sex-Linked Spatial Tasks. Participants completed two female-linked tasks (spatial location memory, spatial working memory) and two male-linked tasks (mental rotation, targeting) in counterbalanced order (see Appendix A for task examples). Memory for object locations was measured using the Silverman and Eals Location Memory Task (Silverman & Eals, 1992). A stimulus card with an array of 27 common objects (i.e.,

bird, flower, umbrella, iron, briefcase, teapot) was displayed for one minute followed by two response cards. The first response card, measuring object identity, displayed the 27 original objects plus 20 added objects. Participants were asked to indicate which objects were new or had been added. The second response card, measuring location memory, consisted of the 27 original objects. However, the positions of seven pairs of objects were exchanged. Participants were asked to indicate which objects had been moved. Response cards were displayed for a period of two minutes, or until the participant was finished. Performance on both the identity and location task was measured using the following formula: 1 – [(omissions + commissions)/N], where N equals the total number of objects.

Spatial working memory was assessed with a game, similar to the card game "Memory" (Duff & Hampson, 2001). A 5 x 4 array made of beige-colored felt was mounted on the wall at participant's eye level. Ten pairs of colored dots (green, yellow, blue, orange, brown, red, black, gray, purple, and pink) were dispersed throughout the array in random order. Dots were hidden beneath cutout flaps and could only be seen when participants lifted the flap. To show participants the possible range of colors, sample dots of each color were displayed in a column to the right of the array. Participants were instructed to match the pairs of colored dots as quickly as possible, but only turning over two flaps at a time. Before starting the task, the experimenter removed all sample dots from the side display. Every time a participant matched a pair of dots, the experimenter placed the sample dot of that color back on the side display. The latter allowed participants to keep track of colors they already matched and, hence, locations

they already searched. Performance on this task was recorded by a video camera and viewed at a later time for coding purposes. For each search attempt, experimenters recorded pairs of locations checked by participants using paper grids similar to the 5 x 4 array. Performance on this task was assessed by two dependent variables: total time required and total number of working memory errors (i.e., the number of times participants returned to already searched locations but did not produce a match plus the number of times they searched already matched locations).

Spatial rotation ability was assessed via the Mental Rotations Test (MRT-A; Peters et al., 1995). There are 24 items consisting of three-dimensional figures. For each item, a sample block design is given, along with four possible rotated representations of the design. The test-taker was asked to choose the two block patterns that matched the original figure. Participants were allowed three minutes to complete the first two pages, followed by a two-minute break, and then another three minutes for the last two pages. Performance on this task was based on the total number of correct items, those with both figures correctly identified.

To measure projectile throwing ability (Watson & Kimura, 1991), a target was constructed using a 36-inch-by-36-inch square of black felt. A bull's eye, made of white Velcro, was placed at the center of the square, 18 inches across and 18 inches vertically. Five ping-pong balls were also covered in Velcro, allowing them to adhere to the target upon contact. Participants were given ten opportunities to hit the bull's eye. Distance from the center was measured for each trial and averaged across the ten trials to yield a throw accuracy score. Higher scores indicate greater distances from the center, and thus

worse performance on targeting.

Procedure

Upon signing the consent form, participants were provided a cup of water and instructed to rinse their mouth. In accordance with saliva collection guidelines, the first salivary sample was collected 10 minutes after this procedure. Participants then continued to complete the measures described above in the following order: 1) salivary samples questionnaire 2) handedness questionnaire, 3) POMS, 4) menstrual cycle questionnaire (females only), 5) vocabulary, 6) Draw-A-Person, 7) BAI, 8) BDI, 9) PSAI, 10) PAI Anxiety, Depression, and Borderline Subscales, 11) OAT-PM and OAT-AM, 12) STAI, 13) BSRI, 14) female-linked tasks in randomized order (location memory and spatial memory), 15) male-linked tasks in randomized order (mental rotation and targeting), 16) second salivary sample, 17) POMS. Following completion of the experimental session, hands were scanned into a computer and printed in color. Experimenters then fully debriefed and thanked individuals for their participation. Each session lasted approximately 1 hour and 30 minutes.

RESULTS

Participants' Characteristics

A total of 110 participants (58 male; 52 female) participated in the present study, and 97 (54 male; 43 female) completed the entire protocol. However, some analyses are based on a smaller subset of participants, and these are listed where they occur. Women and men were comparable in age, ethnicity, and in performance on the vocabulary test, a proxy measure of general intelligence (see Table 2).

Preliminary Analysis

The behavioral data were first screened for violations of multivariate assumptions: normality, linearity, homogeneity of variance, and multicollinearity. Although several of the dependent variables were skewed in their distribution, multivariate analyses of variance (MANOVA) performed on both transformed and untransformed data were remarkably similar in the direction and significance of results. For that reason, only the results of the analyses of untransformed data are summarized below. First, the confirmatory analyses of previously reported sex differences and digit ratio effects on behavior are described, followed by the analyses of the novel, proposed associations between psychopathology and hormone levels.

Confirmatory Analyses of Hormone-Behavior Associations

Organizational Effects. Androgen levels in prenatal life are hypothesized to direct the sexual differentiation of behavior in human and nonhuman species and contribute to between-sex and within-sex differences in sex-linked behavior. In this research, the proxy measure of prenatal androgen levels (digit ratio) was lower in males

Table 2. Participants' Demographic Characteristics

	Men (n = 58)	Women (n = 52)
Age, yrs	19.67 (1.85)	20.69 (6.12)
Ethnicity		
Hispanic/Latino	17.2%	11.5%
Not Hispanic/Latino	81.0%	88.5%
No response	1.7%	0%
Race		
White/Caucasian	81.0%	84.6%
Black/African-American	5.2%	3.8%
Asian	3.4%	9.6%
American Indian/Alaska Native	0%	0%
No response	10.3%	1.9%
Vocabulary, number correct	24.05 (6.89)	23.58 (8.15)

(M = .96, SD = .03) compared to females (M = .97, SD = .03) consistent with higher prenatal androgen levels in male development. However, this sex difference in digit ratio did not reach statistical significance, $F(1, 108) = 2.096$, *ns*. To consider the contribution of prenatal androgen levels to within-sex variability in behavior, high (more feminine) versus low (more masculine) digit ratio groups within each sex were created by a median split**.** The resulting high digit ratio group consisted of 27 men and 19 women. The low digit ratio group consisted of 27 men and 24 women. Analyses of prenatal hormone effects on behavior were then evaluated using separate 2 x 2 MANOVAs, with sex (male, female) and the proxy measure of prenatal androgen levels, digit ratio (high, low), as grouping factors.

Tables 3 – 5 present the means and standard deviations for women and men on measures of psychopathology, spatial ability, gender role behavior, and hormone levels, replicating the previously reported sex differences in these measures. As expected, females generally reported higher levels of anxiety on the Anxiety-full scale (ANX) of the PAI, $F(1, 93) = 7.834$, $p < .01$, and all three of its component scales: cognitive (ANX-C), $F(1, 93) = 5.694$, $p < .025$, affective (ANX-A), $F(1, 93) = 9.726$, $p < .01$, and physical symptoms (ANX-P), $F(1, 93) = 4.812$, $p < .05$. A significant main effect of sex was also found on the Anxiety-Related Disorders – Phobias Subscale (ARD-P) of the PAI, $F(1, 93) = 7.007$, p =.01, and a trend towards significance was found for the Beck Anxiety Inventory (BAI), $F(1, 93) = 3.503$, $p < .10$. In contrast, no sex differences were found on state, $F(1, 93) = .004$, *ns*, or trait anxiety, $F(1, 93) = 1.273$, *ns*, and the Anxiety-Related Disorders Obsessions (ARD-O), $F(1, 93) = 1.391$, *ns*, and Trauma

Table 3. Mean Scores (*SD*) on Measures of Psychopathology in Women and Men

Task/ Measure		Sex Difference?	Means Men (n = 54)	Means Women (n = 43)	Effect Size
Anxiety Measures	ANX Scale	Yes**	45.61 (10.00)	51.22 (10.59)	*d* = .54
	ANX – Cognitions Subscale	Yes*	46.81 (9.95)	51.56 (10.99)	*d* = .45
	ANX – Affect Subscale	Yes**	43.96 (9.10)	49.61 (8.58)	*d* = .64
	ANX – Physical Sx Subscale	Yes*	47.68 (9.57)	52.15 (12.12)	*d* = .41
	STAI – State	No	1.56 (0.59)	1.55 (0.48)	*d* = .01
	STAI –Trait	No	1.73 (0.58)	1.84 (0.45)	*d* = .22
	BAI	No	7.63 (5.74)	10.26 (8.26)	*d* = .37
	ARD Scale	No	47.96 (9.89)	50.81 (10.06)	*d* = .29
	ARD – Obsessions Subscale	No	50.27 (9.42)	52.44 (10.21)	*d* = .22
	ARD – Phobias Subscale	Yes*	44.52 (9.66)	49.38 (9.47)	*d* = .51
	ARD-Traumas Subscale	No	49.92 (10.63)	49.83 (10.43)	*d* = .008
Borderline Measures	BOR Scale	No	48.85 (11.08)	50.27 (10.54)	*d* = .13
Depression Measures	BDI	No	7.93 (6.86)	9.26 (7.47)	*d* = .19
	DEP Scale	No	51.03 (12.35)	51.89 (11.96)	*d* = .07

Note. ANX = Anxiety Scale of the Personality Assessment Inventory; STAI = State-Trait Anxiety Inventory; BAI = Beck Anxiety Inventory; ARD = Anxiety-Related Disorders Scale of the Personality Assessment Inventory; BOR = Borderline Personality Scale of the Personality Assessment Inventory; BDI = Beck Depression Inventory; DEP = Depression Scale of the Personality Assessment Inventory. *$p < .05$. **$p < .01$.

Table 4. Mean Scores (*SD*) on Measures of Spatial Ability and Gender Role Behavior in Women and Men Confirming the Expected Sex Differences in Behavior

	Task/ Measure	Sex Difference?	Means		Effect Size
			Men	Women	
Spatial Tasks	Mental Rotation	Yes***	13.78 (4.61) n = 55	9.06 (4.33) n = 50	*d* = 1.06
	Targeting	Yes***	6.23 (1.78) n = 55	9.53 (2.77) n = 50	*d* = 1.41
	Spatial Memory - Errors	No	7.82 (9.71) n = 55	5.60 (6.58) n = 50	*d* = .27
	Location Memory	No	0.74 (0.10) n = 41	0.76 (0.11) n = 38	*d* = .18
Gender Role Measures	PSAI	Yes***	76.26 (9.11) n = 54	36.74 (15.60) n = 43	*d* = 3.09
	OAT-PM – Masculine Score	Yes***	2.43 (0.32) n = 54	2.12 (0.40) n = 43	*d* = .87
	OAT-PM Feminine Score	Yes***	2.00 (0.25) n = 54	2.46 (0.31) n = 43	*d* = 1.65
	OAT-AM	Yes***	0.15 (0.15) n = 54	0.05 (0.09) n = 43	*d* = .78
	BSRI – Feminine Scale	Yes***	4.71 (0.56) n = 54	5.27 (0.68) n = 43	*d* = .89
	BSRI – Masculine Scale	Yes*	5.53 (0.83) n = 54	5.16 (0.65) n = 43	*d* = .49

Note. PSAI = Pre-School Activities Inventory; OAT-PM = Occupations, Activities, Traits – Personal Measure; OAT-AM = Occupations, Activities, Traits – Attitude Measure; BSRI = Bem Sex-Role Inventory.
*$p < .05$. **$p < .01$. ***$p < .001$.

Table 5. Mean Scores (*SD*) on Measures of Hormone Levels in Women and Men

Task/ Measure		Sex Difference?	Means		Effect Size
			Men	Women	
Digit Ratio	Right 2D:4D	No	0.96 (0.03) n = 58	0.97 (0.03) n = 52	*d* =.28
	Left 2D:4D	No	0.96 (0.04) n = 58	0.96 (0.04) n = 52	*d* =.17
Salivary Testosterone (pg/mL)	Average Testosterone	Yes***	230.99 (85.49) n = 58	110.70 (51.05) n = 50	*d* = 1.71

Note. ***$p < .001$.

(ARD-S), $F(1, 93) = .008$, *ns*, Subscales of the PAI. Finally, males and females reported similar levels of depression, as measured by the Beck Depression Inventory (BDI), $F(1, 93) = .992$, *ns*, and the Depression Subscales (DEP), $F(1, 93) = .177$, *ns*, of the PAI: affective (DEP-A), $F(1, 93) = .025$, *ns*, cognitive (DEP-C), $F(1, 93) = .052$, *ns*, and physical symptoms (DEP-P), $F(1, 93) = 2.154$, *ns*. Males and females also did not differ on the Borderline Scale (BOR), $F(1, 93) = .579$, *ns*, and its component subscales: affective instability (BOR-A), $F(1, 93) = .114$, *ns*, identity conflict (BOR-I), $F(1, 93) = .315$, *ns*, negative relationships (BOR-N), $F(1, 93) = 2.737$, *ns*, and self-harm (BOR-S), $F(1, 93) = .205$, *ns*.

Consistent with previous research findings, males compared to females reported more "masculine" play and activities on all gender identity and gender role measures: PSAI, $F(1, 93) = 252.574$, $p < .001$, OAT-PM masculine scale, $F(1, 93) = 17.515$, $p < .001$, OAT-PM feminine scale, $F(1, 93) = 64.843$, $p < .001$, OAT-AM, $F(1, 93) = 13.018$, $p < .001$, BSRI feminine scale, $F(1, 93) = 18.402$, $p < .001$, and the BSRI masculine scale, $F(1, 93) = 5.506$, $p < .025$. Additionally, a large majority of males (49/54) drew male figures compared to female figures on the Draw-A-Person Task, whereas a smaller majority of females (24/43) drew female figures compared to male figures on that task ($\chi^2[df = 1] = 24.754$, $p < .001$). The analyses of spatial ability also showed the expected large male advantage in performance on the mental rotation task, $F(1, 101) = 29.320$, $p < .001$, and on the targeting task, $F(1, 101) = 52.871$, $p < .001$. However, this study failed to replicate sex differences on location memory, $F(1, 74) = .029$, *ns*, and spatial working memory, $F(1, 101) = 1.873$, *ns*, tasks which have

previously shown a slight to moderate female advantage (Alexander et al., 2002; Duff & Hampson, 2001; Eals & Silverman, 1994).

The main effect of digit ratio and the sex by digit ratio interaction effects showed no support for organizational effects of androgens on male-linked and female-linked spatial tasks. However, individuals with a lower (i.e., more masculine) digit ratio reported more male-typical play preferences on the PSAI ($M = 60.05$, $SD = 22.23$), whereas individuals with a higher (i.e., more feminine) digit ratio reported more female-typical play preferences ($M = 57.29$, $SD = 24.54$), $F(1, 93) = 4.359$, $p < .05$. Additionally, a significant interaction between sex and digit ratio groups emerged for the BSRI masculine scale, $F(1, 93) = 10.510$, $p < .01$, such that males with low digit ratio ($M = 5.84$, $SD = .54$) reported more male-typical traits as compared to males with high digit ratio ($M = 5.21$, $SD = .95$). The differences between females with low digit ratio ($M = 5.03$, $SD = .74$) and females with high digit ratio ($M = 5.34$, $SD = .47$) were non-significant.

Correlations between digit ratio and sex-linked behavior across women and men showed only a negative relation between digit ratio and PSAI scores ($r = -.230$, $p < .025$), such that a higher (i.e., more feminine) 2D:4D ratio was associated with lower (i.e., more feminine) scores on the PSAI. Within the group of men, more male-typical digit ratios were associated with more masculine-typical behavior as measured by the PSAI ($r = -.285$, $p < .05$), the BSRI ($r = -.345$, $p < .025$), and OAT-PM ($r = -.271$, $p < .05$). In females, a higher (i.e., more feminine) digit ratio was associated with more gender congruent behavior, as measured by the OAT-AM ($r = .309$, $p < .05$) but,

unexpectedly, higher digit ratio was also associated with better spatial ability, as measured by the mental rotation task ($r = .318$, $p < .025$).

Activational Effects. A repeated measures ANOVA showed no significant change in testosterone (male – $F(1, 57) = .931$, *ns*; female – $F(1, 49) = 1.118$, *ns*) or estradiol levels (female – $F(1,46) = 2.304$, *ns*) from time 1 to time 2. For that reason, average hormone levels were calculated and used as the measure of testosterone and estradiol in the analyses of activational effects on behavior. As expected, males had significantly higher levels of circulating testosterone than females, $F(1, 104) = 75.926$, $p < .001$. Sex differences in estradiol levels were not determined as only female samples were analyzed for this hormone. Additionally, the female sample included 14 oral contraceptive (OC)-users and 37 non-users. OC-users were not included in the analyses of activational effects because synthetic steroid levels are not measured by the salivary assay.

Bivariate correlations were conducted to examine previously reported associations between circulating hormone levels and spatial tasks, gender role behavior, and digit ratio. In general, male behavior did not appear sensitive to testosterone levels, as androgen levels were unrelated to spatial task performance or gender role measures. However, multiple associations were found in females who were not taking oral contraceptives. On spatial tasks, females with higher estrogen levels performed better on mental rotation ($r = .417$, $p < .025$) and committed less spatial memory errors than females with lower estrogen levels ($r = -.356$, $p < .05$). A negative correlation was also found between testosterone and performance on location memory, such that lower

testosterone levels were associated with better performance (-.534, $p < .01$). On gender role measures, testosterone correlated negatively to scores on the BSRI feminine scale ($r = -.450$, $p < .025$) and positively to the OAT-PM masculine scale ($r = .358$, $p < .05$), suggesting lower levels of testosterone among those with female-typical traits and higher levels among those with masculine-typical behavior. No relation was found between hormone levels and digit ratio.

Because previous researchers have reported menstrual cycle changes in mood, spatial task performance, and gender role behavior, female participants not using oral contraceptives were divided into three groups according to menstrual cycle phase: follicular (days 1-12 of cycle), ovulatory (days 12-16 of cycle), and luteal (days 17-highest). Separate MANOVAs with cycle phase as a factor were then conducted. As might be expected, females differed significantly in state anxiety across the menstrual cycle, $F(2, 28) = 3.757$, $p < .05$, $n = 31$. As shown by post-hoc tests, females in the luteal phase ($M = 1.87$, $SD = .60$, $n = 12$) reported significantly more state anxiety than females in the ovulatory phase ($M = 1.23$, $SD = .23$, $n = 7$). Furthermore, a significant effect was found across the menstrual cycle for targeting accuracy, $F(2, 32) = 3.936$, $p < .05$, $n = 35$. Unexpectedly, post-hoc tests show that females in the ovulatory phase ($M = 7.10$, $SD = 1.46$, $n = 7$) performed the best in targeting as compared to females in the follicular ($M = 9.88$, $SD = 1.92$, $n = 16$) and luteal phases ($M = 9.85$, $SD = 3.11$, $n = 12$). The small sample size, however, limits the interpretation of these results.

Analyses of Proposed Associations Among Gender, Hormone Levels and Psychopathology

Correlations Between Anxiety and Gender Role. Recall that anxiety was hypothesized to covary with gender role behavior, as previous research suggests that sexually dimorphic tasks share similar hormonal determinants. Correlations calculated between anxiety measures and the four sex-linked spatial tasks were significant only for the two abilities that typically show a male advantage, (i.e., mental rotation and targeting). Higher scores (i.e., better performance) on the mental rotation task were associated with lower levels of anxiety as measured by the STAI-trait (r = -.192, p < .05), the ANX-full scale (r = -.328, p = .001), ANX-C (r = -.311, p < .01), ANX-A (r = -.378, p < .001), ANX-P (r = -.203, p < .05), and ARD-P (r = -.284, p < .01). Similarly, lower scores on targeting (i.e., better targeting accuracy) were associated with lower levels of anxiety as measured by the STAI-trait (r = .213, p < .05), ANX-full scale (r = .225, p < .05), ANX-C (r = .238, p < .025), and ANX-A (r = .245, p < .025).

Anxiety levels also covaried with other measures of gender role behavior, such that more female-typical behavior on the PSAI was associated with higher levels of anxiety as measured by the BAI (r = -.207, p < .05), ANX-full scale (r = -.306, p < .01), ANX-C (r = -.278, p < .01), ANX-A (r = -.316, p < .01), ANX-P (r = -.244, p < .025), and ARD-P (r = -.298, p < .01). Similarly, higher scores on the BSRI masculine scale were associated with lower anxiety levels as measured by the STAI-trait (r = -.310, p < .01), ANX-full scale (r = .250, p < .025), ANX-C (r = -.208, p < .05), ANX-A (r = -.302, p < .01), and ARD-P (r = -.323, p = .001). Finally, higher female-typical scores on the

OAT-PM feminine scale were associated with increased anxiety levels as measured by the ARD-full scale (r = .214, p < .05) and ARD-O (r = .313, p < .01), whereas higher scores on the BSRI feminine scale were associated with greater levels of anxiety on ARD-P (r = .245, p < .025).

Organizational Effects. A series of MANOVAs, with sex and digit ratio as factors, were also conducted separately on anxiety, borderline personality, and depression measures. Results from these analyses are summarized in Figures 1-5. A significant main effect of digit ratio was found on STAI-trait, $F(1, 93) = 4.587$, $p < .05$, and ARD-P, $F(1, 93) = 4.235$, $p < .05$. Regardless of sex, individuals with a high 2D:4D reported greater trait anxiety and phobia symptoms (M = 1.90, SD = .53; M = 48.57, SD = 9.97, respectively) than individuals with a low 2D:4D (M = 1.67, SD = .50; M = 44.96, SD = 9.47, respectively). A difference between high and low digit ratio groups also approached significance on a third measure, ANX-C, $F(1, 93) = 3.409$, $p < .10$, such that individuals with high digit ratio reported greater cognitive symptoms of anxiety (M = 50.73, SD = 11.03) than individuals with a low digit ratio (M = 47.27, SD = 10.09).

A significant main effect of digit ratio was also found for the BOR-full scale, $F(1, 93) = 4.356$, $p < .05$, and the BOR-A subscale, $F(1, 93) = 9.371$, $p < .01$, of the PAI. Individuals with a high 2D:4D reported greater borderline personality characteristics overall and greater affective instability, in particular, (M = 51.86, SD = 12.01; M = 52.70, SD = 11.57, respectively) than did individuals with a low 2D:4D (M = 47.33, SD = 9.21; M = 46.15, SD = 9.21, respectively). A trend towards significance was additionally found for the BOR-N subscale, $F(1,93) = 3.530$, $p < .10$. Those individuals

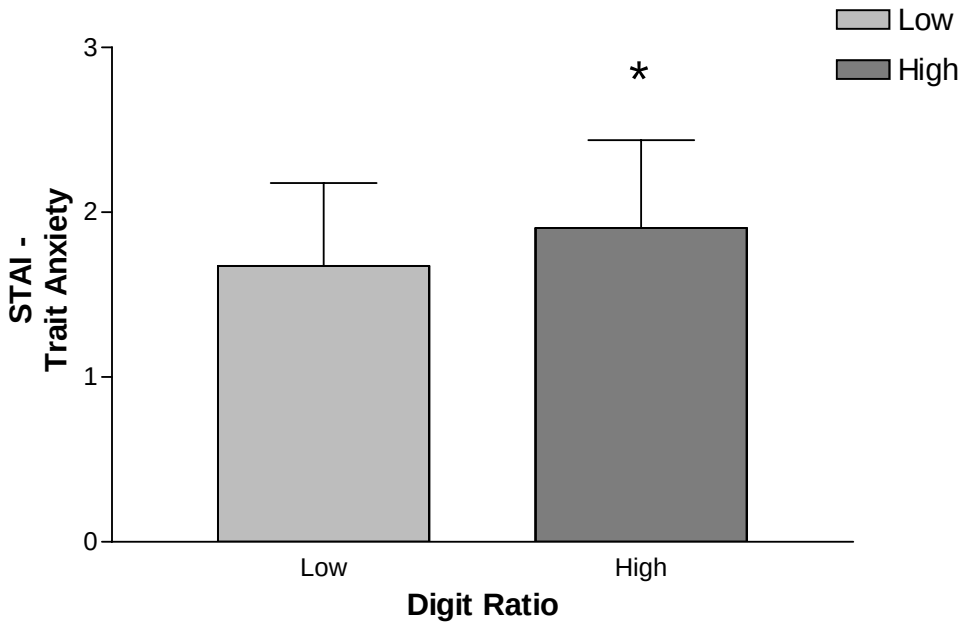

Fig. 1. Mean scores (+*SD*) on the State-Trait Anxiety Inventory-Trait Scale for low digit ratio ($n = 51$) and high digit ratio ($n = 46$) groups. *$p < .05$.

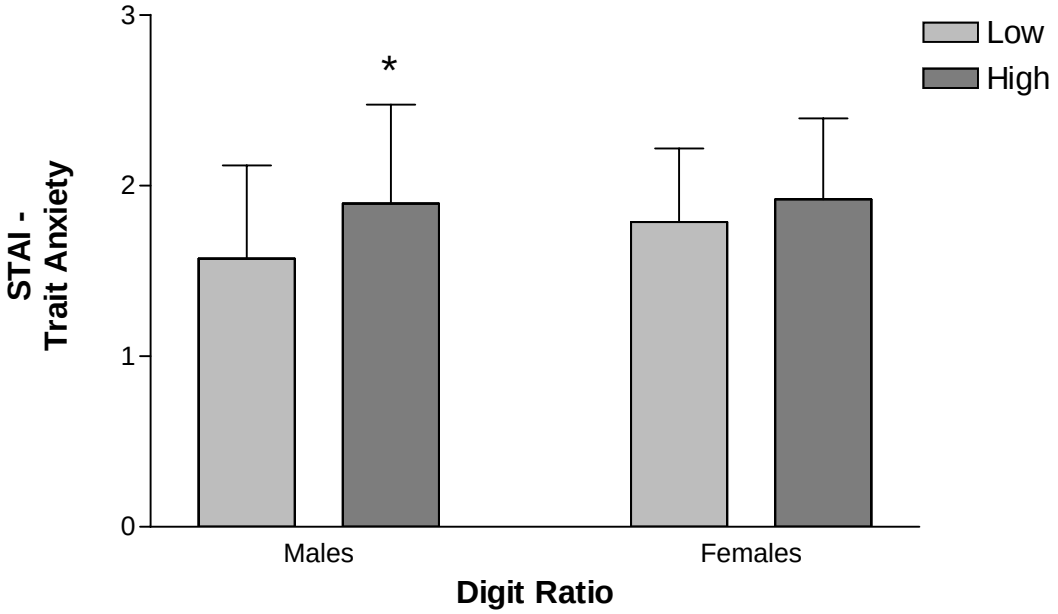

Fig. 2. Mean scores (+*SD*) on the State-Trait Anxiety Inventory-Trait Scale for males with low (n = 27) and high (n = 27) digit ratios and females with low (n = 24) and high (n = 19) digit ratios. *$p < .05$.

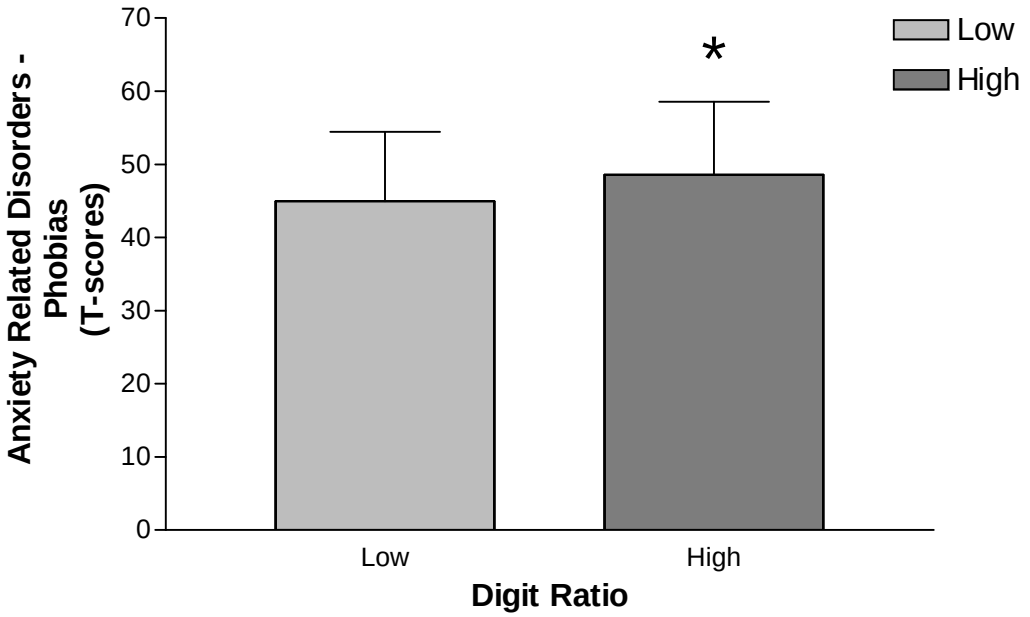

Fig. 3. Mean scores (+*SD*) on the Anxiety-Related Disorders-Phobias Subscale of the Personality Assessment Inventory for low digit ratio ($n = 51$) and high digit ratio ($n = 46$) groups. *$p < .05$.

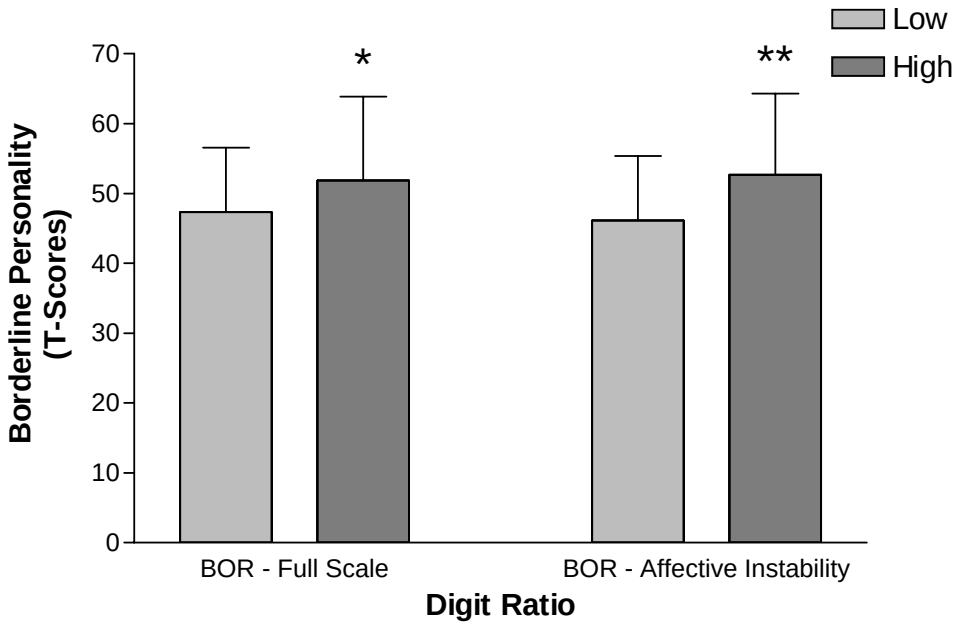

Fig. 4. Mean scores (+*SD*) on the Borderline Personality Scale of the Personality Assessment Inventory for low digit ratio ($n = 51$) and high digit ratio ($n = 46$) groups. *$p < .05$. **$p < .01$.

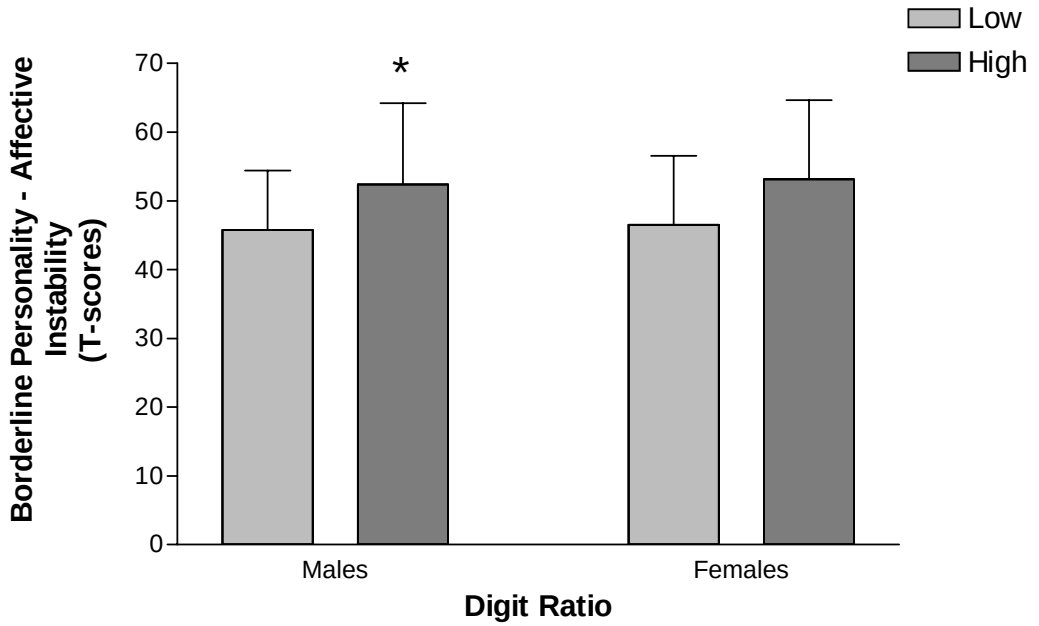

Fig. 5. Mean scores (+*SD*) on the Borderline Personality-Affective Instability Subscale of the Personality Assessment Inventory for males with low ($n = 27$) and high ($n = 27$) digit ratios and females with low ($n = 24$) and high ($n = 19$) digit ratios. *$p < .05$.

with a high digit ratio (M = 51.92, SD = 12.74) tended to report greater negative relationship symptoms than those with a low digit ratio (M = 47.91, SD = 9.71).

No significant differences in high versus low ratio groups were found for the depression measures, though differences appeared to approach significance on the DEP-A subscale of the PAI, $F(1, 93) = 3.159$, $p < .10$. Across sex, individuals having a high 2D:4D (M = 52.99, SD = 14.80) reported greater affective symptoms of depression than individuals with a low digit ratio (M = 48.34, SD = 10.81).

Within-sex analyses using MANOVA with digit ratio as a factor revealed a significant difference in STAI-trait, $F(1, 52) = 4.420$, $p < .05$, for males and a close to significant difference in ARD-full scale, $F(1, 41) = 3.877$, $p < .10$, for females. Males with a high digit ratio reported higher levels of trait anxiety (M = 1.89, SD = .58) than males with a low digit ratio (M = 1.57, SD = .55) and females with a high digit ratio tended to report greater symptoms of anxiety disorders (M = 54.09, SD = 10.47) than females with low digit ratio (M = 48.21, SD = 9.10).

Similarly, a within-sex MANOVA with digit ratio as a factor revealed a significant difference in the BOR-A subscale for males, $F(1, 52) = 5.517$, $p < .025$, and a similar non-significant trend for females, $F(1, 41) = 4.009$, $p < .10$. Males (M = 52.40, SD = 11.81) and females (M = 53.12, SD = 11.53) with a high digit ratio endorsed greater affective instability than males (M = 45.80, SD = 8.61) and females (M = 46.54, SD = 10.02) with a low digit ratio. Further analyses of depressive symptomatology within males and females revealed no differences between high and low 2D:4D groups.

Bivariate correlations between the continuous measure of digit ratio and

psychopathology measures across sex showed that digit ratio was positively associated with trait anxiety ($r = .202$, $p < .05$) on the STAI and with both the BOR-full scale ($r = .206$, $p < .05$) and the BOR-A subscale ($r = .268$, $p < .01$) of the PAI. Individuals with a larger (i.e., more feminine) digit ratio tended to report greater anxiety and borderline symptoms on these measures. However, within-sex correlations found a positive relation between digit ratio and STAI-trait ($r = .260$, $p < .05$) and between digit ratio and BOR-A ($r = .299$, $p < .05$) only for males. Men with larger (i.e., more feminine) digit ratios reported greater trait anxiety and affective instability than men with smaller (i.e., more masculine) digit ratios. Females' 2D:4D ratios were not significantly associated to any measure of psychopathology.

Activational Effects. Bivariate correlations reveal no significant association between circulating hormone levels and measures of psychopathology. Average testosterone and estradiol levels appear unrelated to anxiety, depression, and borderline symptoms in males and in females not taking oral contraceptives. Furthermore, as previous studies have shown a worsening of psychopathology across the menstrual cycle, separate MANOVAs with cycle phase as a factor were conducted for the anxiety, depression, and borderline scales of the PAI. No differences were found between females in the follicular, ovulatory, and luteal phases on any of these measures. Table 6 provides a summary of these results.

Regression Analyses. To test the relative contributions of sex, gender role measures, spatial task performance, and hormonal levels to anxiety and borderline personality, three separate hierarchichal regression analyses were conducted. The ANX-

Table 6. Mean Scores (*SD*) on the Anxiety, Depression, and Borderline Personality Subscales of the Personality Assessment Inventory by Menstrual Cycle Phase in Females Not Taking Oral Contraceptives

	Follicular (*n* = 12)	Ovulatory (*n* = 7)	Luteal (*n* = 12)
ANX Scale	55.53 (11.47)	48.72 (13.04)	50.20 (11.17)
ARD Scale	55.37 (11.15)	51.40 (10.23)	48.54 (9.44)
DEP Scale	54.86 (12.31)	52.24 (8.56)	55.43 (15.59)
BOR Scale	53.05 (11.40)	47.86 (13.16)	52.25 (9.89)

Note. ANX = Anxiety Scale of the Personality Assessment Inventory; ARD = Anxiety-Related Disorders Scale of the Personality Assessment Inventory; DEP = Depression Scale of the Personality Assessment Inventory; BOR = Borderline Scale of the Personality Assessment Inventory. Follicular = day 1-12 of cycle; Ovulatory = day 12-16 of cycle; Luteal = day 17-highest.

full scale, ARD-full scale, and the BOR-full scale served as the dependent variables. For each model, the BSRI masculine and feminine scales were entered at the first step, mental rotation and targeting scores were entered at the second step, digit ratio and testosterone levels were entered at the third step, and sex was entered at the fourth step. Hormone measures and sex were entered last to determine if they added any significant contribution beyond gender role behavior and spatial task performance.

Results for the ANX-full scale regression analysis are provided in Table 7. For this scale, inclusion of the BSRI masculine and feminine scales at the first step produced a significant $F_{change,}$ $F_{change} = 3.865$, $R^2 = .078$, $p < .025$. Scores on the BSRI accounted for 7.8% of the variance in anxiety as measured by this scale. At the second step, inclusion of the mental rotation and targeting scores also yielded a significant $F_{change,}$ $F_{change} = 4.870$, $R^2 = .168$, $p = .01$. Male-linked tasks contributed an additional 9% of the variance in ANX-full scale scores. Lastly, hormonal measures, $F_{change} = .349$, $R^2 = .174$, *ns*, and sex, $F_{change} = .155$, $R^2 = .176$, *ns*, did not appear to add significantly to the model. Examination of the beta weight sizes in the final model suggest that the BSRI masculine scale, $\beta = -.22$, $p < .05$, and mental rotation, $\beta = -.25$, $p < .05$, are the best predictors of anxiety. In contrast, the variance in ARD-full scale and the BOR-full scale could not be significantly accounted by any of the four models or predictor variables.

As this study found mean differences between low and high digit ratio groups on the BOR-A subscale, an additional regression analysis was conducted with BOR-A as the dependent variable. Independent variables were entered in the same four-step fashion as above, and a summary of results is provided in Table 8. At the first step, the

Table 7. Hierarchical Regression Analysis Exploring the Contribution of Gender Role Measures, Spatial Ability, Hormone Levels, and Sex to Scores on the Anxiety Scale of the PAI

Step and Independent Variable	*B*	*SE B*	*β*
Step 1			
BSRI masculine scale	-3.53	1.38	-.26*
BSRI feminine scale	1.41	1.59	.09
Step 2			
BSRI masculine scale	-2.97	1.33	-.22*
BSRI feminine scale	.07	1.60	.005
Mental Rotation	-.54	.23	-.25*
Targeting	.44	.42	.11
Step 3			
BSRI masculine scale	-3.05	1.40	-.22*
BSRI feminine scale	.31	1.65	.02
Mental Rotation	-.57	.24	-.27*
Targeting	.51	.46	.13
2D:4D	13.30	31.77	.04
Average Testosterone	.01	.01	.08
Step 4			
BSRI masculine scale	-2.98	1.42	-.22*
BSRI feminine scale	.16	1.71	.01
Mental Rotation	-.54	.25	-.25*
Targeting	.43	.50	.11
2D:4D	13.20	31.92	.04
Average Testosterone	.01	.02	.10
Sex	1.38	3.51	.07

Note. BSRI = Bem Sex-Role Inventory.
R^2 = .078 for Step 1 ($p < .025$); ΔR^2 = .090 for Step 2 ($p = .01$);
ΔR^2 = .007 for Step 3 (*ns*); ΔR^2 = .001 for Step 4 (*ns*).
*$p < .05$.

Table 8. Hierarchical Regression Analysis Exploring the Contribution of Gender Role Measures, Spatial Ability, Hormone Levels, and Sex to Scores on the Borderline Personality-Affective Instability Subscale of the PAI

Step and Independent Variable	*B*	*SE B*	*β*
Step 1			
BSRI masculine scale	-1.46	1.44	-.10
BSRI feminine scale	-2.42	1.66	-.15
Step 2			
BSRI masculine scale	-1.15	1.45	-.08
BSRI feminine scale	-3.25	1.74	-.20
Mental Rotation	-.26	.25	-.12
Targeting	.37	.46	.09
Step 3			
BSRI masculine scale	-.53	1.48	-.04
BSRI feminine scale	-3.28	1.74	-.20
Mental Rotation	-.29	.25	-.13
Targeting	.20	.48	.05
2D:4D	78.84	33.56	.24*
Average Testosterone	.00	.02	-.004
Step 4			
BSRI masculine scale	-.59	1.50	-.04
BSRI feminine scale	-3.15	1.80	-.20
Mental Rotation	-.31	.26	-.14
Targeting	.27	.53	.07
2D:4D	78.92	33.73	.24*
Average Testosterone	-.003	.02	-.02
Sex	-1.13	3.71	-.05

Note. BSRI = Bem Sex-Role Inventory.
R^2 = .031 for Step 1 (*ns*); ΔR^2 = .029 for Step 2 (*ns*); ΔR^2 = .056 for Step 3 ($p < .10$); ΔR^2 = .001 for Step 4 (*ns*).
*$p < .05$.

BSRI masculine and feminine scales accounted for a small but non-significant portion of the variance in affective instability scores, $F_{change} = 1.482$, $R^2 = .031$, *ns*. At the second step, addition of mental rotation and targeting performance did not significantly improve the model, $F_{change} = 1.382$, $R^2 = .060$, ns. However, inclusion of hormonal measures at the third step produced a near-significant F_{change}, $F_{change} = 2.780$, $R^2 = .116$, $p < .10$. Examination of individual beta weights suggests that digit ratio, $\beta = .244$, $p < .025$, is significantly contributing to the variance in BOR-A scores. Finally, the addition of sex at the fourth step did not significantly improve the model, $F_{change} = .093$, $R^2 = .117$, *ns*.

DISCUSSION

The results of the present study provide the first evidence that levels of androgens during prenatal life may influence the development of anxiety disorders, typically female-linked conditions. Consistent with the primary hypothesis, a negative association was found between adult levels of anxiety and prenatal androgen levels as measured by both phenotypic markers (2D:4D) and behavioral correlates (i.e., gender role and spatial abilities). In general, individuals with a higher digit ratio (i.e., more feminine or lower levels of prenatal androgens) reported greater levels of trait anxiety and phobic symptoms and a trend for greater cognitive anxiety symptoms than individuals with a lower digit ratio. This novel finding is consistent with a previous report of an association between higher 2D:4D and neuroticism in adults (Fink et al., 2004).

The association between 2D:4D and anxiety in this research suggests further that the relationship between androgens and anxiety is strongest in males. Specifically, men with 2D:4D indicating low androgen levels in prenatal life reported greater trait anxiety than men with 2D:4D indicating a high androgen environment. Results are consistent with findings of an association between 2D:4D and anxious behavior in preschool boys (Williams et al., 2003) and with more feminized anxious behavior in castrated male rats (Lucion et al., 1996). Only a non-significant trend emerged in females, such that women with lower prenatal androgen levels (higher 2D:4D) endorsed greater symptoms of anxiety disorders than women with higher prenatal androgen levels (lower 2D:4D). This pattern of results suggests a protective role for prenatal androgens, particularly in males,

such that a “low androgen” environment in prenatal life is associated with anxiety vulnerability and a “high androgen” environment is protective against anxiety symptoms.

In addition to the association between anxiety and 2D:4D, a relation was found between anxiety and male-linked, spatial tasks. Individuals who performed better (i.e., more male-typical) on measures of mental rotation and targeting reported less anxiety than those who performed more poorly. One possible explanation for this association is that increased anxiety impairs task performance. Several other findings argue against this interpretation. First, higher anxiety levels were not associated with poorer performance on female-typical tasks (i.e., location memory and spatial working memory). Further, measures of state anxiety were also unrelated to performance on male-typical tasks. A second possible explanation is that perceived failure or success on the tasks themselves may have produced changes in mood. However, the design of the study precludes this interpretation as psychopathology measures were completed prior to spatial task performance. Rather, it is believed that as sexually dimorphic behaviors, both anxiety and spatial tasks are organized by prenatal androgens. As anxiety, location memory, and spatial memory are all female-linked behaviors, they are theorized to result from a low androgen environment. Conversely, a high androgen environment is credited for the male advantage in mental rotation and targeting ability. Given the direction of hormone-behavior associations, anxiety should correlate positively with other female-linked behaviors (i.e., location memory and spatial memory) and negatively with male-linked behaviors (i.e., mental rotation and targeting). Evidence for the latter was found

in this study.

Correlations were also found between anxiety and gender role behavior, and unlike results for spatial abilities, these were found for both the feminine and masculine gender role. Masculine play preferences and gendered behavior were associated with lower trait anxiety, decreased symptoms on all three components of anxiety (cognitive, affective, and physical), and less phobic symptoms. Conversely, more feminine-typed individuals reported greater anxiety disorder symptoms, specifically in relation to obsessions, compulsions, and phobias. Results are consistent with the theory that both masculinity and femininity are contributors to anxiety. While the masculine gender role's emphasis on instrumental traits may be protective of anxiety (Arrindell et al., 1993; Chambless & Mason, 1986; Eisler et al., 1988; Ginsburg & Silverman, 2000), the feminine gender role's reliance on expressive traits may be conducive to this condition (Fodor, 1974; Perez Blasco & Serra Desfilis, 1997).

Regression analyses provided further evidence of the relation between anxiety and behavioral correlates of prenatal androgen levels. Male-typical behavior and spatial ability emerged as the best explanation of anxiety, such that higher scores on the BSRI masculine scale and better performance on mental rotation were associated with decreased anxiety symptoms. Interestingly, hormonal measures and sex did not add significantly to the explanation of anxiety beyond the variables mentioned. The absence of a direct contribution by testosterone levels and the proxy measure of prenatal androgens (2D:4D), however, does not preclude an organizational and activational effect on anxiety. It is likely that the prenatal androgen contribution and the contribution by

sex were subsumed by the gender role measures and spatial tasks, as these have been strongly linked to prenatal androgens (Berenbaum & Hines, 1992; Hampson et al., 1998; Leveroni & Berenbaum, 1998; Meyer-Bahlburg et al., 2004; Servin et al., 2003; Udry, 2000; Udry et al., 1995).

In view of the novel findings of this research, it is important to note that this study also replicated previously reported sex differences in behavior. As reported in epidemiological literature (Bijl et al., 2002; Halbreich, 2003; Pigott, 1999; Pigott, 2003; Reich, 1986; Shear et al., 2000; Weissman, 1988) and studies of non-clinical populations (Armstrong & Khawaja, 2002), females in this study reported significantly more anxiety symptoms than males. The present study also replicated sex differences in gender identity and gender role behavior (i.e., play preferences) and supported the typical male advantage on mental rotation and targeting (Linn & Petersen, 1985; Watson & Kimura, 1991). Contrary to previous findings, however, (Alexander et al., 2002; Duff & Hampson, 2001; Eals & Silverman, 1994), no evidence was found for a female advantage on spatial working memory and location memory. As expected, testosterone levels were significantly higher in men than in women, and as with previous research (Brown et al., 2002; Rahman & Wilson, 2003), males had a lower mean digit ratio than females, though the latter did not reach statistical significance.

The present findings of hormone-behavior associations were also generally consistent with the results of previous investigations. For instance, the present study did not find support for a negative association between 2D:4D and performance on male-typical spatial tasks. This is inconsistent with some research on 2D:4D showing a

negative correlation with mental rotation in men (Manning, 2002) but consistent with the lack of association between 2D:4D and spatial tasks found by other researchers (Coolican & Peters, 2003). Furthermore, gender role has been linked in the literature to prenatal androgen levels (Berenbaum & Hines, 1992; Leveroni & Berenbaum, 1998; Meyer-Bahlburg et al., 2004; Servin et al., 2003; Udry, 2000; Udry et al., 1995) and to 2D:4D, in particular (Csatho et al., 2003). Similarly, this study found play preferences to be strongly related to 2D:4D, such that individuals with low (i.e., more masculine) digit ratios reported more masculine play preferences than individuals with high (i.e., more feminine) digit ratios. Additionally, males with a low digit ratio tended to report more male-typical traits and occupation and activity preferences than males with a high digit ratio, whereas females with a high digit ratio reported more gender-typed attitudes than females with a low digit ratio.

Hormone levels in this study also appear to have activational effects on sexually dimorphic behavior, as previously found in the literature. In contrast with the organizational effects, which were more evident in males, the effects of circulating hormones on behavior were exclusive to females. Testosterone levels were not associated with gender role behavior, spatial task performance, or 2D:4D ratio in males. However, estrogen and testosterone associations were found in females who were not taking oral contraceptives. As expected, females with higher estrogen levels performed better on spatial working memory as compared to those with lower estrogen levels. This result is consistent with research showing that female performance on female-linked tasks is best during the ovulatory and mid-luteal phases, times of increased estrogen

levels (Kimura & Hampson, 1994). Contrary to predictions, however, females with higher estrogen levels also had better performance on mental rotation, a task traditionally showing a male advantage. This finding is at odds with menstrual cycle studies showing decreased performance on male-linked tasks during times of high estrogen (Kimura & Hampson, 1994). In addition, higher testosterone levels in females were associated with worsened performance on location memory and more male-typical traits, activities, and occupations. No relation was found between circulating estrogen and testosterone levels and 2D:4D ratio in females, adding to the mixed pattern of results in the literature. Although some studies have found a negative association between testosterone and digit ratio in men and a positive association with estrogen in both sexes (Manning et al., 1998), other studies find no relation between circulating hormones and 2D:4D (Neave et al., 2003).

To evaluate activational effects across the menstrual cycle, this study also compared the behavior of females found in three different phases: follicular, ovulatory, and luteal. Only females who were not taking oral contraceptives were included in these analyses. Consistent with retrospective studies showing an increase in anxiety symptoms during the premenstrual phase (Cameron et al., 1988; Cook et al., 1990; McLeod et al., 1993; Williams & Koran, 1997) females in the luteal/premenstrual phase (days 17-highest) reported the highest levels of state anxiety. In addition, females in the ovulatory phase, a phase normally associated with hindered performance on male-linked tasks (Kimura & Hampson, 1994), unexpectedly obtained the best targeting accuracy.

As an extension of the primary research question, this study also explored the

relation between prenatal androgens and two other female-linked disorders: depression and borderline personality disorder. Although the literature reports a female vulnerability for these two disorders (American Psychiatric Association, 2000), this study found no significant difference between males and females in reports of depressive or borderline symptoms, consistent with normalization data from the PAI (Morey, 1991). Furthermore, the relation between 2D:4D and depression has yielded inconsistent results in the literature. While previous studies find no relation between 2D:4D and depression (Martin et al., 1999), recent findings suggest that men with higher digit ratios are more prone to a depressive personality style (Bailey & Hurd, 2005). In support of the former, this study failed to find a significant relation between 2D:4D and depression. It should be noted, however, that the depressive measures used in this study were designed to assess clinical, rather than trait depression.

In contrast, results suggest a novel association between 2D:4D and borderline personality characteristics, affective instability in particular. In fact, regression analyses reveal 2D:4D as the only significant predictor for the affective instability component of borderline personality. Individuals with a higher (i.e., more feminine) digit ratio reported greater irritability and shifts in mood than individuals with a lower (i.e., more masculine) digit ratio. Like with anxiety, this effect was most prominent in males, suggesting that prenatal androgen effects on female-linked psychopathology may be more sensitive in men than in women. Men with low prenatal androgen levels appear more vulnerable to borderline personality, while men with high prenatal androgen levels seem to be protected. Given the relation between 2D:4D and borderline personality, activational

effects of circulating hormone levels were also expected. This hypothesis, however, was not confirmed. Contrary to previous research (DeSoto et al., 2003), borderline symptoms did not appear sensitive to estrogen and testosterone levels and did not seem to fluctuate across the menstrual cycle.

Though the present study has many strengths, such as the comprehensive measurement of anxiety through a broad array of measures, there were a number of limitations that may have influenced the general pattern of results. One limitation of this study is the use of scanned copies to derive 2D:4D measurements. The quality of the images may have distorted the accuracy of measurements leading to a non-significant sex difference in 2D:4D. Future studies should incorporate direct measurements of the hand, as digit ratio measurements from hand copies may differ markedly from direct measurements (Manning, Fink, Neave, & Caswell, 2005). Additionally, this study included both women who were and were not taking oral contraceptives, and personality and hormonal factors associated with the use of hormonal contraceptives may influence the magnitude of some hormone-behavior associations. Finally, the large number of analyses and measures in this exploratory study may have yielded some illusory associations. Future studies might benefit from including a smaller subset of variables and statistical analyses, or using a larger number of participants to gain statistical power.

SUMMARY AND CONCLUSIONS

Despite its limitations, this study has replicated many of the common sex differences in behavior and provided further evidence for both organizational and activational hormone-behavior associations, particularly in the areas of spatial ability and gender role behavior. More importantly, it has provided the first evidence that two female-linked disorders, anxiety and borderline personality, appear sensitive to the organizational effects of sex hormones. Like previously studied male-typical (i.e., Tourette's and autism) and female-typical (i.e., depression) psychopathology, (Alexander & Peterson, 2004; Bailey & Hurd, 2005; Manning et al. 2001), the expression of anxiety and borderline personality symptoms appears susceptible to levels of prenatal androgens. Lower levels of prenatal androgens, such as those that occur in female development, may predispose individuals to anxiety and to borderline characteristics, such as affective instability, whereas higher levels of androgens appear to serve as a protective element against these two female disorders.

Interestingly, organizational effects in this study appeared to be more prominent in males whereas activational effects were only evident in females. This pattern of results lends itself to two primary explanations. First, women may be more sensitive to fluctuations in adult sex hormones because as compared to men they experience greater fluctuations in hormone levels, for example across the menstrual cycle. Second, organizational (i.e., 2D:4D) effects in relation to female-linked disorders may be easier to detect in men than in women, as women may be already predisposed towards these disorders. Additionally, reports of psychopathology in women may be confounded by

activational hormone influences, such as the menstrual cycle and oral contraceptive use, making it more difficult to partial out the effects of prenatal and postnatal hormones. Nevertheless, it should be noted that though non-significant, there was a trend for females with higher 2D:4D to report greater borderline personality-affective instability symptoms and greater anxiety disorder symptoms in general. It should also be noted that a smaller number of females than males were included in this study. Perhaps if more females were included, organizational effects would have been equally found across men and women. Despite being more prominent in males, the general results of this research suggest that prenatal androgens may play a significant role in the development of anxiety and borderline personality, in addition to the contribution of genetics (Hettema et al., 2001; Kendler et al., 1995; Silove et al., 1995; Stein et al., 1999), neurotransmitters (Brawman-Mintzer & Lydiard, 1997; Charney et al., 1993; Nutt, 2000; Nutt et al., 1998; Pigott 2003), and gender role socialization (Arrindell et al., 1993; Chambless & Mason, 1986; Eisler et al., 1988; Ginsburg & Silverman, 2000; Perez Blasco & Serra Desfilis, 1997; Thornton & Leo, 1992). Further research is needed to confirm our findings and extend them to a clinical sample.

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APPENDIX A

EXAMPLES OF SEX-LINKED SPATIAL TASKS

Fig. A-1. Stimulus Card for Location Memory Task

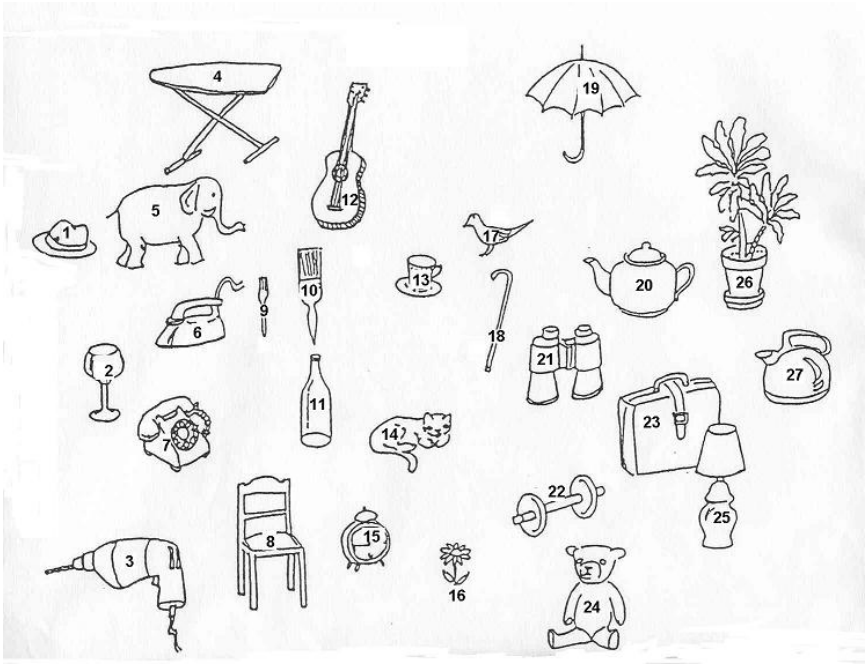

Fig. A-2. Response Card with Exchanged Objects

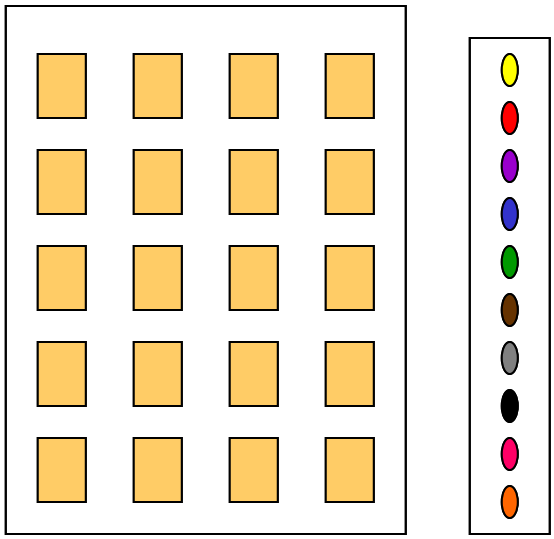

Fig. A-3. Diagram Representation of Spatial Working Memory Task

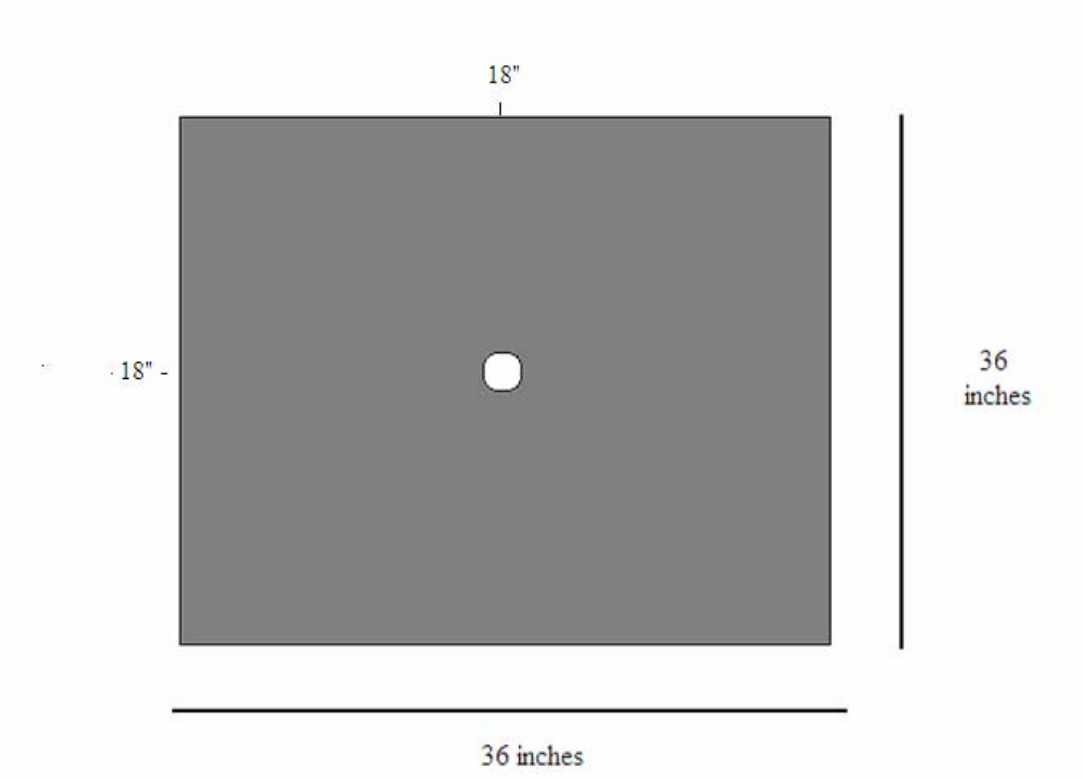

Fig. A-4. Diagram Representation of Targeting Task

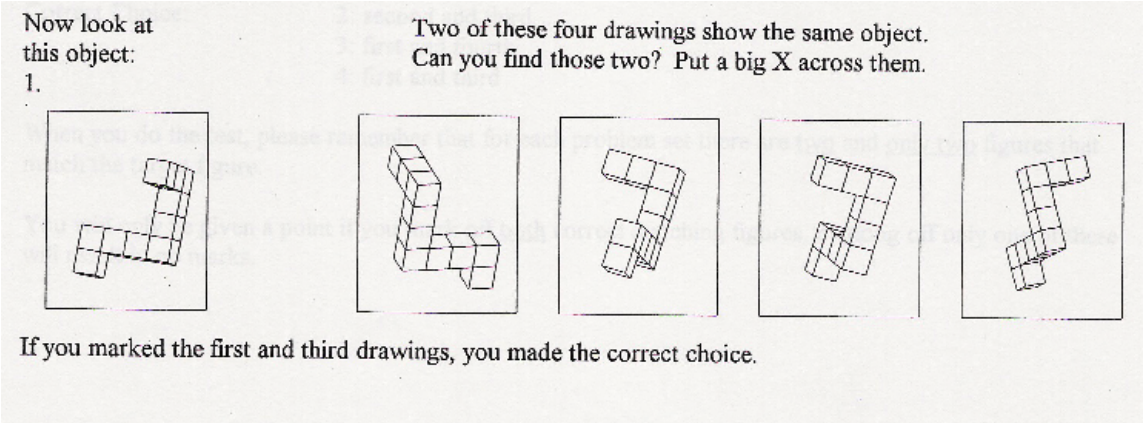

Fig. A-5. Sample Mental Rotation Problem

APPENDIX B

CORRELATION MATRIX – DIGIT RATIO AND PSYCHOPATHOLOGY MEASURES

		Digit Ratio	BDI	BAI	STAI-State	STAI-Trait	ANX	ANX-C	ANX-A
Digit Ratio	**Pearson**	1	.107	.002	.112	.202*	.126	.162	.074
	Sig.	.	.267	.984	.246	.034	.219	.113	.473
	N	110	110	97	110	110	97	97	97
BDI	**Pearson**	.107	1	.806	.626	.720*	.682*	.607*	.575*
	Sig.	.267	.	.000	.000	.000	.000	.000	.000
	N	110	110	97	110	110	97	97	97
BAI	**Pearson**	.002	.806	1	.559	.561*	.676*	.607*	.585*
	Sig.	.984	.000	.	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
STAI-State	**Pearson**	.112	.626	.559	1	.739*	.570*	.518*	.509*
	Sig.	.246	.000	.000	.	.000	.000	.000	.000
	N	110	110	97	110	110	97	97	97
STAI-Trait	**Pearson**	.202*	.720*	.561*	.739*	1	.704*	.651*	.684*
	Sig.	.034	.000	.000	.000	.	.000	.000	.000
	N	110	110	97	110	110	97	97	97
ANX	**Pearson**	.126	.682*	.676*	.570*	.704*	1	.948*	.907*
	Sig.	.219	.000	.000	.000	.000	.	.000	.000
	N	97	97	97	97	97	97	97	97
ANX-C	**Pearson**	.162	.607*	.607*	.518*	.651*	.948*	1	.807*
	Sig.	.113	.000	.000	.000	.000	.000	.	.000
	N	97	97	97	97	97	97	97	97
ANX-A	**Pearson**	.074	.575*	.585*	.509*	.684*	.907*	.807*	1
	Sig.	.473	.000	.000	.000	.000	.000	.000	.
	N	97	97	97	97	97	97	97	97
ANX-P	**Pearson**	.091	.699*	.667*	.537*	.593*	.874*	.741*	.684*
	Sig.	.373	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
ARD	**Pearson**	.106	.477*	.449*	.386*	.546*	.683*	.681*	.584*
	Sig.	.302	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
ARD-O	**Pearson**	.058	.159	.181	.071	.105	.339*	.385*	.184
	Sig.	.575	.120	.076	.488	.308	.001	.000	.071
	N	97	97	97	97	97	97	97	97
ARD-P	**Pearson**	.114	.417*	.461*	.458*	.606*	.712*	.694*	.701*
	Sig.	.268	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
ARD-S	**Pearson**	.069	.464*	.362*	.338*	.505*	.486*	.457*	.440*
	Sig.	.504	.000	.000	.001	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
DEP	**Pearson**	.155	.809*	.646*	.615*	.752*	.721*	.621*	.615*
	Sig.	.130	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97

		Digit Ratio	BDI	BAI	STAI-State	STAI-Trait	ANX	ANX-C	ANX-A
DEP-C	**Pearson**	.140	.715*	.550*	.497*	.682*	.626*	.542*	.517*
	Sig.	.172	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
DEP-A	**Pearson**	.163	.821*	.674*	.670*	.737*	.701*	.619*	.588*
	Sig.	.110	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
DEP-P	**Pearson**	.099	.565*	.451*	.423*	.539*	.547*	.451*	.492*
	Sig.	.335	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
BOR	**Pearson**	.206*	.693*	.637*	.518*	.704*	.676*	.598*	.663*
	Sig.	.043	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
BOR-A	**Pearson**	.268*	.543*	.502*	.494*	.621*	.619*	.524*	.597*
	Sig.	.008	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
BOR-I	**Pearson**	.172	.734*	.657*	.498*	.661*	.669*	.637*	.627*
	Sig.	.091	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
BOR-N	**Pearson**	.148	.588*	.569*	.392*	.591*	.584*	.530*	.564*
	Sig.	.149	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
BOR-S	**Pearson**	.020	.302*	.249*	.227*	.326*	.192	.122	.263*
	Sig.	.843	.003	.014	.026	.001	.059	.232	.009
	N	97	97	97	97	97	97	97	97
POMS – TMD 1	**Pearson**	.043	.762*	.746*	.624*	.682*	.663*	.619*	.588*
	Sig.	.676	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
POMS – TMD 2	**Pearson**	.106	.598*	.591*	.686*	.695*	.642*	.585*	.627*
	Sig.	.302	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97

		ANX-P	ARD	ARD-O	ARD-P	ARD-S	DEP	DEP-C	DEP-A
Digit Ratio	**Pearson**	.091	.106	.058	.114	.069	.155	.140	.163
	Sig.	.373	.302	.575	.268	.504	.130	.172	.110
	N	97	97	97	97	97	97	97	97
BDI	**Pearson**	.699*	.477*	.159	.417*	.464*	.809*	.715*	.821*
	Sig.	.000	.000	.120	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
BAI	**Pearson**	.667*	.449*	.181	.461*	.362*	.646*	.550*	.674*
	Sig.	.000	.000	.076	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
STAI-State	**Pearson**	.537*	.386*	.071	.458*	.338*	.615*	.497*	.670*
	Sig.	.000	.000	.488	.000	.001	.000	.000	.000
	N	97	97	97	97	97	97	97	97
STAI-Trait	**Pearson**	.593*	.546*	.105	.606*	.505*	.752*	.682*	.737*
	Sig.	.000	.000	.308	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
ANX	**Pearson**	.874*	.683*	.339*	.712*	.486*	.721*	.626*	.701*
	Sig.	.000	.000	.001	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
ANX-C	**Pearson**	.741*	.681*	.385*	.694*	.457*	.621*	.542*	.619*
	Sig.	.000	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
ANX-A	**Pearson**	.684*	.584*	.184	.701*	.440*	.615*	.517*	.588*
	Sig.	.000	.000	.071	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
ANX-P	**Pearson**	1	.585*	.335*	.539*	.430*	.759*	.672*	.726*
	Sig.	.	.000	.001	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
ARD	**Pearson**	.585*	1	.691*	.683*	.803*	.512*	.405*	.523*
	Sig.	.000	.	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
ARD-O	**Pearson**	.335*	.691*	1	.230*	.284*	.132	.028	.155
	Sig.	.001	.000	.	.024	.005	.197	.785	.130
	N	97	97	97	97	97	97	97	97
ARD-P	**Pearson**	.539*	.683*	.230*	1	.375*	.492*	.442*	.505*
	Sig.	.000	.000	.024	.	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
ARD-S	**Pearson**	.430*	.803*	.284*	.375*	1	.498*	.421*	.490*
	Sig.	.000	.000	.005	.000	.	.000	.000	.000
	N	97	97	97	97	97	97	97	97
DEP	**Pearson**	.759*	.512*	.132	.492*	.498*	1	.869*	.908*
	Sig.	.000	.000	.197	.000	.000	.	.000	.000
	N	97	97	97	97	97	97	97	97
DEP-C	**Pearson**	.672*	.405*	.028	.442*	.421*	.869*	1	.747*
	Sig.	.000	.000	.785	.000	.000	.000	.	.000
	N	97	97	97	97	97	97	97	97
DEP-A	**Pearson**	.726*	.523*	.155	.505*	.490*	.908*	.747*	1
	Sig.	.000	.000	.130	.000	.000	.000	.000	.
	N	97	97	97	97	97	97	97	97

		ANX-P	ARD	ARD-O	ARD-P	ARD-S	DEP	DEP-C	DEP-A
DEP-P	**Pearson**	.577*	.397*	.150	.331*	.382*	.826*	.542*	.601*
	Sig.	.000	.000	.143	.001	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
BOR	**Pearson**	.600*	.543*	.104	.456*	.607*	.709*	.663*	.698*
	Sig.	.000	.000	.310	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
BOR-A	**Pearson**	.594*	.462*	.109	.373*	.510*	.626*	.571*	.593*
	Sig.	.000	.000	.289	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
BOR-I	**Pearson**	.560*	.544*	.196	.533*	.476*	.668*	.625*	.715*
	Sig.	.000	.000	.055	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
BOR-N	**Pearson**	.510*	.517*	.065	.440*	.603*	.608*	.538*	.590*
	Sig.	.000	.000	.529	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
BOR-S	**Pearson**	.161	.129	-.095	.008	.319*	.306*	.355*	.260*
	Sig.	.115	.207	.354	.941	.001	.000	.000	.010
	N	97	97	97	97	97	97	97	97
POMS – TMD 1	**Pearson**	.605*	.512*	.159	.403*	.540*	.688*	.595*	.749*
	Sig.	.000	.000	.120	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
POMS – TMD 2	**Pearson**	.547*	.527*	.144	.518*	.498*	.635*	.525*	.710*
	Sig.	.000	.000	.158	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97

		DEP-P	BOR	BOR-A	BOR-I	BOR-N	BOR-S	POMS TMD-1	POMS TMD-2
Digit Ratio	**Pearson**	.099	.206*	.268*	.172	.148	.020	.043	.106
	Sig.	.335	.043	.008	.091	.149	.843	.676	.302
	N	97	97	97	97	97	97	97	97
BDI	**Pearson**	.565*	.693*	.543*	.734*	.588*	.302*	.762*	.598*
	Sig.	.000	.000	.000	.000	.000	.003	.000	.000
	N	97	97	97	97	97	97	97	97
BAI	**Pearson**	.451*	.637*	.502*	.657*	.569*	.249*	.746*	.591*
	Sig.	.000	.000	.000	.000	.000	.014	.000	.000
	N	97	97	97	97	97	97	97	97
STAI-State	**Pearson**	.423*	.518*	.494*	.498*	.392*	.227*	.624*	.686*
	Sig.	.000	.000	.000	.000	.000	.026	.000	.000
	N	97	97	97	97	97	97	97	97
STAI-Trait	**Pearson**	.539*	.704*	.621*	.661*	.591*	.326*	.682*	.695*
	Sig.	.000	.000	.000	.000	.000	.001	.000	.000
	N	97	97	97	97	97	97	97	97
ANX	**Pearson**	.547*	.676*	.619*	.669*	.584*	.192	.663*	.642*
	Sig.	.000	.000	.000	.000	.000	.059	.000	.000
	N	97	97	97	97	97	97	97	97
ANX-C	**Pearson**	.451*	.598*	.524*	.637*	.530*	.122	.619*	.585*
	Sig.	.000	.000	.000	.000	.000	.232	.000	.000
	N	97	97	97	97	97	97	97	97
ANX-A	**Pearson**	.492*	.663*	.597*	.627*	.564*	.263*	.588*	.627*
	Sig.	.000	.000	.000	.000	.000	.009	.000	.000
	N	97	97	97	97	97	97	97	97
ANX-P	**Pearson**	.577*	.600*	.594*	.560*	.510*	.161	.605*	.547*
	Sig.	.000	.000	.000	.000	.000	.115	.000	.000
	N	97	97	97	97	97	97	97	97
ARD	**Pearson**	.397*	.543*	.462*	.544*	.517*	.129	.512*	.527*
	Sig.	.000	.000	.000	.000	.000	.207	.000	.000
	N	97	97	97	97	97	97	97	97
ARD-O	**Pearson**	.150	.104	.109	.196	.065	-.095	.159	.144
	Sig.	.143	.310	.289	.055	.529	.354	.120	.158
	N	97	97	97	97	97	97	97	97
ARD-P	**Pearson**	.331*	.456*	.373*	.533*	.440*	.008	.403*	.518*
	Sig.	.001	.000	.000	.000	.000	.941	.000	.000
	N	97	97	97	97	97	97	97	97
ARD-S	**Pearson**	.382*	.607*	.510*	.476*	.603*	.319*	.540*	.498*
	Sig.	.000	.000	.000	.000	.000	.001	.000	.000
	N	97	97	97	97	97	97	97	97
DEP	**Pearson**	.826*	.709*	.626*	.668*	.608*	.306*	.688*	.635*
	Sig.	.000	.000	.000	.000	.000	.002	.000	.000
	N	97	97	97	97	97	97	97	97
DEP-C	**Pearson**	.542*	.663*	.571*	.625*	.538*	.355*	.595*	.525*
	Sig.	.000	.000	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
DEP-A	**Pearson**	.601*	.698*	.593*	.715*	.590*	.260*	.749*	.710*
	Sig.	.000	.000	.000	.000	.000	.010	.000	.000
	N	97	97	97	97	97	97	97	97

		DEP-P	BOR	BOR-A	BOR-I	BOR-N	BOR-S	POMS TMD-1	POMS TMD-2
DEP-P	**Pearson**	1	.488*	.465*	.395*	.453*	.191	.439*	.406*
	Sig.	.	.000	.000	.000	.000	.060	.000	.000
	N	97	97	97	97	97	97	97	97
BOR	**Pearson**	.488*	1	.886*	.834*	.852*	.594*	.753*	.698*
	Sig.	.000	.	.000	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
BOR-A	**Pearson**	.465*	.886*	1	.643*	.656*	.466*	.654*	.590*
	Sig.	.000	.000	.	.000	.000	.000	.000	.000
	N	97	97	97	97	97	97	97	97
BOR-I	**Pearson**	.395*	.834*	.643*	1	.638*	.307*	.674*	.650*
	Sig.	.000	.000	.000	.	.000	.002	.000	.000
	N	97	97	97	97	97	97	97	97
BOR-N	**Pearson**	.453*	.852*	.656*	.638*	1	.348*	.681*	.575*
	Sig.	.000	.000	.000	.000	.	.000	.000	.000
	N	97	97	97	97	97	97	97	97
BOR-S	**Pearson**	.191	.594*	.466*	.307*	.348*	1	.337*	.390*
	Sig.	.060	.000	.000	.002	.000	.	.001	.000
	N	97	97	97	97	97	97	97	97
POMS–TMD 1	**Pearson**	.439*	.753*	.654*	.674*	.681*	.337*	1	.776*
	Sig.	.000	.000	.000	.000	.000	.001	.	.000
	N	97	97	97	97	97	97	97	97
POMS–TMD 2	**Pearson**	.406*	.698*	.590*	.650*	.575*	.390*	.776*	1
	Sig.	.000	.000	.000	.000	.000	.000	.000	.
	N	97	97	97	97	97	97	97	97

CORRELATION MATRIX – DIGIT RATIO AND SPATIAL TASKS

		Digit Ratio	Mental Rotation - # correct	Targeting - Accuracy	Location Memory - Identity	Location Memory - Location	Spatial Memory - Errors	Spatial Memory - Time
Digit Ratio	**Pearson**	1	-.023	.176	-.189	-.002	.015	-.022
	Sig.	.	.814	.066	.090	.987	.882	.821
	N	110	110	110	81	79	105	106
Mental Rotation	**Pearson**	-.023	1	-.362*	.118	.229*	-.016	-.145
	Sig.	.814	.	.000	.294	.042	.875	.138
	N	110	110	110	81	79	105	106
Targeting	**Pearson**	.176	-.362*	1	.183	.094	-.053	-.019
	Sig.	.066	.000	.	.102	.411	.595	.849
	N	110	110	110	81	79	105	106
Location Memory-Identity	**Pearson**	-.189	.118	.183	1	.546*	-.169	-.032
	Sig.	.090	.294	.102	.	.000	.145	.785
	N	81	81	81	81	79	76	77
Location Memory-Location	**Pearson**	-.002	.229*	.094	.546*	1	-.141	-.159
	Sig.	.987	.042	.411	.000	.	.230	.172
	N	79	79	79	79	79	74	75
Spatial Memory-Errors	**Pearson**	.015	-.016	-.053	-.169	-.141	1	.427*
	Sig.	.882	.875	.595	.145	.230	.	.000
	N	105	105	105	76	74	105	105
Spatial Memory-Time	**Pearson**	-.022	-.145	-.019	-.032	-.159	.427*	1
	Sig.	.821	.138	.849	.785	.172	.000	.
	N	106	106	106	77	75	105	106

CORRELATION MATRIX – DIGIT RATIO AND GENDER ROLE MEASURES

		Digit Ratio	PSAI	OAT-PM Masculine	OAT-PM Feminine	OAT-AM	BSRI Masculine	BSRI Feminine
Digit Ratio	**Pearson**	1	-.230*	-.154	.052	.034	-.175	.024
	Sig.	.	.015	.133	.610	.742	.087	.817
	N	110	110	97	97	97	97	97
PSAI	**Pearson**	-.230*	1	.585*	-.588*	.353*	.323*	-.397*
	Sig.	.015	.	.000	.000	.000	.001	.000
	N	110	110	97	97	97	97	97
OAT-PM Masculine	**Pearson**	-.154	.585*	1	-.172	.153	.272*	-.338*
	Sig.	.133	.000	.	.092	.135	.007	.001
	N	97	97	97	97	97	97	97
OAT-PM Feminine	**Pearson**	.052	-.588*	-.172	1	-.137	-.095	.365*
	Sig.	.610	.000	.092	.	.182	.354	.000
	N	97	97	97	97	97	97	97
OAT-AM	**Pearson**	.034	.353*	.153	-.137	1	.115	-.077
	Sig.	.742	.000	.135	.182	.	.260	.452
	N	97	97	97	97	97	97	97
BSRI Masculine	**Pearson**	-.175	.323*	.272*	-.095	.115	1	-.078
	Sig.	.087	.001	.007	.354	.260	.	.445
	N	97	97	97	97	97	97	97
BSRI Feminine	**Pearson**	.024	-.397*	-.338*	.365*	-.077	-.078	1
	Sig.	.817	.000	.001	.000	.452	.445	.
	N	97	97	97	97	97	97	97

CORRELATION MATRIX - PSYCHOPATHOLOGY MEASURES AND SPATIAL TASKS

		Mental Rotation - # correct	Targeting - Accuracy	Location Memory - Identity	Location Memory - Location	Spatial Memory - Errors	Spatial Memory - Time
BDI	**Pearson**	-.046	.073	.044	.017	-.017	-.079
	Sig.	.633	.447	.695	.883	.862	.420
	N	110	110	81	79	105	106
BAI	**Pearson**	-.152	.140	.033	-.099	.039	-.039
	Sig.	.137	.171	.781	.410	.710	.713
	N	97	97	73	71	92	93
STAI-State	**Pearson**	-.082	.080	.101	-.007	.126	.006
	Sig.	.397	.405	.371	.952	.201	.953
	N	110	110	81	79	105	106
STAI-Trait	**Pearson**	-.192*	.213*	-.048	-.020	.114	.015
	Sig.	.045	.026	.671	.859	.246	.877
	N	110	110	81	79	105	106
ANX	**Pearson**	-.328*	.225*	-.047	-.039	.097	.073
	Sig.	.001	.027	.694	.747	.357	.488
	N	97	97	73	71	92	93
ANX-C	**Pearson**	-.311*	.238*	-.021	-.110	.081	.068
	Sig.	.002	.019	.861	.361	.444	.519
	N	97	97	73	71	92	93
ANX-A	**Pearson**	-.378*	.245*	-.121	-.012	.143	.086
	Sig.	.000	.016	.306	.919	.174	.411
	N	97	97	73	71	92	93
ANX-P	**Pearson**	-.203*	.121	.003	.046	.044	.045
	Sig.	.046	.238	.981	.706	.676	.671
	N	97	97	73	71	92	93
ARD	**Pearson**	-.192	-.020	-.016	-.028	.052	.130
	Sig.	.060	.846	.894	.815	.622	.214
	N	97	97	73	71	92	93
ARD-O	**Pearson**	-.006	-.061	.124	-.047	-.025	.145
	Sig.	.951	.556	.296	.695	.813	.167
	N	97	97	73	71	92	93
ARD-P	**Pearson**	-.284*	.154	.072	-.051	.172	.195
	Sig.	.005	.131	.545	.673	.101	.061
	N	97	97	73	71	92	93
ARD-S	**Pearson**	-.152	-.096	-.188	.030	-.005	-.018
	Sig.	.137	.349	.111	.804	.962	.864
	N	97	97	73	71	92	93
DEP	**Pearson**	-.080	.062	.009	.121	.094	-.002
	Sig.	.438	.548	.943	.316	.372	.986
	N	97	97	73	71	92	93
DEP-C	**Pearson**	.007	-.011	.010	.135	.105	-.029
	Sig.	.947	.913	.932	.261	.318	.786
	N	97	97	73	71	92	93

		Mental Rotation - # correct	Targeting - Accuracy	Location Memory - Identity	Location Memory - Location	Spatial Memory - Errors	Spatial Memory - Time
DEP-A	**Pearson**	-.002	.024	.008	.093	.073	-.026
	Sig.	.984	.815	.946	.443	.488	.802
	N	97	97	73	71	92	93
DEP-P	**Pearson**	-.209*	.144	.005	.097	.067	.052
	Sig.	.040	.161	.969	.423	.526	.621
	N	97	97	73	71	92	93
BOR	**Pearson**	-.112	.081	-.138	.101	.083	-.107
	Sig.	.275	.431	.244	.402	.433	.306
	N	97	97	73	71	92	93
BOR-A	**Pearson**	-.108	.091	-.197	.024	.188	.010
	Sig.	.291	.375	.095	.842	.072	.922
	N	97	97	73	71	92	93
BOR-I	**Pearson**	-.029	.040	-.050	.066	.013	-.130
	Sig.	.780	.699	.675	.582	.899	.212
	N	97	97	73	71	92	93
BOR-N	**Pearson**	-.162	.111	-.100	.147	.053	-.163
	Sig.	.113	.277	.399	.220	.615	.118
	N	97	97	73	71	92	93
BOR-S	**Pearson**	-.043	-.011	-.083	.096	-.028	-.064
	Sig.	.676	.916	.485	.427	.794	.543
	N	97	97	73	71	92	93
POMS–TMD 1	**Pearson**	.028	-.012	-.108	-.117	.052	-.101
	Sig.	.784	.911	.365	.332	.623	.337
	N	97	97	73	71	92	93
POMS–TMD 2	**Pearson**	-.133	.030	-.121	-.212	.123	-.003
	Sig.	.195	.773	.307	.076	.242	.976
	N	97	97	73	71	92	93

CORRELATION MATRIX – PSYCHOPATHOLOGY MEASURES AND GENDER ROLE MEASURES

		PSAI	OAT-PM Masculine	OAT-PM Feminine	OAT-AM	BSRI Masculine	BSRI Feminine
BDI	**Pearson**	-.148	-.026	.070	-.023	-.217*	.037
	Sig.	.123	.804	.495	.825	.033	.717
	N	110	97	97	97	97	97
BAI	**Pearson**	-.207*	-.008	.119	-.127	-.120	-.013
	Sig.	.042	.938	.246	.216	.243	.901
	N	97	97	97	97	97	97
STAI-State	**Pearson**	-.092	-.063	.024	-.021	-.105	.003
	Sig.	.339	.540	.819	.836	.305	.975
	N	110	97	97	97	97	97
STAI-Trait	**Pearson**	-.159	-.093	.066	-.010	-.310*	.065
	Sig.	.098	.365	.518	.919	.002	.525
	N	110	97	97	97	97	97
ANX	**Pearson**	-.306*	-.112	.179	-.045	-.250*	.088
	Sig.	.002	.275	.079	.665	.013	.394
	N	97	97	97	97	97	97
ANX-C	**Pearson**	-.278*	-.124	.182	-.036	-.208*	.119
	Sig.	.006	.225	.074	.726	.041	.246
	N	97	97	97	97	97	97
ANX-A	**Pearson**	-.316*	-.145	.192	-.060	-.302*	.120
	Sig.	.002	.157	.060	.560	.003	.242
	N	97	97	97	97	97	97
ANX-P	**Pearson**	-.244*	-.028	.110	-.027	-.184	-.016
	Sig.	.016	.788	.283	.790	.072	.880
	N	97	97	97	97	97	97
ARD	**Pearson**	-.180	-.056	.214*	.031	-.176	.039
	Sig.	.078	.587	.035	.762	.085	.706
	N	97	97	97	97	97	97
ARD-O	**Pearson**	-.145	-.049	.313*	.108	-.078	.032
	Sig.	.157	.637	.002	.294	.445	.759
	N	97	97	97	97	97	97
ARD-P	**Pearson**	-.298*	-.164	.138	-.058	-.323*	.245*
	Sig.	.003	.109	.176	.571	.001	.016
	N	97	97	97	97	97	97
ARD-S	**Pearson**	-.001	.054	.038	.009	-.033	-.129
	Sig.	.990	.603	.712	.933	.748	.208
	N	97	97	97	97	97	97
DEP	**Pearson**	-.082	-.046	-.067	-.049	-.239*	-.085
	Sig.	.422	.655	.514	.633	.019	.409
	N	97	97	97	97	97	97
DEP-C	**Pearson**	-.006	.003	-.172	-.020	-.247*	-.022
	Sig.	.951	.976	.091	.844	.015	.829
	N	97	97	97	97	97	97
DEP-A	**Pearson**	-.071	-.096	-.083	-.043	-.240*	-.145
	Sig.	.489	.352	.418	.678	.018	.156
	N	97	97	97	97	97	97

		PSAI	OAT-PM Masculine	OAT-PM Feminine	OAT-AM	BSRI Masculine	BSRI Feminine
DEP-P	**Pearson**	-.131	-.019	.071	-.063	-.136	-.044
	Sig.	.201	.852	.490	.542	.184	.670
	N	97	97	97	97	97	97
BOR	**Pearson**	-.069	-.004	-.052	-.056	-.131	-.037
	Sig.	.504	.969	.612	.584	.201	.718
	N	97	97	97	97	97	97
BOR-A	**Pearson**	-.072	.010	-.025	-.053	-.091	-.158
	Sig.	.484	.921	.809	.603	.375	.121
	N	97	97	97	97	97	97
BOR-I	**Pearson**	-.075	-.116	-.081	-.059	-.253*	.029
	Sig.	.468	.256	.433	.567	.012	.778
	N	97	97	97	97	97	97
BOR-N	**Pearson**	-.117	.030	.008	-.084	-.084	.030
	Sig.	.253	.772	.940	.413	.411	.773
	N	97	97	97	97	97	97
BOR-S	**Pearson**	.099	.095	-.095	.049	.057	.002
	Sig.	.333	.357	.356	.631	.576	.985
	N	97	97	97	97	97	97
POMS–TMD 1	**Pearson**	-.058	.055	-.004	-.059	-.093	-.143
	Sig.	.574	.590	.972	.564	.366	.161
	N	97	97	97	97	97	97
POMS–TMD 2	**Pearson**	-.059	-.019	-.036	.033	-.174	-.076
	Sig.	.565	.850	.728	.746	.087	.458
	N	97	97	97	97	97	97

CORRELATION MATRIX – GENDER ROLE MEASURES AND SPATIAL TASKS

		Mental Rotation - # correct	Targeting - Accuracy	Location Memory - Identity	Location Memory - Location	Spatial Memory - Errors	Spatial Memory - Time
PSAI	**Pearson**	.381*	-.544*	-.068	-.083	.044	.023
	Sig.	.000	.000	.544	.467	.659	.817
	N	110	110	81	79	105	106
OAT-PM Masculine	**Pearson**	.245*	-.234*	.119	.043	.063	.051
	Sig.	.016	.021	.316	.724	.551	.629
	N	97	97	73	71	92	93
OAT-PM Feminine	**Pearson**	-.385*	.389*	.025	-.050	.036	.201
	Sig.	.000	.000	.835	.676	.732	.053
	N	97	97	73	71	92	93
OAT-AM	**Pearson**	.096	-.219*	-.112	-.176	-.003	.284*
	Sig.	.347	.031	.347	.143	.974	.006
	N	97	97	73	71	92	93
BSRI Masculine	**Pearson**	.141	-.099	.075	-.060	-.108	-.218*
	Sig.	.169	.332	.528	.619	.304	.036
	N	97	97	73	71	92	93
BSRI Feminine	**Pearson**	-.221*	.300*	.157	.079	-.032	.013
	Sig.	.029	.003	.184	.513	.759	.900
	N	97	97	73	71	92	93

CORRELATION MATRIX – HORMONE LEVELS AND PSYCHOPATHOLOGY

MEASURES

Males

		BDI	BAI	STAI-State	STAI-Trait	ANX	ANX-C	ANX-A
Average Testosterone	**Pearson**	-.032	-.010	.159	.011	.026	.101	.012
	Sig.	.809	.945	.232	.937	.850	.469	.932
	N	58	54	58	58	54	54	54

		ANX-P	ARD	ARD-O	ARD-P	ARD-S	DEP	DEP-C
Average Testosterone	**Pearson**	-.076	-.011	-.102	.027	.043	.044	.056
	Sig.	.585	.937	.464	.844	.757	.752	.688
	N	54	54	54	54	54	54	54

		DEP-A	DEP-P	BOR	BOR-A	BOR-I	BOR-N	BOR-S
Average Testosterone	**Pearson**	-.024	.089	-.008	-.024	-.001	.149	-.216
	Sig.	.866	.520	.953	.865	.992	.283	.117
	N	54	54	54	54	54	54	54

		POMS TMD-1	POMS TMD-2
Average Testosterone	**Pearson**	.108	-.008
	Sig.	.437	.955
	N	54	54

Females Not Taking Oral Contraceptives

		BDI	BAI	STAI-State	STAI-Trait	ANX	ANX-C	ANX-A
Average Estradiol	**Pearson**	.280	.141	.158	.173	-.248	-.309	-.285
	Sig.	.115	.483	.380	.336	.213	.117	.150
	N	33	27	33	33	27	27	27
Average Testosterone	**Pearson**	.137	.338	-.118	-.165	.053	.070	-.040
	Sig.	.420	.063	.485	.328	.776	.710	.833
	N	37	31	37	37	31	31	31

		ANX-P	ARD	ARD-O	ARD-P	ARD-S	DEP	DEP-C
Average Estradiol	**Pearson**	-.080	-.279	-.331	-.122	-.160	-.027	.001
	Sig.	.693	.159	.092	.543	.425	.895	.997
	N	27	27	27	27	27	27	27
Average Testosterone	**Pearson**	.092	-.096	-.012	-.004	-.159	.112	-.036
	Sig.	.622	.607	.949	.981	.393	.548	.847
	N	31	31	31	31	31	31	31

		DEP-A	DEP-P	BOR	BOR-A	BOR-I	BOR-N	BOR-S
Average Estradiol	**Pearson**	.048	-.165	.107	.030	.330	-.105	.120
	Sig.	.810	.410	.594	.881	.093	.603	.552
	N	27	27	27	27	27	27	27
Average Testosterone	**Pearson**	.136	.203	.054	.031	.037	.161	-.163
	Sig.	.466	.274	.774	.870	.844	.386	.382
	N	31	31	31	31	31	31	31

		POMS TMD-1	POMS TMD-2
Average Estradiol	**Pearson**	.074	-.030
	Sig.	.713	.882
	N	27	27
Average Testosterone	**Pearson**	.176	.088
	Sig.	.342	.638
	N	31	31

CORRELATION MATRIX – HORMONE LEVELS AND SPATIAL TASKS

Males

		Mental Rotation - # correct	Targeting - Accuracy	Location Memory - Identity	Location Memory - Location	Spatial Memory - Errors	Spatial Memory - Time
Average Testosterone	**Pearson**	.022	.023	-.012	.012	-.167	-.091
	Sig.	.869	.864	.938	.942	.222	.504
	N	58	58	42	41	55	56

Females Not Taking Oral Contraceptives

		Mental Rotation - # correct	Targeting - Accuracy	Location Memory - Identity	Location Memory - Location	Spatial Memory - Errors	Spatial Memory - Time
Average Estradiol	**Pearson**	.417*	.161	.172	.041	-.356*	-.308
	Sig.	.016	.371	.401	.847	.049	.092
	N	33	33	26	25	31	31
Average Testosterone	**Pearson**	-.007	-.099	-.373	-.534*	.011	.006
	Sig.	.966	.561	.056	.005	.952	.974
	N	37	37	27	26	35	35

CORRELATION MATRIX – HORMONE LEVELS AND GENDER ROLE MEASURES

Males

		PSAI	OAT-PM Masculine	OAT-PM Feminine	OAT-AM	BSRI Masculine	BSRI Feminine
Average Testosterone	**Pearson**	.083	-.106	.206	-.074	.118	.161
	Sig.	.534	.446	.136	.593	.397	.245
	N	58	54	54	54	54	54

Females Not Taking Oral Contraceptives

		PSAI	OAT-PM Masculine	OAT-PM Feminine	OAT-AM	BSRI Masculine	BSRI Feminine
Average Estradiol	**Pearson**	-.242	-.015	.017	-.194	-.026	-.085
	Sig.	.175	.940	.932	.332	.899	.672
	N	33	27	27	27	27	27
Average Testosterone	**Pearson**	.132	.358*	-.061	.009	.159	-.450*
	Sig.	.437	.048	.746	.962	.394	.011
	N	37	31	31	31	31	31

CORRELATION MATRIX – DIGIT RATIO AND HORMONE LEVELS

Males

		Digit Ratio
Average Testosterone	**Pearson**	.149
	Sig.	.264
	N	58

Females Not Taking Oral Contraceptives

		Digit Ratio
Average Estradiol	**Pearson**	.155
	Sig.	.389
	N	33
Average Testosterone	**Pearson**	.060
	Sig.	.725
	N	37

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