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Revisiting the Sex-differentiation Hypothesis: A Refinement and Examination of Lilienfeld's (1992) Hypotheses

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REVISITING THE SEX-DIFFERENTIATION HYPOTHESIS:

A REFINEMENT AND EXAMINATION OF LILIENFELD'S (1992) HYPOTHESES

By

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ABSTRACT

The current study investigated Lilienfeld's (1992) sex differentiation hypothesis. Modifications were made to highlight the importance of examining social expectations of acceptable behavior for women and men. Modifications were also made to move away from broad constructs (i.e. negative emotionality and behavioral disinhibition) and instead examine narrower constructs (i.e., anxiety and impulsivity) with more explanatory utility. Results support the utility gender role in predicting antisocial symptoms, but not somatic symptoms. Some support for a relationship between somatization and impulsivity as predicted by the behavioral disinhibition hypothesis was found, but results indicate that the relationship between somatization and antisociality is better accounted for by anxiety, and this is consistent with the negative emotionality model. The sex-differentiation aspect of Lilienfeld's hypotheses was largely unsupported. Future research in the area can benefit from attending to the cautions presented in this paper, as well as the limitations noted.

CHAPTER 1
INTRODUCTION

There exists a differential lifetime prevalence for somatization disorder of 0.2-2% in women and less than 0.2% in men (American Psychiatric Association [APA], 2000). This finding of differential prevalence rates holds across a variety of populations, including community samples (Haug, Mykletun, & Dahl, 2004; Ladwig, Marten-Mittag, Erazo, & Gündel, 2001; Piccinelli, & Simon, 1997; Rief, Hessel, & Braehler, 2001), alcohol abuse samples (Milani, Parrott, Turner, & Fox, 2004), individuals with depression (Silverstein, 1999; Silverstein, 2002), and college samples (Wilson, Frick, & Clements, 1999). Similarly, antisocial personality disorder (ASPD) and antisocial symptomatology have long been known to exist with greater prevalence and severity in men compared to women, with differential prevalence rates of 3% and 1%, respectively (APA, 2000). This finding has also been found to generalize across a variety of populations, including mental health outpatients (Grilo, 2002; Zlotnick, Rothchild, & Zimmerman, 2002; Zlotnick, Zimmerman, Wolfsdorf, & Mattia, 2001), drug treatment patients (Barber et al, 1996; Grella, Joshi, & Hser, 2003), and college samples (Cale, & Lilienfeld, 2002; Lilienfeld, & Hess, 2001).

Somatization and antisocial personality bear a rather interesting relationship to each other, as several lines of evidence point to the co-occurrence of these disorders within individuals and within families at greater than chance levels. Cloninger, Guze, and colleagues originally observed this relationship (Cloninger & Guze, 1970; Cloninger & Guze, 1973; Woerner & Guze, 1968) and made the argument for a common genetic diathesis expressed differentially depending on the sex of the individual, and based this assertion on an impressive series of family and adoption studies (Bohman, Cloninger, von Knorring, & Sigvardsson, 1984; Cloninger, Reich, & Guze, 1975a; Cloninger, Reich, & Guze, 1975b; Cloninger, Sigvardsson, von Knorring, & Bohman, 1984; Sigvardsson, von Knorring, Bohman, & Cloninger 1984).

Lilienfeld later revisited the arguments by Cloninger and colleagues when he proposed the *sex-differentiation hypothesis* (Lilienfeld, 1992). He also argued that biological sex interacts with the common diathesis for somatic and antisocial symptoms, and delineated two possible etiological models of this sex-differentiated expression. The first model, *behavioral disinhibition,* argues that one's tendency toward behavioral disinhibition is expressed differentially in women and men. Women who are high in behavioral disinhibition will be more

likely to display somatic symptoms, whereas men high in behavioral disinhibition will be more likely to display antisocial symptoms. Presumably, this differential expression occurs because it may be more socially appropriate for men to exhibit antisocial symptoms and women to exhibit somatic symptoms. Thus, according to Lilienfeld, biological sex should moderate the relationship between behavioral disinhibition and somatization, as well as the relationship between behavioral disinhibition and antisocial behavior.

The second model, *negative emotionality,* argues that the relationship between antisocial and somatic symptoms is mediated by negative emotionality, such that engaging in antisocial behavior leads to somatic symptoms due to increases in negative emotionality caused by the stress of leading an antisocial lifestyle. Presumably, a consequence of women engaging in such highly gender non-congruent behavior is that they experience more negative emotionality than men. Thus, according to this model, the relationship between antisocial symptoms and somatic symptoms is mediated by negative emotionality, and the relationship between antisocial symptoms and negative emotionality is moderated by sex.

The current study sought to refine and test Lilienfeld's (1992) hypotheses. In addition to testing the moderating effect of sex as predicted by Lilienfeld, the current study examined gender role as a moderator. It was also suggested here that, where Lilienfeld proposes an effect of behavioral disinhibition and negative emotionality, these constructs might be too broad to be useful as explanatory mechanisms for the antisociality-somatization relationship. The current study sought to make more specific those mechanisms that may be underlying the relationship between antisocial and somatic symptomatology in each of his proposed models. Thus, the current study proposed to narrow behavioral disinhibition to the facet of impulsivity labeled (lack of) premeditation and narrow negative emotionality to anxiety.

Incorporating Gender Role

The concept of gender role is arguably best described by Bem (1978). According to Bem, individuals who endorse traits that are viewed by society as most appropriate for women or traditionally associated with women are feminine in gender role, whereas those individuals who endorse traits viewed as most appropriate for men or traditionally associated with men are considered masculine in gender role. It is important to note this definition implies that gender role is socially constructed and, therefore, the gender role with which one identifies may not necessarily be analogous to one's sex (i.e., there are masculine women and feminine men). Thus,

a major goal for this study was to move away from the presumption that observed gender differences in behavior are purely biological, and instead examine how socialization and expectations about male and female behavior may drive what appear to be sex differences.

Research on the influence of gender role on personality traits indicates that in some situations, individuals will refrain from engaging in behaviors that certain personality traits would otherwise predict they are prone to displaying if that behavior is inconsistent with the social expectations of the individual's gender (Carbonell, 1984; Carbonell & Castro, in press). Further, early research on gender role indicates that individuals who identify strongly with either the masculine or feminine gender role are likely to engage in behavior that is consistent with their gender role, regardless of their sex (Bem, 1975; Bem, Martyna, & Watson, 1976). These findings may have implications for Lilienfeld's (1992) behavioral disinhibition model. For example, although impulsivity is a good predictor of antisocial behaviors, these behaviors are described in terms more readily associated with men (e.g., impulsive, risk-taking, aggressive). Thus, a feminine individual who is highly impulsive may refrain from expressing impulsivity with antisocial behavior because doing so would be inconsistent with that person's gender role. They may instead express impulsivity with somatic behaviors. This argument is highly consistent with Lilienfeld's behavioral disinhibition hypothesis.

Bem (1985) posits that masculine and feminine individuals have strong gender schemas. That is, they more readily process information about the world and themselves around the concept of gender. Also, research on gender role indicates that masculine and feminine individuals may be more prone to distress in the form of gender role strain (Roades, 2000); or rather, distress that is experienced when individuals behave in a manner that is inconsistent with their gender role. For example, a single mother with a strong gender schema may experience distress from having to compromise her role as mother and caretaker in order to fulfill the traditionally masculine role of breadwinner. Similarly, if antisocial behavior is described in terms more readily associated with men, then feminine individuals who engage in such behavior may experience gender role strain. As such, they may be more prone to psychological distress (e.g., anxiety, depression, and somatization). This argument is highly consistent with Lilienfeld's (1992) negative emotionality model.

Previous Tests of Lilienfeld's (1992) Hypotheses

For other reasons, one must be open to the possibility that using biological sex as a moderator may not adequately explain the antisociality-somatization relationship. Previous work has generally found weak or no support for sex differentiation. For example, Wilson and colleagues (1999) used the subscales measuring antisocial-impulsive traits from three measures of psychopathy to obtain a composite scale representing impulsivity-antisocial lifestyle. They also examined a measure of somatization. The correlations between these measures revealed a similar relationship for men and women (r = .33 for men; r = .35 for women). They also compared men's and women's scores on the composite scales and the somatization scale. Women scored significantly higher on somatization, but there was no significant difference between men and women on the composite impulsivity-antisocial lifestyle scale. The authors concluded that, because there were no differences between men and women in the pattern or sizes of the correlations, their study did not support a sex differentiation hypothesis. A later analysis of this data by Cale and Lilienfeld (2002) also concluded that there were no differences between men and women in the relationship between antisocial and somatic symptomatology, further negating the sex differentiation hypothesis.

Other work has only tested parts of these theories. Frick, Kuper, Silverthorn and Cotter (1995) examined the behavioral disinhibition hypothesis in a sample of mothers of clinic-referred children. They examined the correlations between two measures of somatic symptoms and two measures of antisocial symptoms. They also examined levels of sensation seeking as an indicator of behavioral disinhibition. The correlations among these scales revealed that three of the four possible correlations between antisocial behavior and somatization were significant (.37, .36, and .39). Sensation seeking had a small association with each measure of somatization (.23 and .24) and with one measure of antisocial behavior (.28). The authors concluded that the significant associations between sensation seeking and somatization provided preliminary support for the behavioral disinhibition hypothesis. However, they acknowledged having found only small support for the relationship between antisocial behavior and sensation seeking in women.

Lilienfeld and Hess (2001) tested both the behavioral disinhibition and negative emotionality models. They examined sex differences on antisocial-impulsive traits using the relevant subscales of three self-report measures of psychopathy. Two measures of somatization were used to compute a composite measure of somatization, and two measures of negative

emotionality were used to make a composite scale of negative emotionality. Also, they collected measures of behavioral inhibition and behavioral activation (the BIS/BAS scales; Carver, & White, 1994) as indicators of behavioral disinhibition. The authors found that men scored higher than women on two of the three measures of antisocial-impulsive traits. Men and women did not differ in levels of somatic symptomatology. Men and women also did not differ in levels of BAS, but women showed significantly higher levels of BIS. The BIS measure was negatively correlated with one of the three measures of antisocial-impulsive traits (-.29). BAS was positively correlated with two measures of antisocial-impulsive traits (.27 and .17). Neither BIS nor BAS were significantly correlated with the composite somatization scale. There were no significant relationships between any of the three measures of antisocial-impulsive traits and somatization for men, while there were significant positive relationships with all three measures for women (.35, .40, and .50). When the sample was combined, the relationships between all three measures antisocial-impulsive traits and somatization were positive and significant in all three cases (.28, .31, and .42). Correlations between antisocial-impulsive traits and the composite negative emotionality measure were reported only for the entire sample (.19, .48, and .55), as was the relationship between the composite negative emotionality measure and composite somatization (.62).

Lilienfeld and Hess (2001) proceeded to examine part of the negative emotionality model by testing for an interaction effect of antisocial-impulsive traits and biological sex with somatization as the dependent variable.[1] For two measures of antisocial-impulsive traits, the interaction with biological sex was significant for each using a p-value of .05 and the R^2 change for the interactions did not exceed .03. The authors tested the mediation effects of negative emotionality on the relationship between antisocial-impulsive traits and somatic symptomatology. Partial correlations controlling for negative emotionality reduced the correlations between somatization and each of the three antisocial measures: from .31 to .02 for, from .42 to .13, and from .25 to .17. Only the correlation of .17 remained significant.

In sum, Wilson, Frick and Clements (1999) found no sex differences in the relationships between antisocial and somatic symptoms, and Cale and Lilienfeld (2000) also found no differences between the sexes in the magnitude of this relationship in Wilson et al's sample.

[1] Although a proper test of the negative emotionality model (according to Lilienfeld, 1992) would have required the authors to use negative emotionality as the dependent variable.

Frick and colleagues (1999) found preliminary support for the behavioral disinhibition hypothesis, and did not have a sample of men against which they could compare their findings for women, thus allowing for no insights on sex differentiation. Lilienfeld and Hess's (2001) found support for the mediation effect proposed by the negative emotionality model. They further found some support for the effect of BIS and BAS on antisocial symptoms, but none for somatization. Finally, having found some evidence of a sex difference in antisocial behavior but no sex difference in somatic symptoms, they found weak support for sex differentiation. Thus, across these three studies, little information exists to infer there is utility in either the behavioral disinhibition or negative emotionality models. Additionally, even a liberal interpretation of the findings provides weak support for an effect of biological sex on the relationship between somatization and antisociality, and alternatives to this model should be explored. It is also necessary to consider that biological sex will not adequately explain the apparent sex differentiation of antisocial behavior and somatization and alternative moderating variables, such as gender role, should also be explored.

Narrowing Negative Emotionality to Anxiety

Lilienfeld and Hess (2001) found evidence to support the idea that negative emotionality accounts for the relationship between antisocial and somatic symptoms. However, John and Srivastava (1999) put it best when they stated that broad personality factors such as negative emotionality "are to personality what the categories 'plant' and 'animal' are to the world of biological objects—extremely useful for some initial rough distinctions but of less value for predicting specific behaviors of a particular object" (p.124). Indeed, Tellegen (1985) defines negative emotionality as the predisposition toward *a variety* of negative affects. Further, there exists the general finding that negative emotionality appears to be related to many psychological disorders, including several substance use disorders (Elkins, King, McGue, & Iacono, 2006), eating disorders (Cassin & von Ranson, 2005), personality disorders (Trull, Widiger, & Burr, 2005), and most mood and anxiety disorders (Watson, Gamez, & Simms, 2005). These facts call the usefulness of this Lilienfeld and Hess' findings—and the usefulness of the negative emotionality model—into question, as they infer that a significant mediation effect of negative emotionality tells us nothing about what facet of negative emotionality accounts for this effect. When examining of possible mediators of the antisociality-somatization relationship proposed in the negative emotionality model, researchers may want to consider those traits that are more

narrow facets of negative emotionality, and one area worth exploration is the construct of anxiety.

Anxiety and somatization. Somatic symptomatology has been consistently found to have a strong relationship with anxious and depressive symptomatology (APA, 2000; Dhossche, Ferdinand, van der Ende, & Verhulst, 2002; Rief, Schaefer, Hiller, & Fichter, 1992; Simon, & VonKorff, 1991). Other studies have found that number of somatic symptoms significantly predicts anxious and depressive symptomatology. For example, using data from the Epidemiological Catchment Area (ECA) study, Simon and VonKorff (1991) found that the risk for depressive disorder and phobia diagnoses increased as the number of somatic complaints increased. Similarly, Dhossche, et al. found that number of somatic complaints significantly predicted concurrent depressive and anxiety disorder diagnoses, but not substance use disorder or ASPD diagnoses. Most recently, Haug, et al. (2004) examined the relationships between somatic, depressive, anxious, and comorbid anxious/depressive symptomatology. The authors found a positive relationship between somatic symptomatology and each of their three anxious and depressive symptom measures. Additionally, they found that the relationships between somatic and anxious symptoms and somatic and depressive symptoms were similar, but comorbid anxiety/depression resulted in greater risk for somatic symptomatology than did anxious or depressive symptoms alone. Finally, although previous work has found that somatic symptomatology predicts concurrent anxious and depressive diagnoses, it should be noted that Dhossche, et al. (2002) failed to find that somatic symptomatology in adolescence was predictive of any mental disorder in young adulthood, including depressive and anxiety diagnoses. Thus, it seems more likely that if a causal relationship exists, it is that depressive and anxious symptomatology cause somatic symptomatology, rather than the other way around.

Anxiety and antisociality. Several studies have demonstrated elevated rates of anxiety disorders in incarcerated samples (Bland, Newman, Thompson, & Dyck, 1998; Powell, Holt, & Fondacaro, 1997; Teplin, Abram, & McClelland, 1996). Often in these studies, anxiety disorders were the second or third most common axis I disorder present, behind substance use disorder or substance use disorder and major depressive disorder. Regarding other samples, Walker, et al. (1991) reported the rates of conduct disorder and anxiety disorders in a sample of 177 clinic-referred boys. Of the 68 boys who met criteria for conduct disorder, 62% also met criteria for an anxiety disorder. Compare this to the 29% who met criteria for major depression and 19% who

met criteria for dysthymia. Of the boys who met criteria for an anxiety disorder, 40% met criteria for CD, compared to 21% who met criteria for depression and 15% who met for dysthymia.

Others have used large-scale community surveys to examine the comorbidity of anxiety disorders and antisocial behaviors. Sareen, Stein, Cox, and Hassard (2004) used two epidemiological samples (Ontario Health Survey [OHS], and the National Comorbidity Study [NCS]) to examine the comorbidity rates of any anxiety disorder, individual anxiety disorder diagnoses and antisocial diagnoses (conduct disorder, adult antisocial behavior, and ASPD). In the OHS sample, they found that 36% of individuals with an antisocial diagnosis had experienced an anxiety disorder in their lifetime. In the NCS sample, the rate was 48%. These numbers were compared to those in each sample that did not have an antisocial diagnosis controlling for age, sex, and education level. Adjusted odds ratios (AORs) of 3.88 for the NCS data and 2.82 were found for the OHS data, indicating that those with an antisocial diagnosis are roughly three times more likely to also have a lifetime diagnosis of any anxiety disorder after controlling for demographic characteristics.

Goodwin and Hamilton (2003) also examined the NCS data to investigate the relationships between lifetime CD or ASPD diagnosis and lifetime anxiety disorder diagnoses. They examined the risk of ASPD or CD in those with anxiety disorders. Their results indicated that, after controlling for demographic characteristics, those with a history of any anxiety disorder were more than two times as likely to have CD than those without a lifetime diagnosis of any anxiety disorder (AOR= 2.33). Those with a history of any anxiety disorder were three times as likely to have an ASPD diagnosis compared to those without a history of any anxiety disorder (AOR 2.9). In sum, several studies corroborate a relationship between somatization and anxiety, as well as anxiety and antisociality. Individuals with antisocial diagnoses are much more likely to also suffer from anxiety disorders than individuals without an antisocial diagnosis. Anxious and depressive symptoms reliably predict somatic symptomatology, but anxiety bears a stronger relationship to antisocial symptoms than depression. Further, although individuals with antisocial diagnoses are also more likely to suffer from other comorbid disorders, especially depression and substance use disorders, the relationship between anxiety and antisociality remains even after controlling for demographic variables. These findings are highly consistent with the negative emotionality model. Further, given these facts, it is reasonable to speculate that the effect of negative emotionality might be accounted for by anxious symptomatology. As such,

the current study tested a revised negative emotionality model predicting that anxiety would account for the relationship between antisocial and somatic symptomatology.

Narrowing Behavioral Disinhibition to (Lack of) Premeditation

Lilienfeld and Hess (2001) found no relationship between BIS/BAS measures and somatization, whereas Frick, et al. (1995) found weak evidence for a relationship between sensation seeking and somatization in women. Lilienfeld and Hess argued that they did not find clear support for a relationship between antisocial-impulsive traits and behavioral disinhibition in a combined sample of men and women. However, Lilienfeld and Hess may have overlooked some important information regarding their findings in this area. They note that a single significant correlation between BIS and the three antisocial-impulsive measures is poor support for the behavioral disinhibition hypothesis. On the other hand, Fowles (Fowles, 1987; Fowles, 2000; also see Newman, MacCoon, Vaughn, & Sedah, 2005, for empirical evidence corroborating this prediction) argues that BAS is more relevant to the reward sensitivity that is characteristic of antisocial behavior. Reexamination of Lilienfeld and Hess' BIS/BAS data with Fowles argument in mind actually yields reasonable support for a relationship between antisocial behavior and behavioral disinhibition, as two of three antisocial-impulsive scales were positively related to BAS (.27 and .17), albeit weakly related. Taking into mind this reinterpretation, Lilienfeld and Hess' findings are somewhat stronger in favor of the behavioral disinhibition model, especially when combined with Frick and colleagues (1995) finding of a small relationship between a measure of antisocial behavior and the sensation seeking in a sample of women. Thus, the behavioral disinhibition hypothesis merits further investigation.

Elkins, et al. (2006) defined behavioral disinhibition as a general tendency to fail to inhibit behavior. Other researchers have found that behavioral disinhibition can be underlain by a variety of traits. For example, although Lilienfeld and Hess (2001) used BIS and BAS to operationally define behavioral disinhibition, they were actually measuring insensitivity toward punishment and reward sensitivity; or rather, specific traits that lead to disinhibited behavior. Similarly, Frick, et al. (1995) operationally defined behavioral disinhibition with a measure of sensation seeking, meaning they actually measuring a more specific trait that leads to behavioral disinhibition. Thus, behavioral disinhibition may be too broad a construct to use effectively in predicting the mechanism that underlies Lilienfeld's (1992) proposed sex differentiation. Therefore, further investigation of the behavioral disinhibition hypothesis should first better

specify what aspects of behavioral disinhibition might be involved in antisocial behavior or somatization. Recent research on impulsivity can provide more insights in this area.

Incorporating new conceptualizations of impulsivity. In a factor analysis of 10 existing scales that relate to impulsivity, Whiteside and Lynam (2001) found evidence for a four-factor model of impulsivity and created the UPPS Impulsive Behaviour Scale to reflect these four factors. They argue that each of these factors may represent a pathway to different kinds of impulsive behavior. Most relevant to the current study are the factors (*lack of) premeditation*, which refers to an inability "to think and reflect on the consequences of an act before engaging in that act," (p. 685), and *sensation seeking*, which refers to "a tendency to enjoy and pursue activities that are exciting and an openness to trying new experiences that may or may not be dangerous," (p. 686).

Lynam and Miller (2004) found these two factors to be related to antisocial behavior and substance abuse in a community sample of adults. After replicating the UPPS factor structure in a sample of undergraduates, they used structural equation modeling to examine the relationships between the factors and several indicators of antisocial behavior. They found that (lack of) premeditation significantly predicted childhood conduct problems ($\beta = .47$) and several forms of substance use in youth (heavy alcohol, $\beta = .16$; marijuana, $\beta = .23$; and "harder" drug use, $\beta = .19$ for men and $\beta = .08$ for women). In addition, (lack of) premeditation predicted adult antisocial behavior ($\beta = .12$) and alcohol ($\beta = .05$) and marijuana abuse ($\beta = .04$) in adulthood. Finally, (lack of) premeditation was related to participants' generation of aggressive responses ($\beta = .06$) and choosing of aggressive responses ($\beta = .13$) in response to hypothetical hostile vignettes. Sensation seeking was also found to be important, but predicted fewer forms of deviance: conduct problems for men only ($\beta = .39$), heavy alcohol ($\beta = .08$) and marijuana ($\beta = .08$) use in youth, and alcohol abuse in adulthood ($\beta = .02$).

Miller, Flory, Lynam, and Leukfield (2003) found similar results. (Lack of) premeditation significantly predicted ASPD diagnosis ($\beta = .28$), variety of conduct problems ($\beta = .28$), and total score on an index of psychopathy ($\beta = .32$). In addition, it predicted cigarette ($\beta = .33$), alcohol ($\beta = .30$), and other drug use problems ($\beta = .16$), as well as number of sexual partners ($\beta = .26$) and age of sexual debut ($\beta = -.13$). Sensation seeking predicted half of these behaviors: ASPD ($\beta = .13$), a variety of conduct problems ($\beta = .15$), alcohol ($\beta = .21$) and other drug use ($\beta = .13$), and number of sexual partners ($\beta = .12$). In every instance, (lack of)

premeditation was the better predictor. Thus, since Lynam and colleagues' current conceptualization of impulsivity shows significant promise in the prediction of antisocial behaviors, it seems reasonable to propose that the (lack of) premeditation facet might be more useful in examining Lilienfeld's (1992) behavioral disinhibition model than would be the broad construct of behavioral disinhibition. Thus, in this study behavioral disinhibition was replaced with (lack of) premeditation in Lilienfeld's model. Under this revised behavioral disinhibition model, it was predicted that (lack of) premeditation will predict antisocial and somatic symptomatology.

The Current Study

The current study sought to test the behavioral disinhibition and negative emotionality models as originally proposed by Lilienfeld (1992), but also tested "modified" versions of these models. Specifically, the following predictions under the behavioral disinhibition model, taking into consideration the refinements made thus far: first, (lack of) premeditation will predict antisocial and somatic symptomatology. Second, under the sex differentiation hypothesis, these relationships will be moderated by sex, such that the relationship between (lack of) premeditation and antisocial symptomatology will be stronger for men compared to women and the relationship between (lack of) premeditation and somatic symptomatology will be stronger for women compared to men.

The current study makes the following predictions under the negative emotionality model, taking into consideration the refinements made thus far: first, the interaction of biological sex and antisocial symptomatology will predict anxious and somatic symptoms. Second, anxious symptomatology will predict somatic symptomatology. Third, the relationship between antisocial and somatic symptoms will be accounted for by anxiety

Further, because previous research in this area has found inconsistent evidence regarding the moderating effects of biological sex, the current study will also examine the moderating effects of gender role, by repeating these same analyses with gender role in place of biological sex. It was predicted that gender role orientation would have the moderating effects predicted of biological sex, and a model utilizing gender role would better explain patterns in the data than a model utilizing biological sex.

CHAPTER 2

METHODS

Participants

Participants were 206 undergraduate students at a southeastern state university who received course credit for their participation in this study. All students were treated in accordance with the ethical guidelines for research put forth by the American Psychological Association and the university's Internal Review Board. The sample consisted of 98 (47.6%) men and 108 (52.4%) women. The average age of the sample was 19.3 years (SD = 1.49). Regarding race, 163 (79.1%) self-identified as white, 25 (12.1%) as black, 4 (1.9%) as Asian, 3 (1.5%) as American Indian, and 11 declined to report race (5.4%). Five (2.5%) individuals identified as biracial. Regarding ethnicity, 28 (13.6%) participants self-identified hispanic/latino, 172 (83.5%) as non-hispanic/latino, and 6 (2.9%), declined to report ethnicity.

Power analysis. A power analysis revealed that, with a sample size of 206, an alpha level of .01 and five predictors (the greatest number of predictors that were used in the current study), power to detect a medium effect ($f^2 = .15$; Cohen, 1988) was .97.

Instruments

Barratt Impulsiveness Scale-11 (BIS-11, Patton, Stanford, & Barratt, 1995). The BIS-11 is a 30 item self-report questionnaire designed to assess general impulsiveness. It consists of three subscales that reflect second order factors of the construct of impulsiveness as argued by Patton, et al. These are attentional impulsiveness, motor impulsiveness, and non-planning impulsiveness. A total impulsiveness score can also be derived by summing the subscales, and it was this information from the BIS-11 that was utilized in the current study. Participants rate themselves on each of the items on a scale of one ("Rarely/Never") to four ("Almost Always/Always"). Internal consistency reliability (Cronbach's alpha) for the BIS-11 total score in this sample was .83.

Bem Sex Roles Inventory-Short Form (BSRI-SF, Bem, 1978). The BSRI-SF is a 30 item self-report questionnaire designed to assess the extent to which a person describes him or herself with traditionally or stereotypically masculine and feminine characteristics. It consists of two subscales, masculinity and femininity, each of which is comprised of personality characteristics that have been rated as more socially expected of men or women in American

society, respectively. Participants rated these characteristics on a scale from one (‘never or almost never true”) to seven (“always or almost always true”) in reference to the extent to which they feel that characteristic is true of them. Test takers can be divided into four categories depending on their scores on the masculinity and femininity scales: androgynous (high on both scales), undifferentiated (low on both scales), masculine (high on masculinity and low on femininity) or feminine (high on femininity and low on masculinity), or the scores on masculinity and femininity can be examined dimensionally, as was done in the current study. In this sample, internal consistency scores were .80 for masculinity and .90 for femininity.

Positive and Negative Affect Schedule (PANAS, Watson, Clark, & Tellegen, 1988). The PANAS is a 20-item self-report measure designed to assess these two dimensions of mood. The current study was concerned only with the negative affect subscale, which measures one’s report of distress and unpleasurable engagement that is characteristic of a variety of negative moods. This measure can be administered as a state measure (“you feel this way right now,”) or a trait measure (“you generally feel this way”). When administered with the latter instructions, the measure is considered a good indicator of negative emotionality (Watson, et al.). That is, measuring ones general experience of negative mood serves as an effective analogue to one’s level of negative emotional reactivity. Thus, it was in this manner that the PANAS was used in the current study. Participants rated items on a scale of one (“very slightly or not at all”) to five (“extremely). Internal consistency for the negative affect scale was .85 for the current sample.

Stop Task (Logan, Schachar, & Tannock, 1997). The stop task is a computerized performance task designed to measure one’s ability inhibit a prepotent response in the presence of a signal to inhibit that response. As such, it is a measure of inhibitory control (Logan, et al.). Participants are instructed to respond to a stimulus on the computer screen by pressing a particular key on the computer’s keyboard. A full description of the task can be found in Logan et al. The variable of interest in this task is stop-signal reaction time, which is an estimate of the latency of the participant’s response to the signal to stop their prepotent response. Longer stop signal reaction times are indicative of poor inhibitory control. In support of this, Logan and colleagues have found long stop signal reaction times are indicative of children with attention deficit/hyperactivity disorder (AD/HD; Schachar, & Logan, 1990; Schachar, Tannock, Marriott, & Logan, 1995), and similar results have been found in adults with AD/HD (Aron, Dowson, Sahakian, & Robbins, 2003).

Structured Interview for the DSM-IV, Axis II, Personality Disorders (SCID-II; First, Gibbon, Spitzer, Williams, & Benjamin, 1997). The SCID-II is a semi-structured interview designed to assist in diagnosing any of the 10 personality disorders listed in the DSM-IV, and is accompanied by a self-report screening questionnaire. The participant completes the screening questionnaire by answering "YES" or "NO" to in regards to whether or not they feel the item applies to them. Participants in this study completed all items of the screener relevant to cluster B personality disorders only. In this study, the interviewer inquired only about those items which are answered "YES", except for the items of the ASPD module that measure criterion A of the disorder (i.e., "a pervasive pattern of disregard and violation of the rights of others since age 15"). Participants in this study were administered all criterion A items at interview. Further, this study was concerned with only items of the ASPD module; specifically, the seven items that measure criterion A of antisocial personality disorder and the 15 items that measures criterion B of antisocial personality disorder (i.e., "some evidence of conduct disorder before age 15"). The seven items of criterion A were considered individually in the current analyses, where as the 15 conduct disorder items were used to compute a single variable measuring "some evidence of conduct disorder before age 15". Trained student raters coded each symptom as any of the following: ? = "inadequate information", 1 = "absent or false", 2 = "sub threshold", or 3 = "threshold or true". Symptom counts were computed using "liberal" ratings. That is, where the rater coded either a 2 or a 3 the symptom was added into the total symptom count.

Inter-rater reliability (Cohen's kappa) was computed for a sub-sample of 90 participants (43.6% of the sample). For the seven items of criterion A, scores ranged from .73 to.92 with a mean kappa of .83, demonstrating acceptable inter-rater reliability. Kappa for six of the 15 individual conduct disorder items could not be computed because they were not endorsed on the screener by any of the 90 participants, and thus these items were not rated at interview. For the remaining nine conduct disorder items, kappas ranged from .91 to 1.0 with a mean kappa of .98.

Symptom Checklist 90 Revised (SCL-90-R, Derogatis, 1994). The SCL-90-R is a 90 item self-report inventory of psychological symptoms. It is comprised of nine non-overlapping symptom indices and three global distress indices. Participants were asked to rate how much they have been distressed or bothered by the symptom described in each item over the past two weeks. Participants rate each item from zero ("not at all") to four ("extremely"). This study

utilized the somatization symptom index, which measures perceived bodily dysfunction, and anxiety symptom index, which measures signs of general anxiety. Internal consistency for the current sample was .80 for somatization and .84 for anxiety. While these two measures have been found to be moderately to highly correlated, discriminant validity has been observed such that somatization is associated with self-report of pain and somatic complaints where anxiety is not (Arrindell, Barelds, Janssen, Bulwada, & van der Ende, 2006).

UPPS Impulsive Behavior Scale (UPPS; Whiteside & Lynam, 2001). The UPPS is a 45 item self-report measure of four facets of the trait of impulsivity. Whiteside and Lynam (2001) designed the UPPS as a measure that not only taps at impulsivity as a personality trait, but one whose factors also align with facets of the Five Factor Model (McCrae & Costa, 1990) of personality. The UPPS has four factors, each of which Lynam and Whiteside predict should be related to different kinds of impulsive behaviors. The current study was particularly concerned with the (lack of) premeditation scale, which measures "the ability to think through the potential consequences of his or her behavior before acting." Participants rate each item from one ('strongly agree") to 5 ("strongly disagree") in reference to the extent to which they agree the statement is true about them. Internal consistency reliability for (lack of) premeditation in this sample was .89. For this measure, lower scores indicate a lack of premeditation.

Procedure

Participants completed the abovementioned questionnaires, other questionnaires, the stop task and an interview administering the cluster B modules of the SCID-II in a two and a half hour time block. Participants completed the questionnaires in groups of up to three, and the stop task and clinical interview were administered individually. Most participants were administered the SCID-II by a single trained student rater. However, a sub-sample of 90 participants in this sample were administered the SCID-II with a primary rater, who conducted the interview, and a secondary rater, who did not participate in the interview but rated the participant's responses. The primary and secondary raters did not consult each other in regards to the ratings. This was done in order to have a sub-sample of participants for whom inter-rater reliability could be computed. After the measures and interview were completed, the participants received a debriefing form that articulated the aims of the study and contained contact information, to be used if they have any questions about the study.

CHAPTER 3

RESULTS

Means and standard deviations for all measures are presented in Table 1 for the whole sample, as well as by sex. Independent samples t-tests were computed for all means presented in the Table 1 with an a priori p-value set at .01. Results indicate that men and women differed significantly only on the femininity scale, with women scoring significantly higher than men on femininity (t [204] = 4.038, $p < .001$, $d = .57$).

Before beginning analyses all variables and interaction terms were z-scored for the purposes of centering the variables and allowing for examination of variables on the same scale of measurement. VIF and tolerance statistics for all measures were examined and determined not to be indicative of multicollinearity. Specifically, all VIF statistics were less than 10 and all tolerance statistics were greater than .2. Correlations among all variables used in this study are presented in Table 2.

Behavioral Disinhibition

Simple Effects

Simple effects for all variables used to test the behavioral disinhibition hypothesis are presented in Table 3. For these analyses and all subsequent analyses an a priori p-value of .01 was set to determine statistical significance. Analyses using antisocial behavior as the dependent variable indicate that femininity, masculinity, and premeditation were all significant predictors of antisocial behavior, whereas biological sex and stop signal reaction time did not predict antisocial behavior. Because masculinity and femininity were significant predictors of antisocial behavior, they were statistically controlled for in all analyses predicting antisocial behavior where they were not the predictor of interest. For analyses examining somatization as the dependent variable, none of the five main variables were significant predictors.

Hierarchical Regression Analyses Predicting Antisocial Behavior

Lilienfeld's (1992) hypotheses. Hierarchical regression analyses were used to examine Lilienfeld's assertion that the interaction of biological sex and behavioral disinhibition should predict antisocial behavior. Masculinity and femininity were entered in the first step, biological sex and stop signal reaction time were entered in the second step, and the interaction term for biological sex and stop signal reaction time were entered in the third step. Results indicate that

masculinity and femininity together accounted for 16.1% of the variance in antisocial behavior ($F [2, 170] = 16.4$, $p < .001$). Examination of the coefficients indicates that femininity was negatively predicted antisocial behavior (B = -.249, SE B = .072, β = -.244, $t [170] = -3.48$, $p < .01$) and masculinity positively predicted antisocial behavior (B = .334, SE B = .073, β = .322, $t [170] = 4.59$, $p < .001$). Addition of biological sex and stop signal reaction time in step 2 did not significantly improve the model ($R^2\Delta = .023$, $F [2, 168] = 2.38$, $p = .095$), and neither did the addition of the interaction term of biological sex and stop signal reaction time in step 3 ($R^2\Delta = .001$, $F [2, 167] = .23$, $p = .631$).

The analysis was repeated with femininity in place of biological sex, and with masculinity as a control variable. Masculinity was entered in step 1, femininity and stop signal reaction time were entered in step 2, and the interaction of femininity and stop signal reaction time was entered in step 3. Results indicate masculinity accounted for 10.2% of the variance in antisocial behavior ($F [1, 171] = 19.38$, $p < .001$). Examination of the coefficients again indicates that masculinity is positively related antisocial behavior (B = .331, SE B = .075, β = .319, $t [171] = 4.40$, $p < .001$). Addition of femininity and stop signal reaction time in step 2 significantly improved the model ($R^2\Delta = .078$, $F [2, 169] = 8.09$ $p < .001$), but this appeared to be due mostly to the addition of femininity (B = -.230, SE B = .072, β = -.225, $t [169] = -3.2$, $p < .001$), and less to stop signal reaction time (B = .141, SE B = .071, β = ,139, $t [169] = 1.969$, $p = .051$). Addition of the interaction term in step 3 did not significantly improve the model ($R^2\Delta = .027$, $F [1, 168] = 5.62$, $p = .019$).

The analysis was repeated a third time with masculinity in place of femininity. Femininity was entered in step 1 as a control variable, masculinity and stop signal reaction time were entered in step 2, and the interaction of masculinity and stop signal reaction time was entered in step 3. Results indicate femininity accounted for 5.8% of the variance in antisocial behavior ($F [1, 171] = 10.43$, $p < .01$). Examination of the coefficients again indicates that femininity negatively predicted antisocial behavior (B = -.245, SE B = .076, β = -.240, $t [171] = -3.23$, $p < .01$). Addition of masculinity and stop signal reaction time in step 2 significantly improved the model ($R^2\Delta = .123$, $F [2, 169] = 12.66$, $p < .001$), but this appears to be due mostly to the addition of masculinity (B = .324, SE B = .072, β = .313, $t [169] = 4.486$, $p < .001$), and less to stop signal reaction time (B = .141, SE B = .071, β = .139, $t [169] = 1.97$, $p = .051$). Addition of

the interaction term in step 3 did not significantly improve the model ($R^2\Delta$ = .014, F [1, 168] = 3.024, p = .084).

Modified behavioral disinhibition hypothesis. Three separate hierarchical regression analyses were conducted to examine the current hypothesis narrowing the construct of behavioral disinhibition to premeditation replacing biological sex with gender role identification will produce more consistent findings where previous work has not. First an analysis was conducted examining biological sex, premeditation, and the interaction of biological sex and premeditation using masculinity and femininity as control variables. A summary of the significant blocks is presented in Table 4a. Results indicate that masculinity and femininity together accounted a significant amount of the variance in antisocial behavior (F [2, 194] = 14.67, p < .001). Examination of the coefficients indicates that femininity was negatively predicted antisocial behavior (t [194] = -3.40, p < .01) and masculinity positively predicted antisocial behavior (t [194] = 4.28, p < .001). Addition of biological sex and premeditation step 2 significantly improved the model (F [2, 192] = 6.16, p < .01), and this appeared to be due to the significant effect of premeditation, (t [192] = -3.50, p < .01) but not biological sex (t [192] = 1.30, p = .196). Addition of the interaction in step 3 did not improve the model ($R^2\Delta$ = .00, F [1, 191] = .00, p = 1.00).

The second regression repeated these analyses using femininity in place of biological sex, and controlling for masculinity. A summary of the significant blocks is presented in Table 4a. Here, masculinity in step 1 again predicted antisocial behavior (F [1,195] = 16.85, p < .001), and the addition of femininity and premeditation in step 2 significantly improved the model (F [2, 193] = 11.86, p < .001), but addition of the interaction of femininity and premeditation in step 3 did not significantly improve the model ($R^2\Delta$ = .003, F [1, 192] = .788, p = .376).

The analysis was repeated a third time with masculinity in place of femininity, and a summary of the significant blocks is also presented in Table 4a. Femininity was entered in step 1 as a control variable, masculinity and premeditation were entered in step 2, and the interaction of masculinity and premeditation was entered in step 3. Results indicate femininity accounted for 4.9% of the variance in antisocial behavior (F [1, 195] = 10.10, p < .01). Examination of the coefficients again indicates that femininity negatively predicted antisocial behavior (B = -.226, SE B = .071, β = -.222, t [195] = -3.17, p < .01). Addition of masculinity and premeditation in

step 2 significantly improved the model (F [2, 193] = 15.43, $p < .001$). Addition of the interaction term in step 3 did not significantly improve the model (F [1, 192] = .899, $p = .344$).

Hierarchical Regression Analyses Predicting Somatization

The same series of hierarchical regression analyses were repeated using somatization as the dependent variable to complete testing of the behavioral disinhibition model and its modified model. However, given that of biological sex, femininity, masculinity and premeditation did not significantly predict somatization in the simple effects (see Table 3), no control variables were necessary. For all tests examining somatization, no point were significant findings produced. Model summaries of the six analyses conducted for somatization can be found in Table 4b.[2]

Negative Emotionality

Hierarchical Regression Analyses: Interaction Effects

Hierarchical regression analyses were used to examine Lilienfeld's assertion that the interaction of biological sex and antisocial behavior should predict negative emotionality. Antisocial behavior and biological sex were entered in step 1 and the interaction term for antisocial behavior and biological sex were entered in step 2. Results indicate that antisocial behavior and biological sex together accounted for 8.3% of the variance in negative emotionality (F [2, 195] = 8.87, $p < .001$). Examination of the coefficients indicates that this was due mostly to the effect of antisocial behavior (B = .269, SE B = .063, β = .273, t [195] = -3.96, $p < .001$) and less to biological sex (B = -.256, SE B = .138, β = -.128, t [195] = 1.862, $p = .064$). Addition of the interaction step 2 did not significantly improve the model ($R^2\Delta = .007$, F [1, 194] = 1.45, $p = .229$). Results were identical when the analysis was repeated using femininity and masculinity in place of biological sex. Antisocial behavior and femininity together accounted for 7.2% of the variance in negative emotionality (F [2, 195] = 7.51, $p < .01$), apparently due more to the effect of antisocial behavior (B = .240, SE B = .070, β = .244, t [195] = 3.45, $p < .01$) and less to femininity (B = -.069, SE B = .071, β = -.068, t [195] = -.964 $p = .34$). Addition of the interaction step 2 did not significantly improve the model ($R^2\Delta = .014$, F [1, 194] = 1.93, $p = .089$). Antisocial behavior and masculinity together accounted for 6.7 % of the variance in negative

[2] Given that neither premeditation scale or stop signal reaction time significantly predicted both antisocial behavior and somatization, and the complete lack of significant findings for somatization, all analyses for the behavioral disinhibition model were again run using a more traditional measure of impulsivity than either the stop task or the UPPS: the Barratt Impulsiveness Scale-11 (BIS-11, Patton, Stanford, & Barratt, 1995). With the exception that the BIS-11 significantly predicted somatization, results were identical to those reported here. A summary of these analyses are presented in Table 7.

emotionality (F [2, 195] = 7.038, $p < .001$), apparently due more to the effect of antisocial behavior (B = .251, SE B = .071, β = .255, t [195] = 3.54, $p < .001$) and less to masculinity (B = .015, SE B = .073, β = .015, t [195] = .210, $p = .83$). Addition of the interaction step 2 did not significantly improve the model ($R^2\Delta = .005$, F [1, 194] = 1.15, $p = .286$).[3]

Hierarchical Regression Analyses: Mediation Effects

Lilienfeld's (1992) hypothesis. The interaction effect as proposed in the negative emotionality model was not supported, thus tests of the intervening effects of negative emotionality and anxiety proposed in the current study was examined with the main effects. A summary of simple regression analyses for all variables used to test the negative emotionality model are presented in Table 5. It can be observed in Table 5 that antisocial behavior predicted anxiety, negative affect and somatization. Further, negative affect and anxiety each predicted somatization. Thus sufficient reason exists to test the current hypotheses that negative affect or anxiety account for the relationship between antisocial behavior and somatization.

A hierarchical regression predicting somatization was run in which antisocial behavior was entered at step 1 and negative affect was entered at step 2. A summary of this analysis is presented in Table 6. Results indicate that the addition of negative affect at step 2 significantly improves the model (F [1, 192] = 83.020, $p < .001$), and examination of the coefficients indicates that the effect of antisocial behavior is substantially reduced and becomes non-significant when negative affect is included in the model. A Sobel test further indicates a significant indirect effect of antisocial behavior on somatization (Sobel z =3.11, $p < .01$).

Modified negative emotionality model. A hierarchical regression predicting somatization was conducted in which antisocial behavior was entered at step 1 and anxiety was entered at step 2. A summary of this analysis is also presented in Table 6. Results indicate that the addition of anxiety at step 2 significantly improves the model (F [1, 196] = 127.89, $p < .001$), and examination of the coefficients indicates that the effect of antisocial behavior is substantially reduced and becomes non-significant when anxiety is included in the model. A Sobel test further

[3] In a final attempt to uncover an interaction effect that may predict both somatization and antisocial behavior, a three-way interaction model using impulsivity, negative affect, and gender (masculinity and femininity were also examined in place of gender) was tested. Of the six hierarchical regression analyses computed to examine this model, none revealed a significant effect of this three-way interaction on somatization or antisocial behavior. A summary of these analyses can be found in Tables 8a and 8b.

indicates a significant indirect effect of antisocial behavior on somatization (Sobel z =3.67, $p <$ .001).

When examining the simple effects in Table 5 and comparing the two analyses presented in Table 6, it can be observed that, in both cases, anxiety accounts for more variance in somatization than negative affect. Additionally, a multiple regression predicting somatization, in which negative affect and anxiety are entered simultaneously reveals that, while together they account for 46.5% of the variance in somatization (F [2, 192] = 83.32, $p < .001$), and both were significant predictors in the model when the other was held constant, anxiety was the better predictor (B = .47, SE B = .067, β = .468 t [192] = 7.049, $p < .001$ for anxiety, versus B =.28, SE B = .067, β = .287 t [192] = 4.32, $p < .001$ for negative affect).

Post Hoc Tests

To determine whether gender role would remain a significant predictor of antisocial behavior after accounting for (lack of) premeditation, a hierarchal regression analysis was conducted using in which antisocial behavior was entered as the dependent variable, premeditation was entered in step 1 as a control variable, and both masculinity and femininity were entered in step 2. Results of step 2 indicate that the addition of masculinity and femininity significantly contributed to the prediction of antisocial behavior above and beyond (lack of) premeditation ($R^2\Delta$ = .108, F [1, 193] = 12.757, $p < .001$), and both were significant predictors in the model when the other was held constant (B = .261, SE B = .068, β = .253 t [193] = 3.382, $p < .001$ for masculinity, (B = -.225, SE B = .066, β = -.221 t [193] = -3.385, $p < .01$).

To understand the unique effects of anxiety and negative affect on somatization, a multiple regression analysis was run in which somatization was entered as the dependent variable, and anxiety and negative affect were entered together in one step. Results indicate that a model including both negative affect and anxiety is a good predictor of somatization (R^2 = .465, F [2, 192] = 83.32, p < .001). Examination of the coefficients indicates that, both variables made significant contributions to prediction (B = .228, SE B = .067, β = .287 t [192] = 4.32 $p <$ .001 for negative affect; B = .470, SE B = .067, β = .468 t [192] = 7.049 $p < .001$ for anxiety).

To understand the unique effects of anxiety and impulsivity on somatization, a multiple regression analysis was run in which somatization was entered as the dependent variable, and anxiety and impulsivity were entered together in one step. Results indicate that a model

including both impulsivity and anxiety is a good predictor of somatization ($R^2 = .416$, F [2, 193] = 82.18, $p < .001$). Examination of the coefficients indicates that, only anxiety made a significant contribution to prediction (B = .627, SE B = .058, β = .623 *t* [193] = 10.74 *p* < .001 for anxiety, versus B = .062, SE B = .058, β = .061 *t* [193] = 1.06 *p* = .29 for impulsivity).

CHAPTER 4

DISCUSSION

Early research in behavioral genetics established that somatization and antisociality co-occur at greater than chance levels both within individuals and within families, and proposed that these two disorders share a common genetic diathesis that is expressed differentially depending on the biological sex of the individual. Lilienfeld (1992) proposed that two models to explain the co-occurrence of these disorders and the apparent sex-differentiated expression of these disorders; these are the behavioral disinhibition and negative emotionality models. Three previous studies have examined Lilienfeld's hypotheses. Of the two studies that examined sex differentiation, one found no support for a differential relationship between biological sex and self-report of symptoms, and a liberal interpretation of the second study's findings yields weak support for sex differentiation. Regarding the negative emotionality model, one study found support for the mediation effect proposed is this model. Regarding the behavioral disinhibition model, two studies found weak support antisocial symptoms, for an effect of behavioral disinhibition (as measured by sensation seeking and BIS/BAS) on somatization.

The current study sought to add to the literature on Lilienfeld's (1992) sex-differentiation hypothesis. The current study took note of the weak support for biological sex as a moderator of symptom expression, and additionally examined the effect of gender role. Further, the current study sought to use a more appropriate measure of behavioral disinhibition compared to previous studies, but also argued that the negative emotionality and behavioral disinhibition models could benefit from the use of more specific constructs (i.e., anxiety and (lack of) premeditation) that may better explain the relationship between somatization and antisociality.

Gender Role Versus Biological Sex

The current study found no evidence to support an effect of biological sex on antisocial behavior or somatization. No differences in mean levels of symptomatology were found in the current study and, more importantly, biological sex showed no utility in predicting antisocial behavior or somatization in any of the regression analyses conducted. This is consistent with previous studies that have not consistently found men and women to differ in levels of symptomatology, and found no support for biological sex as a predictor of antisocial behavior or somatization. However, gender role proved useful in predicting antisocial behavior. Averaging

across the analyses that included masculinity and femininity in the first step, together they accounted for roughly 14 % of the variance in antisocial behavior. A post-hoc analysis revealed that, after controlling for premeditation, masculinity and femininity together still accounted for 10.8% of the variance in antisocial behavior.

The nature of the relationship between gender role and antisocial behavior was found to be in line with previous research examining the effect of gender role on gender-typed behavior. Previous research has found that individuals who identify strongly with their gender role are more likely to engage in behaviors consistent with their gender role, regardless of their biological sex. Similarly, in the current study, masculinity showed a positive relationship with antisocial behavior, indicating that those who hold a strong masculine gender role may be more prone to exhibiting antisocial behavior. Also, femininity showed a negative relationship to antisocial behavior, indicating that those who hold a strong feminine gender role may be less prone to exhibiting antisocial behavior. This finding could have implications on the apparent sex difference in antisociality, as the fraction of women with serious antisocial symptoms or ASPD may actually consist of women who identify strongly with the masculine gender role. Future research should examine this assertion.

Gender role was not useful for predicting somatization. Within the context of previous research on gender role, this finding may imply that somatic symptoms are not strongly gender-typed and so masculine and feminine people may be equally likely to express somatic symptoms. The current findings on gender role do not help explain the differential prevalence of somatization between men and women. However, another avenue for clarification of this issue may lie in the relationship between somatization and anxiety. The current found that anxiety was the best predictor of somatization, and the gender difference in anxiety and most anxiety disorders very well established (APA, 2000; Eaton, et al., 1989; Grater, et al., 1998; Kessler et al., 1994). Future research might there for consider examining the apparent gender difference in somatization as a function of its strong relationship to anxiety. Indeed, Kroenke and Spitzer (1998), provide preliminary support for this contention.

The current study is among the first to examine the effect of gender role on somatic and antisocial symptomatology, and so an obvious recommendation for future research is replication of these findings. Nevertheless, the positive findings of the current study underscore the need to consider that observed sex differences in antisocial behavior might actually be affected by

gender role. More generally, it highlights the importance of considering societal expectations of male and female behavior when examining apparent sex differences in behavior. Interestingly, where previous work highlighting this necessity has focused on prosocial behavior, and resolving gender-based injustices (Eagly, Koenig, Dindia, & Canary, 2006), the current study extends this caution to pathological behavior.

Sex Differentiation and the Behavioral Disinhibition Model

In the current study biological sex, masculinity, and femininity did not prove to be significant predictors of somatization. Further, none of the analyses examining the moderating effects of biological sex, masculinity, or femininity on either antisocial behavior or somatization supported the current hypotheses regarding sex-differentiation. Each of masculinity, femininity, and impulsivity (as measured by the UPPS premeditation scale and the BIS-11) made significant, independent contributions to the prediction of antisocial behavior, but the interaction of impulsivity with either biological sex or gender role did not improve prediction. The current results, combined with a similar lack of findings regarding sex-differentiation in previous studies, do not support the idea that somatization and antisocial behavior sex-specific manifestations of behavioral disinhibition or impulsivity. Neither do the current findings support the idea that gender role identification influences the expression of behavioral disinhibition or impulsivity.

The current study compared a measure of a narrower construct (i.e., premeditation) to a broader measure of behavioral disinhibition, expecting the more specific measure would better predict antisocial behavior and somatization. In fact the broad measure, stop signal reaction time, purported to be a pure measure of one's ability to inhibit behavior (Logan, et al., 1997), did not significantly predict antisocial behavior or somatization. Thus, the current findings lend support to this author's assertion that more specific measures of processes that lead to disinhibited behavior may be more useful in than broad measures, that do not take into account the many paths to disinhibited behavior (e.g., Whiteside & Lynam, 2001, Patton et al. 1995).

It was unexpected that stop signal reaction time did not predict antisocial behavior, as the role of behavioral dysregulation (measured in various ways) in the expression of antisocial behavior is well established in the literature. Further, antisocial behavior showed strong relationships with two measures of impulsivity in this study. In the current study, stop signal reaction time was modestly correlated with BIS-11 scores, but not with premeditation or

antisocial behavior as would be expected. Thus, one might argue that, while stop signal reaction time appears to be tapping impulsivity somewhat, it may not actually be a measure of general inhibitory control. For example, it might be better conceptualized as a measure of motor impulsivity (i.e., ability to inhibit pressing a key on the key board). If so it would clearly not be an appropriate proxy for behavioral disinhibition. Thus, the findings of the current study do not support the use of this measure as and indicator of behavioral disinhibition.

The Negative Emotionality Model

Antisocial behavior was found to predict somatic symptoms, and this is consistent with Wilson and colleagues (1999) and Lilienfeld and Hess (2001) findings of a significant correlation between somatic and antisocial symptoms. Thus, this aspect of the negative emotionality model appears to be reasonably well replicated. Antisocial behavior was also found to predict negative affect and anxiety, and the sizes of the relationships were highly similar. Anxiety was a slightly better predictor of somatization than negative affect when examined separately. In separate analyses, negative affect and anxiety were each found to fully account for the relationship between somatization and antisocial behavior, but anxiety appeared to have a somewhat stronger effect. A post hoc analysis revealed that, when examined together, anxiety was a better predictor of somatization than negative affect. As such, the more narrow construct served as the more useful predictor, and it can be inferred that the relationship between antisocial behavior and somatization is accounted for by the relationship each has to anxiety. However, it must be noted that the current study can make no direct inference about a causal path from antisocial behavior to somatization by way of anxiety. As noted earlier, research in somatization indicates a likely causal path from anxiety to somatization, but it is not clear from a review of the literature if a causal path exists from antisocial behavior to anxiety, as is predicted in the negative emotionality model. Additional longitudinal research is needed to address this question about the relationship between anxiety and antisocial behavior.

A notable limitation of the current study lies in the use of a non-clinical sample. Use of a non-clinical sample necessarily limits variability and amount of psychopathology that can be measured to test the current hypotheses. Further, use of a young college sample places particular limits on the degree of somatic symptomatology that can be uncovered, given that the criteria for this disorder allows fro an initial manifestation of symptoms up to age 30. A more appropriate sample would be one that allows for sufficient variability in somatic and antisocial symptoms for

both men and women, as well as more variability in age. One excellent suggestion is an adult incarcerated sample.

Additionally, a caution that must be noted lies in the relatively low correlation of somatization with antisocial behavior in this sample (.20) compared to that of other studies (.35 and .33 [Wilson, et al. 1999; .28, .31, and .42 [Lilienfeld & Hess, 2001]), which may have to do with a difference in measures used. Wilson, et al., used the Wahler Physical Symptoms Inventory (WPSI; Wahler, 1973) while Lilienfeld and Hess used a composite of the WPSI and SCL-90-R. Kirmayer and Robins (1991) describe three forms of somatization: multiple unexplained somatic symptoms, predominant illness worry or hypochondriacal beliefs, and somatization as a manifestation of major depression or an anxiety disorder. Behavior genetics research has demonstrated a relationship between antisocial personality and the first type (which is more indicative of somatization disorder). While evidence for discriminant validity between anxiety and somatization exists, it is possible that the somatization scale of the SCL-90-R may tap more at the third form of somatization, which would explain its moderately high correlations with negative affect and anxiety, and the lower than expected correlation with antisocial symptoms. On the other hand, it is quite possible the higher but more variable correlations between somatization and antisocial-impulsive traits found in previous studies is due to those studies use of subscales from self-report inventories of psychopathy, rather than the actual diagnostic criteria for antisocial personality disorder, as was used here. Regardless of the source of the variability observed in the relationship between somatization and antisocial behavior, one obvious recommendation for future researchers is to rely primarily on direct measures of the disorders, such as diagnostic interviews.

Behavioral genetics research has repeatedly found that antisociality and somatic problems occur at greater than chance levels with in families and within individuals, indicating a common genetic diathesis. Lilienfeld's (1992) hypothesis that this diathesis is a tendency toward behavioral disinhibition has been inconsistently supported. Frick, et al. (1995) found a small relationship between somatization and sensation seeking, but Lilienfeld and Hess (2001) found no relationship between BIS/BAS and somatization. The current study found a small relationship between somatization and one of three indicators of impulsivity, consistent with one of two previous studies. However, a post-hoc analysis revealed that the relationship between somatization and impulsivity disappeared when the effect of anxiety was accounted for. This

finding and previous findings together infer that if a common diathesis exists for antisocial behavior and somatization, it is unlikely the diathesis is a source of behavioral disinhibition. However future research would benefit from a test of this intervening effect using a more direct measure of somatization disorder.

Finally, future research should investigate whether a different facet of impulsivity bears a relationship to somatization disorder. Barratt and colleagues (Barratt, 1993,; Patton, et al., 1995) and Whiteside and Lynam (Lynam & Whiteside, 2004; Whiteside & Lynam, 2001) describe impulsivity as multifaceted, and this is reflected in the structure of each of their measures: each measure is comprised of subscales reflecting different facets of impulsivity as argued by each of the authors. Lynam and Whiteside (2004) in particular, have shown that different facets of impulsivity are related to different impulsive behaviors. Only the total impulsivity score of the BIS-11 was used, and only the premeditation subscale of the UPPS was used in the current study, but one might consider that a facet of impulsivity not examined in the current study might show a stronger relationship to somatization. Future research can investigate the remaining UPPS subscales, as well as the subscales of the BIS-11 for their effects on somatization. However, any presence of a relationship between a facet impulsivity and somatization would have to be examined for its value above and beyond anxiety.

In sum, the results of the current study provided support for the utility gender role in predicting antisocial symptoms, but not somatic symptoms. Further, it is among first to consider gender role in the expression of pathological behavior. The current results also provided support for the utility of using more narrow constructs in predicting antisocial and somatic symptoms. The current study found some support for a relationship between somatization and impulsivity as predicted by the behavioral disinhibition hypothesis, results indicate that the relationship between somatization and antisociality is better accounted for by anxiety. This finding is consistent with the negative emotionality model. Finally, despite finding more useful constructs with which to refine Lilienfeld's (1992) models, the sex-differentiation hypothesis remains largely unsupported. Specifically, the current study does not support the idea that somatization and antisociality are sex- (or gender role-) specific manifestations of behavioral disinhibition or impulsivity. Also, it does not support the idea that antisocial behavior differentially influences levels anxiety depending on sex (or gender role). The current study is arguably the strongest and most comprehensive test of Lilienfeld's sex differentiation hypothesis and previous studies have

also found weak support for it. As such, future research attempting to understand the relationship between antisociality, somatization and gender may benefit more from focusing on developing and testing alternative hypotheses.

Table 1
Sample Means and Standard Deviations for All Measures

Measure	Total Sample M (SD)	Men M (SD)	Women M (SD)
BIS-11	63.40 (10.45)	64.56 (10.27)	62.34 (10.56)
BSRI-SF Femininity	**5.38 (.90)**	**5.12 (.93)**	**5.61 (.81)**
BSRI-SF Masculinity	4.68 (.81)	4.64 (.79)	4.71 (.82)
PANAS NA	18.11 (5.59)	17.54 (5.57)	18.63 (5.58)
Stop Task SSRT	203.28 (46.81)	205.00 (48.25)	201.58 (45.55)
SCID-II ASPD "liberal" symptom count	1.80 (2.5)	2.07 (2.8)	1.55 (2.19)
SCL-90-R ANX	.41 (.47)	.40 (.48)	.41 (.46)
SCl-90-R SOM	.50 (.44)	.47 (.46)	.52 (.43)
UPPS premeditation	38.0 (11.0)	38.91 (12.65)	37.24 (9.16)

Note: Significant differences highlighted in **bold**.

Table 2
Correlations Among Variables Used in the Current Study

	1±	2	3	4	5	6	7	8	9	10
1	-									
2	.107	-								
3	-.253*	-.199*	-							
4	-.044	.106	.013	-						
5	-.105	.339*	-.122	.084	-					
6	.017	.299*	-.195*	.050	.209*	-				
7	.093	.355*	-.221*	.281*	.259*	.118	-			
8	-.028	.313*	-.041	.002	.607*	.158	.243*	-		
9	-.085	.256*	-.110	.009	.571*	.081	.200*	.646*	_	
10	.008	-.320*	.028	-.151	-.110	-.150	-.268*	-.168*	-.147*	-

Note: 1 = Biological Sex (males coded 1); 2 = BIS-11; 3 = BSRI-SF Femininity; 4 = BSRI-SF Masculinity; 5 = PANAS NA; 6 = Stop Task stop signal reaction time; 7 = SCID-II ASPD "liberal" symptom count; 8 = SCL-90-R ANX; 9 = SCl-90-R SOM; 10 = UPPS premeditation.
*$p < .01$
± Correlations in this column are Spearman's Rho.

Table 3
Summary of Simple Regression Analyses for Lilienfeld's (1992) and Modified Behavioral Disinhibition Model

DV = Antisocial Behavior			
Predictor	B	SE B	β
Sex	.210	.139	.105
Femininity	-.221	.068	-.221*
Masculinity	.283	.068	.281**
SSRT	.193	.076	.190
Premeditation	-.274	.070	-.268**
BIS-11	.361	.068	.356**

DV = Somatization			
Predictor	B	SE B	B
Sex	-.114	.143	-.057
Femininity	-.112	.072	-.110
Masculinity	.009	.071	.009
SSRT	.023	.079	.023
Premeditation	-.148	.072	-.147
BIS-11	.257	.070	.256**

*$p < .01$.
**$p < .001$.

Table 4a
Summary of Significant Blocks of Modified Behavioral Disinhibition Model

DV = Antisocial Behavior					
	B	SE B	β	R^2	$R^2\Delta$
Step 1				.131	.131**
Femininity	.296	.069	.287**		
Masculinity	-.232	.068	-.228*		
Step 2				.187	.056*
Femininity	-.200	.069	-.196*		
Masculinity	.262	.068	.254**		
Sex	.179	.138	.088		
Premeditation	-.236	.067	-.231*		
Step 1				.080	.080**
Masculinity	.292	.070	.282**		
Step 2				.180	.101**
Masculinity	.261	.068	.253**		
Femininity	-.225	.066	-.221*		
Premeditation	-.228	.067	-.224*		
Step 1				.049	.049*
Femininity	-.226	.071	-.222*		
Step 2				.180	.131**
Femininity	-.225	.066	-.221*		
Masculinity	.261	.068	.253**		
Premeditation	-.228	.067	-.224*		

*$p < .01$.
**$p < .001$.

Table 4b
Model Summaries for Regression Analysis for Lilienfeld's (1992) and the Modified Behavioral Disinhibition Model

DV = Somatization			
	R^2	$R^2\Delta$	F Change
Step1: Sex, SSRT	.006	.006	.516
Step 2: Sex X SSRT	.015	.009	1.449
Step1: Femininity, SSRT	.013	.013	1.078
Step 2: Femininity X SSRT	.022	.009	1.53
Step1: Masculinity, SSRT	.005	.005	.408
Step 2: Masculinity X SSRT	.019	.014	2.294
Step1: Sex, Premeditation	.024	.024	2.35
Step 2: Sex X Premeditation	.028	.004	.768
Step1: Femininity, Premeditation	.030	.030	3.00
Step 2: Femininity X Premeditation	.035	.005	.962
Step 1: Masculinity, Premeditation	.022	.022	2.11
Step 2: Masculinity X Premeditation	.027	.005	1.024

Table 5
Summary of Simple Regression Analyses for the Negative Emotionality Model

DV = Negative Affect			
	B	SE B	B
Sex	-.196	.142	-.098
Femininity	-.123	.072	-.122
Masculinity	.086	.072	.085
Antisocial Behavior	.255	.068	.259**

DV = Anxiety			
	B	SE B	B
Sex	-.008	.142	-.004
Femininity	-.042	.072	.041
Masculinity	.002	.071	.002
Antisocial Behavior	.240	.068	.243**

DV = Somatization			
	B	SE B	B
Antisocial	.199	.069	.200*
Negative Affect	.573	.059	.571**
Anxiety	.651	.055	.646**

*$p < .01$.
**$p < .001$.

Table 6
Summary for Mediation Tests of Lilienfeld's (1992) and the Modified Negative Emotionality Model

DV = Somatization					
	B	SE B	β	R^2	$R^2\Delta$
Step 1				.038	.038**
Antisocial Behavior	.194	.070	.196**		
Step 2				.329	.290***
Antisocial Behavior	.052	.060	.052		
Negative Affect	.560	.061	.558***		
Step 1				.040	.040**
Antisocial Behavior	.199	.069	.200**		
Step 2				.419	.379***
Antisocial Behavior	.045	.056	.045		
Anxiety	.640	.057	.635***		

*$p < .01$.
**$p < .001$.

Table 7
Footnote 2: Hierarchical Regressions Testing Behavioral Disinhibition Using the BIS-11

DV = Antisocial					
	B	SE B	β	R^2	$R^2\Delta$
Step 1				.125	.125**
Femininity	.282	.068	.276**		
Masculinity	-.229	.068	-.224*		
Step 2				.210	.085*
Femininity	-.156	.069	-.153		
Masculinity	.250	.066	.245**		
Gender	.092	.135	.046		
Impulsivity	.298	.067	.294**		
Step 3				.218	.008
Femininity	-.162	.069	-.159		
Masculinity	.249	.066	.243**		
Gender	-1.022	.807	-.505		
Impulsivity	.212	.090	-.209		
Gender X Impulsivity	.578	.413	.571		
Step 1				.074	.074**
Masculinity	.279	.070	.272**		
Step 2				.208	.134**
Masculinity	.249	.066	.243**		
Femininity	-.168	.066	-.165		
Impulsivity	.301	.066	-.296**		
Step 3				.213	.005
Masculinity	.245	.066	.240**		
Femininity	-.565	.371	-.554		

Table 7—continued

DV = Antisocial					
	B	SE B	β	R^2	$R^2\Delta$
Impulsivity	-.095	.370	.094		
Femininity X Impulsivity	.505	.465	-.499		
Step 1				.049	.049
Femininity	-.225	.071	-.221*		
Step 2				.208	.159**
Femininity	-.168	.066	-.165*		
Masculinity	.249	.066	.243**		
Impulsivity	-.301	.066	-.296**		
Step 3				.208	.000
Femininity	-.168	.067	-.165		
Masculinity	.222	.371	.217		
Impulsivity	.276	.335	.273		
Masculinity X Impulsivity	.038	.518	.038		

DV = Somatization					
	B	SE B	β	R^2	$R^2\Delta$
Step 1				.073	.073*
Gender	-.177	.140	-.088		
Impulsivity	.266	.070	.266**		
Step 2				.077	.004
Gender	-.895	.863	-.445		
Impulsivity	.211	.096	.211		
Gender X Impulsivity	.373	.443	.372		

Table 7—continued

DV = Somatization					
	B	SE B	β	R^2	$R^2\Delta$
Step 1				.068	.0068*
Femininity	-.050	.072	-.49		
Impulsivity	.247	.071	.247		
Step 2				.092	.024
Femininity	-.925	.396	-.909		
Impulsivity	-.627	.395	-.625		
Femininity X Impulsivity	1.116	.496	1.106		
Step 1				.066	.066
Masculinity	-.020	.071	-.019		
Impulsivity	.259	.070	-.259**		
Step 2				.066	.000
Masculinity	.050	.400	.049		
Impulsivity	.322	.360	.321		
Masculinity X Impulsivity	-.098	.557	-.098		

*$p < .01$.
**$p < .001$.

Table 8a
Footnote 3: Hierarchical Regressions Testing Three-Way Interaction

DV = Antisocial					
	B	SE B	β	R^2	$R^2\Delta$
Step 1				.157	.157**
Gender	.196	.136	.097		
Impulsivity	.291	.072	.287**		
Negative affect	.174	.072	.171		
Step 2				.180	.022
Gender	-.634	.845	-.313		
Impulsivity	-.241	.280	-.238		
Negative affect	-.564	.480	-.555		
Gender X					
Impulsivity	.756	.463	.746		
Gender X					
Negative affect	-.340	.248	-.339		
Impulsivity X					
Negative affect	1.056	.620	1.039		
Step 3				.196	.016
Gender	5.282	3.153	2.604		
Impulsivity	.244	.374	.241		
Negative affect	.302	.652	.298		
Gender X					
Impulsivity	-2.322	1.646	-2.289		
Gender X					
Negative affect	-3.509	1.646	3.5		
Impulsivity X					
Negative affect	-.086	.850	-.084		
Gender X					
impulsivity X					
negative affect	3.435	1.764	3.394		

Table 8a—continued

DV= Antisocial					
	B	SE B	β	R^2	$R^2\Delta$
Step 1				.168	.168**
Femininity	-.149	.069	-.145		
Impulsivity	.280	.072	.276**		
Negative affect	.150	.071	.148		
Step 2				.193	.024
Femininity	-.822	.392	-.800		
Impulsivity	-.626	.503	-.618		
Negative affect	-1.093	.651	-1.075		
Femininity X Impulsivity	.580	.512	.568		
Femininity X Negative affect	.432	.461	.425		
Impulsivity X Negative affect	1.094	.607	1.076		
Step 3				.204	.012
Femininity	-3.033	1.374	-2.951		
Impulsivity	-2.846	1.414	-2.808		
Negative affect	-5.236	2.552	-5.150		
Femininity X Impulsivity	3.468	1.794	3.395		
Femininity X Negative affect	5.035	2.780	4.955		
Impulsivity X Negative affect	6.583	3.326	6.478		
Femininity X impulsivity X negative affect	-5.722	3.409	-5.631		

Table 8a—continued

DV = Antisocial					
	B	SE B	β	R^2	$R^2\Delta$
Step 1				.202	.202**
Masculinity	.239	.066	.233**		
Impulsivity	.286	.069	.283**		
Negative affect	.146	.069	.143		
Step 2				.246	.044
Masculinity	148	.377	.144		
Impulsivity	.480	.409	.474		
Negative affect	-1.764	.613	-1.735*		
Masculinity X Impulsivity	-.868	.567	-.854		
Masculinity X Negative affect	1.492	.503	1.468*		
Impulsivity X Negative affect	.862	.588	.848		
Step 3				.247	.001
Masculinity	1.022	1.584	.997		
Impulsivity	1.321	1.535	1.303		
Negative affect	-.148	2.908	-.145		
Masculinity X Impulsivity	-2.148	2.323	-2.115		
Masculinity X Negative affect	-.417	3.394	-.410		
Impulsivity X Negative affect	-1.186	3.650	-1.167		
Masculinity X impulsivity X negative affect	2.335	4.107	2.298		

Table 8a—continued

DV = Somatization					
	B	SE B	B	R^2	$R^2\Delta$
Step 1				.330	.330**
Gender	-.030	.121	-.015		
Impulsivity	.070	.064	.070		
Negative affect	.547	.064	.545		
Step 2				.344	.013
Gender	-.365	.751	-.182		
Impulsivity	-.398	.250	-.398		
Negative affect	-.272	.428	-.270		
Gender X Impulsivity	.215	.413	.214		
Gender X Negative affect	-.051	.222	-.051		
Impulsivity X Negative affect	1.070	.553	1.068		
Step 3				.344	.000
Gender	.262	2.836	.130		
Impulsivity	-.346	.339	-.346		
Negative affect	-.179	.589	-.178		
Gender X Impulsivity	-.111	1.484	-.111		
Gender X Negative affect	-.387	1.481	-.390		
Impulsivity X Negative affect	.947	.769	.946		
Gender X impulsivity X negative affect	.364	1.588	.364		

Table 8a—continued

DV = Somatization					
	B	SE B	B	R^2	$R^2\Delta$
Step 1				.330	.330**
Femininity	-.015	.062	-.014		
Impulsivity	.065	.064	.065		
Negative affect	.549	.063	.547**		
Step 2				.382	.052*
Femininity	-.977	.342	-.952*		
Impulsivity	-1.658	.440	-1.656**		
Negative affect	.080	.570	.080		
Femininity X Impulsivity	1.555	.449	1.531*		
Femininity X Negative affect	-.521	-.405	-.518		
Impulsivity X Negative affect	1.226	.530	1.224		
Step 3				.385	.003
Femininity	-.2165	1.211	-2.11		
Impulsivity	-2.851	1.246	-2.847		
Negative affect	-2.141	2.246	-2.133		
Femininity X Impulsivity	3.111	1.587	3.064		
Femininity X Negative affect	1.955	2.455	1.946		
Impulsivity X Negative affect	4.172	2.930	4.166		
Femininity X impulsivity X negative affect	-3.082	3.014	-3.080		

Table 8a—continued

DV = Somatization					
	B	SE B	B	R^2	$R^2\Delta$
Step 1				.332	.332**
Masculinity	-.041	.061	-.040		
Impulsivity	.072	.063	.072		
Negative affect	.552	.063	.550		
Step 2				.347	.016
Masculinity	.271	.349	.267		
Impulsivity	-.202	.379	-.202		
Negative affect	-.089	.568	-.089		
Masculinity X Impulsivity	-.279	.524	-.279		
Masculinity X Negative affect	-.245	.466	-.244		
Impulsivity X Negative affect	1.093	.546	1.091		
Step 3				.359	.012
Masculinity	2.902	1.455	2.856		
Impulsivity	2.335	1.414	2.333		
Negative affect	4.773	2.672	4.754		
Masculinity X Impulsivity	-4.138	2.137	-4.128		
Masculinity X Negative affect	-5.982	3.116	-5.960		
Impulsivity X Negative affect	-5.079	3.359	-5.071		
Masculinity X impulsivity X negative affect	7.030	3.776	7.017		

*$p < .01$.
**$p < .001$.

Table 8b
Footnote 3: Hierarchical Regressions Testing Three-Way Interaction excluding Two-Way Interaction Terms

DV = Antisocial					
	B	SE B	β	R^2	$R^2\Delta$
Step 1				.157	.157**
Gender	.196	.136	.097		
Impulsivity	.291	.072	.287**		
Negative affect	.174	.072	.171		
Step 2				.158	.001
Gender	.098	.362	.048		
Impulsivity	.283	.077	.279**		
Negative affect	.159	.089	.156		
Gender X impulsivity X negative affect	.056	.190	.055		
Step 1				.168	.168**
Femininity	-.149	.069	-.145		
Impulsivity	.280	.072	.276**		
Negative affect	.150	.071	.148		
Step 2				.187	.018
Femininity	-.462	.164	-.449*		
Impulsivity	-.025	.162	-.024		
Negative affect	-.421	.282	-.415		
Femininity X impulsivity X negative affect	.756	.361	.744		

Table 8b—continued

DV = Antisocial					
	B	SE B	β	R^2	$R^2\Delta$
Step 1				.202	.202**
Masculinity	.239	.066	.233**		
Impulsivity	.286	.069	.283**		
Negative affect	.146	.069	.143		
Step 2				.223	.002
Masculinity	-.065	.147			
Impulsivity	.022	.133			
Negative affect	-.368	.232			
Masculinity X impulsivity X negative affect	.763	.329			

DV = Somatization					
	B	SE B	B	R^2	$R^2\Delta$
Step 1				.330	.330**
Gender	-.030	.121	-.015		
Impulsivity	.070	.064	.070		
Negative affect	.547	.064	.545**		
Step 2				.344	.013
Gender	-.179	.321	-.089		
Impulsivity	.058	.068	.058		
Negative affect	.524	.079	.522**		
Gender X Impulsivity X Negative affect	.084	.168	.084		

Table 8b—continued

DV = Somatization					
	B	SE B	B	R^2	$R^2\Delta$
Step 1				.330	.330**
Femininity	-.015	.062	-.014		
Impulsivity	.065	.064	.06588		
Negative affect	.549	.063	.547		
Step 2				.343	.013
Femininity	-.275	.148	-.268		
Impulsivity	-.187	.145	-.186		
Negative affect	.078	.252	.077		
Femininity X impulsivity X negative affect	.624	.323	.623		
Step 1				.332	.332**
Masculinity	-.041	.061	-.040		
Impulsivity	.072	.063	.072		
Negative affect	.552	.063	.550**		
Step 2				.333	.001
Masculinity	-.104	.136	-.103		
Impulsivity	.017	.123	.017		
Negative affect	.445	.214	.443		
Masculinity X impulsivity X negative affect	.159	.303	.159		

*$p < .01$
**$p < .001$.

APPENDIX A

STOP TASK INSTRUCTIONS

Directions for experimenter are in bold. Directions read to participant are in quotations.

"You are going to see some X's and O's appear on the screen in front of you. As soon as you see an X, you press this key (point to the X key on the number pad). And as soon as you see an O, you press this key (point to the O key on the number pad). A few times you will hear a tone; ignore it for now. A dot will always appear right before the letter. It is important to look at the dot because as soon as it disappears, the letter will appear and you will have to press the key. **Press the correct Key as quickly as you can without making mistakes."**

Position the subject so that their dominant hand is ready at the keys. They should use only one hand. If the subject is left handed, the keyboard can be slid over. It is better if they use two fingers (eg index and thumb) but if they prefer to use one finger, it's ok.

"Ok, here it goes."

At the end of the first block, say:

"That is the go part of the game. Now I'll show you the stop part of the game. You will do the same thing, pressing the right key for the X or the O as quickly as you can without making any mistakes, EXCEPT when the computer beeps. If you hear a beep, DON"T press the key. The time of the beep is varied by the computer. Sometimes you will be able to stop easily, other times it will be impossible. Nobody is able to stop every time. Just try to stop as often as you can. Don't let the stopping part of the game interfere with your speed on the go part of the game. Especially, don't wait for the beep—the beeps don't occur that often, so if you wait, you will be slower overall. Also, if you slow down, the computer will slow down so it won't be any easier to stop. So, just try to go your fastest without mistakes, and as often as possible, don't press the key when there's a beep. Ready? Let's begin."

At the end of the second practice trial, make sure the participant understands the task, then say:

"Okay, now we begin the main trials. There will be a few blocks, you'll be able to rest every few minutes if you want to. The rules are the same as on the practice. Press the X or the O as fast as you can without mistakes except when there is a beep."

APPENDIX B

UNIVERSITY INTERNAL REVIEW BOARD APPROVAL LETTER

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APPROVAL MEMORANDUM (for change in research protocol)

Date: 12/4/2006

To:
Joyce Carbonell
Mc 1270

Dept: PSYCHOLOGY DEPARTMENT

From: Thomas L. Jacobson, Chair

Re: Use of Human subjects in Research
Project entitled: Gender Role as a Moderator of Personality Traits

The memorandum that you submitted to this office in regard to the requested change in your research protocol for the above-referenced project have been reviewed and approved. Thank you for informing the Committee of this change.

A reminder that if the project has not been completed by 9/12/2007, you must request renewed approval for continuation of the project.

By copy of this memorandum, the chairman of your department and/or your major professor is reminded that he/she is responsible for being informed concerning research projects involving human subjects in the department, and should review protocols of such investigations as often as needed to insure that the project is being conducted in compliance with our institution and with DHHS regulations.

This institution has an Assurance on file with the Office for Protection from Research Risks. The Assurance Number is IRB00000446..

cc:
APPLICATION NO. 2006.0750-R

APPENDIX C

INFORMED CONSENT FROM

Study # 18: Personality Characteristics

Informed Consent Form

Joyce Carbonell, Ph.D., professor of psychology, has requested my participation in a research study here at Florida State University. The name of this study is Personality Characteristics. The purpose of the research is to understand how certain personality characteristics are related to each other, and how their relationships influence a person's behavior.

My participation will involve completing a number of questionnaires that ask about different feelings I experience and different ways in which I may behave or have behaved. My participation will also involve participating in an interview in which I will be asked about certain ways I may behave and about how I may think about myself and the things and people around me. Last, my participation will involve completing a computer exercise. My participation in this study will be from 2.5 to 3 hours. I understand that I will receive class credit (2.5 credits)for participating in this study; however, I will not be penalized if choose not to participate or withdraw from this study. I understand that I will not be paid any money for my participation in this study. If I leave the study before completion, I will receive credit for the time that I have participated. I will receive

I understand that, if I agree to participate in this study, there are minimal foreseeable risks or discomforts associated with completing the questionnaires, interview, and computer task. Although there may be no direct benefits to me, the possible benefits of my participation in the research include gaining greater knowledge about how personality characteristics interact to influence behavior.

The results of this research study may be published but my name or identity will not be revealed. The researcher will do the following to maintain confidentiality of my records: I will be instructed to NOT write my name on any of the questionnaires and experimenters will also NOT write my name on my questionnaires. The experimenter will assign me an arbitrary identification number, and a file containing my questionnaires, computer task results, and interview will be marked with this number. Notes from the interview and my questionnaires will also be marked with this number. These materials will be kept in a file cabinet and the file cabinet will be kept in a locked room. My informed consent form and a master list of names will be kept separate from the questionnaires, computer task results, and interview notes in a separate locked file cabinet in same locked room. Informed consent forms WILL NOT contain my arbitrary identification number, and no record will be kept that pairs my name with my arbitrary

identification number. Only the experimenters will have access to any of this information. All resulting databases will contain only the arbitrary identification number paired with my answers to the questionnaires and interview questions, and only the experimenters will have access to the databases. The master list of the names of those who participated in this study will be kept only for the purposes of assigning class credit, and will be destroyed after arbitrary identification numbers are assigned. The master list of names will NOT contain my arbitrary identification number. Records will be confidential too the extent allowed by law.

Any questions I have concerning the research study or my participation in it, either before or after my consent, can be answered by Dr. Carbonell (carbonel@psy.fsu.edu), Yezzennya Castro (castro@psy.fsu.edu), or the experimenter I receive. If I have questions about my rights as a subject/participant in this research, or if I feel I have been placed at risk, I can contact the Chair of the Human Subjects Committee, Institutional Review Board, through the Office of the Vice President for Research, at (850) 644-8633.

I have read the above informed consent form. I understand that I may withdraw my consent and discontinue participation at any time without penalty or loss of benefits to which I may otherwise be entitled. In signing this consent form, I am not waiving any legal claims, rights or remedies. A copy of this consent form will be given (offered) to me.

Participant's Name _____

Participant's Signature _____

Date _____

REFERENCES

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Zlotnick, C., Zimmerman, M., Wolfsdorf, B.A., & Mattia, J.I. (2001). Gender differences in patients with posttraumatic stress disorder in a general psychiatric practice. *American Journal of Psychiatry, 158*(11), 1923-1925.

BIOGRAPHICAL SKETCH

Curriculum Vita

Yessenia Castro, M.S.

Educational History

2004-present, Doctoral Candidate

Florida State University

Tallahassee, Florida

Major Area: Clinical Psychology

Major Professor: Joyce Carbonell, Ph.D.

September 2004, Passed Preliminary Doctoral Examination

2002-2004, Master of Science

Florida State University

Tallahassee, Florida

Major Area: Clinical Psychology

Major Professor: Joyce Carbonell, Ph.D.

1998-2002, Bachelor of Arts

University of Southern California

Los Angeles, California

Major Area: Psychology

Major Professor: Joseph Hellige, Ph.D.

Honors and Awards

Society for the Psychology of Women (Division 35) Hyde Graduate Student Research Grant: $ 500.00

Congress of Graduate Students Conference Presentation Grant: $ 300.00

University Fellowship for academic year 2004-2005

Current Research Interests

Issues of culture and gender in psychological assessment, culturally appropriate mental health services provision; gender differences in the expression of mental disorders, personality assessment.

Research Activities

Publications

Carbonell, J.L., & **Castro, Y.** (accepted for publication). The impact of a leader model on high dominant women's self-selection for leadership. *Sex Roles: A Journal of Research.*

Castro, Y., Gordon, K.H., Brown, J.S., Cox, J.C., & Joiner, Jr., T.E. (accepted for publication). Examination of racial differences on the MMPI-2 clinical and Restructured Clinical scales in an outpatient sample. *Assessment.*

Holm-Denoma, J.M., Gordon, K.H., Donohue, K.F., Waesche, M., **Castro, Y.**, Brown, J.S., Jakobsons, L.J., Merrill, K.A., Buckner, J., & Joiner, T.E. Jr. (accepted for publication). Patients affective reactions to receiving diagnostic feedback. *Journal of Social and Clinical Psychology.*

Van Orden, K. A., Witte, T. K., James, L. M., **Castro, Y.,** Gordon, K. H., Braithwaite, S. R., Hollar, D. L., & Joiner, T. E., Jr. (accepted for publication). Suicidal ideation in college students varies across semesters: The mediating role of belongingness. *Suicide and Life Threatening Behavior*.

Buckner, J.D., **Castro, Y.**, Holm-Denoma, J.M., & Joiner, T.E. Jr. (Eds.) (2007). Mental Health Care for People of Diverse Backgrounds. Abington, UK: Radcliffe Publishing.

Castro, Y., Buckner, J.D., & Holm-Denoma, J.M (2007). Introduction to empirically informed mental health services for diverse populations. In, J.D. Buckner, Y. Castro, J.M. Holm-Denoma, & T.E. Joiner, Jr. (Eds.), Mental Health Care for People of Diverse Backgrounds. Abington, UK: Radcliffe Publishing.

Gordon, K.H., & **Castro, Y.** (2007). The assessment, diagnosis, and treatment of psychiatric disorders in lesbian, gay, and bisexual clients. In, J.D. Buckner, Y. Castro, J.M. Holm-Denoma, & T.E. Joiner, Jr. (Eds.), Mental Health Care for People of Diverse Backgrounds. Abington, K: Radcliffe Publishing.

Hunter, L.R., Buckner, J.D., Holm-Denoma, J.M., & **Castro, Y.** (2007). The delivery of mental health services for clients of diverse backgrounds: Summary and future

directions. In, J.D. Buckner, Y. Castro, J.M. Holm-Denoma, & T.E. Joiner, Jr. (Eds.), Mental Health Care for People of Diverse Backgrounds. Abington, UK: Radcliff Publishing.

Lima, E., Stanley, S., Kaboski, B., Reitzel, L. R., Richey, A. J., **Castro, Y.**, Williams, F.W., Tannenbaum, K., Stellrecht, N., Jakobsons, L., Wingate, L.R., & Joiner, T.E. (2005). The incremental validity of the MMPI-2: When does therapist access not enhance treatment outcome? *Psychological Assessment, 17*(4), 462-468.

Professional Activities

Reviewer Activities

2007-present: Criminal Justice and Behavior, Ad Hoc Reviewer

Membership in Professional Associations

2006-present: American Psychological Association, Division 45, Society for the Study of Ethnic Minority Issues, student member

2006-present: American Psychological Association, Division 35, Society for the Psychology of Women, student member

2006-present: American Psychological Association, Division 12, Society for Clinical Psychology, student member

2003–2006: American Association for Correctional and Forensic Psychology, student member

2002-2003: Cognitive Neuroscience Society, student member

Professional Workshops Attended

April, 2007: MMPI-2/MMPI-A Workshops and 42nd Annual Symposia. Ft. Lauderdale, Florida.

March, 2006: Instituto de los Mexicanos en el Exterior, 33a Jornada Informativa: Salud Mental (Institute for Mexicans in the Exterior, 33rd Informative Conference: Mental Health). Guadalajara, Jalisco, Mexico.

Departmental Service

2006-2007: Clinical Graduate Student Advisory Committee. Brad Schmidt, Ph.D., Chair.

2006-2007: Committee for Diversity in Clinical Science. Jeanette Taylor, Ph.D., Chair.

2005-2007: Committee for Increasing Diversity in the Clinical Psychology Curriculum. Brad Schmidt, Ph.D., Chair.

2003-2006: Interview Weekend Committee. Joyce Carbonell, Ph.D., Chair.

2003-2005: Clinical Graduate Student Advisory Committee. Mark Licht, Ph.D., Chair.

Supervised Teaching Experience

Spring 2006, Spring 2007: Lecturer/Teaching Assistant

PSY 5325: Assessment Practicum

Florida State University Department of Psychology

Supervisors: Joyce Carbonell, Ph.D., and Kristen Schmidt, Ph.D., instructors.

Fall 2005, Fall 2006: Teaching Assistant

SOP 3742: Psychology of Women

Florida State University Department of Psychology

Supervisor: Joyce Carbonell, Ph.D., instructor.

Supervised Clinical Experience

October 2007-September 2008: Psychology Intern

Federal Bureau of Prisons

Federal Medical Center, Carswell

Fort Worth, Texas

Director of Clinical Training: Diana del Rio, Psy.D.

August 2006-August 2007: Psychological Trainee

Southwestern State Hospital

Thomasville, Georgia

Supervisor: Leonard Bailey, Ph.D.

July 2006-August 2007: Psychological Trainee

Kristen Schmidt, Ph.D.

Tallahassee, Florida

Supervisor: Kristen Schmidt, Ph.D.

August 2005-August 2007: Psychological Trainee

Florida State University Crisis Management Unit

Tallahassee, Florida

Supervisor: Joyce Carbonell, Ph.D.

October 2004-June 2006: Psychological Trainee

Panhandle Area Educational Consortium

Migrant Education Program

Quincy, Florida

Supervisor: Elena Reyes, Ph.D.

August 2003-April 2005: Psychological Trainee

Florida State University Psychology Clinic

Tallahassee, Florida

Supervisors: Thomas Joiner, Ph.D.; Donald Kerr, Ph.D.

August 2003-August 2004: Psychological Trainee

State of Florida Department of Juvenile Justice

Arthur G. Dozier School for Boys

Specialized Treatment Program

Mariana, Florida

Supervisor: Teion Wells Harrison, Ph.D.