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Psychopathic Personality Traits and Somatization: Sex Differences and the Mediating Role of Negative Emotionality

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Although a number of investigations have provided evidence for an association between antisocial personality disorder and somatization disorder, the variables underlying this association remain unknown. We examined the relations among measures of primary and secondary psychopathy, somatization, and negative emotionality (NE) in 150 undergraduates. Somatization was positively and significantly correlated with measures of secondary, but not primary, psychopathy, and the relations between secondary psychopathy indices and somatization tended to be significantly stronger in females than in males. Some support was found for the hypothesis that the association between secondary psychopathy and somatization is mediated by NE, but not for the hypothesis that low levels of behavioral inhibition lead to somatization. Although the present findings are consistent with the possibility that somatization is a sex-differentiated manifestation of secondary psychopathic traits, replication of these findings in clinical samples will be necessary.

KEY WORDS: psychopathy; antisocial personality disorder; somatization disorder; sex differences; negative emotionality.

The phenomenon of "comorbidity," or diagnostic covariation, represents one of the foremost challenges to contemporary psychopathology researchers (Caron & Rutter, 1991; Frances et al., 1992). Such covariation is particularly rampant among Axis II disorders (Grove & Tellegen, 1991) and is also prevalent between Axis I and Axis II disorders (Lilienfeld, Waldman, & Israel, 1994). Although the application of the term and concept of comorbidity to psychopathological syndromes is controversial (Lilienfeld et al., 1994; Spitzer, 1994), there is a general consensus that a better understanding of the covariation among diagnostic syndromes can provide valuable leads for research on shared etiological factors (Maser & Cloninger, 1990).

Of the comorbidities consistently reported in the psychopathological literature, one of the most enigmatic has been the association between antisocial personality disorder (ASPD) and somatization disorder (SD). The former syndrome is characterized by a chronic history of antisocial and criminal behavior, whereas the latter is characterized by a chronic history of unexplained physical complaints (American Psychiatric Association, 1994; Kirmayer, Robbins, & Paris, 1994). The covariation between ASPD and SD is superficially puzzling because these conditions share few, if any, overt diagnostic features. A sizeable body of evidence suggests that ASPD and SD covary within both individuals and families (see Cloninger, 1978; Lilienfeld, 1992, for reviews).

In a sample of 250 psychiatric patients, for example, Lilienfeld, VanValkenberg, Akiskal, and Larntz (1986) found that ASPD and SD were significantly associated within individuals ($r_q = .55$).[3] Moreover, a significantly higher proportion of probands with SD than without SD reported having a first-degree relative with ASPD ($r_q = .63$). More recently, Frick, Kuper, Silverthorn, and Cotter (1995) found moderate and statistically significant

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[3]Yule's Q (r_q) is an index of the magnitude of the association between categories that is unaffected by the base rates of the disorders in question (Fienberg, 1980).

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correlations between dimensional measures of ASPD and SD in a sample of 90 mothers of clinically referred children. Although Boyd et al. (1984) found no covariation between ASPD and SD in their analyses of the Epidemiological Catchment Area data, the low rates of SD in these data (approximately .1%) render these negative results difficult to interpret (see Cloninger & Guze, 1975; Cloninger, Reich, & Guze, 1975; Guze, Woodruff, & Clayton, 1971, Liskow, Othmer, Penick, DeSouza, & Gabrielli, 1986, Robins, 1966; Spalt, 1980; Zoccolillo & Cloninger, 1986, for further data suggesting significant levels of covariation between SD and ASPD, antisocial behavior, or both, across individuals). In addition, both ASPD and SD are associated with an early age of onset, chronic course, substance abuse, marital and familial discord, high rates of abuse and neglect of children, and poor response to psychotherapy and pharmacotherapy (Cloninger, 1978; Goodwin & Guze, 1996; Zoccolillo & Cloninger, 1986). These similarities provide suggestive, but by no means conclusive, evidence of shared etiological origins for ASPD and SD.

Several authors (e.g., Carothers, 1975; Gorenstein & Newman, 1980; Lilienfeld, 1992) have posited that the psychopathological correlates of SD extend beyond ASPD to psychopathic personality (psychopathy). Although measures of psychopathy tend to correlate moderately with measures of ASPD (Lilienfeld, 1994), the former syndrome, unlike ASPD, is traditionally conceptualized as a constellation of covarying personality traits. These traits, which were first delineated systematically by Cleckley (1941/1982; see also Lykken, 1957; McCord & McCord, 1964), include guiltlessness, callousness, dishonesty, egocentricity, risk taking, poor impulse control, and superficial charm.

A number of authors have argued that ASPD is considerably more etiologically heterogeneous than is psychopathy and incorporates both "primary" (Cleckley) psychopathy as well as a mélange of other conditions (e.g., "neurotic psychopathy," "dyssocial psychopathy") that place individuals at heightened risk for antisocial behavior (Hare, Hart, & Harpur, 1991; Lykken, 1995). These latter antisocial conditions are sometimes collectively referred to as "secondary" psychopathy. Indeed, factor analyses of psychopathy measures have typically revealed two separable dimensions, one corresponding to the core affective and personality deficits described by Cleckley, and the other corresponding to overt antisocial behavior (Harpur, Hare, & Hakstian, 1989). There is little evidence, however, bearing on the association between primary psychopathy and SD, because virtually all controlled studies of the diagnostic correlates of SD have focused on ASPD rather than on primary psychopathy per se (Lilienfeld, 1992).

Several authors (e.g., Goodwin & Guze, 1996; Lilienfeld, 1992) have further conjectured that ASPD and SD are sex-differentiated manifestations of a shared personality diathesis, although the precise nature of this diathesis remains to be determined. According to this hypothesis, males possessing this diathesis are likely to exhibit ASPD features, whereas females possessing this diathesis are likely to exhibit SD features. This "sex differentiation hypothesis" is consistent with data indicating that ASPD is more common in males than in females, whereas SD is more common in females than in males (American Psychiatric Association, 1994). This hypothesis is also consistent with findings demonstrating that males tend to exhibit higher levels of overt aggression than females do (Maccoby & Jacklin, 1974), although sex differences in "relational" (i.e., interpersonal) aggression are less pronounced and perhaps even reversed in direction (Eagly & Steffen, 1985; Werner & Crick, 1999). Thus, one potential interpretation of this literature is that the personality diathesis underlying ASPD and SD tends to be channeled into different phenotypes in males and females depending partly on levels of overt aggression. Nevertheless, there is at present little direct evidence in support of the sex differentiation hypothesis of ASPD and SD (but see Hamburger, Lilienfeld, & Hogben, 1996, for data suggesting that histrionic personality disorder and ASPD may represent sex-differentiated manifestations of psychopathic personality traits). The sex differentiation hypothesis is not inconsistent with the previously noted covariation between ASPD and SD (e.g., Lilienfeld et al., 1986), because biological sex may serve primarily to shift slightly the phenotypic manifestation of the diathesis underlying ASPD and SD. As a consequence, males possessing high levels of this diathesis may be somewhat more likely to exhibit ASPD features than SD features, and vice-versa for females possessing high levels of this diathesis.

The mechanisms underlying the association between ASPD and SD remain largely or entirely unknown. Lilienfeld (1992) reviewed several theoretical models that have been invoked to account for the ASPD–SD link. Among these explanations are the behavioral disinhibition model and the negative emotionality (NE) model. According to the behavioral disinhibition model, both primary psychopaths (including some individuals with ASPD) and individuals with SD are characterized by an underactive behavioral inhibition system (BIS) (Gorenstein & Newman, 1980). Gray (1982) proposed that the BIS, a psychobiological system coursing through the septum, hippocampus, and orbitofrontal cortex, mediates sensitivity to conditioned signals of punishment or "frustrative nonreward" (Amsel, 1958) and is responsible for individual differences in anticipatory anxiety. Moreover, according

to Gray, the BIS exerts tonic inhibitory control over the behavioral activation system (BAS), a psychobiological system that is sensitive to signals of reward and which mediates individual differences in impulsivity. Low levels of BIS activity are therefore posited to result in functionally overactive levels of BAS activity (see also Fowles, 1980).

The behavioral disinhibition model posits that both primary psychopathy and SD are marked by chronically low BIS strength, resulting in low levels of anticipatory anxiety, poor impulse control, and a failure to learn from punishment. Indeed, there is evidence that primary psychopaths (Hare, 1965; Lykken, 1957) and perhaps some individuals with SD (Franks, 1956) exhibit weak electrodermal classical conditioning to aversive stimuli. In addition, primary psychopaths exhibit diminished fear-potentiated startle as measured by the eyeblink reflex (Patrick, Bradley, & Lang, 1993). In support of the behavioral disinhibition model, Frick et al. (1995) found that a measure of behavioral disinhibition, the Sensation Seeking Scale (Zuckerman, 1983), was positively correlated with measures of antisocial behavior and somatization in a sample of mothers of clinically referred children. Nevertheless, the behavioral disinhibition model has been called into question by data indicating that patients with SD and similar syndromes (e.g., conversion disorder) tend to exhibit high resting levels of skin conductance, a high rate of spontaneous skin conductance fluctuations, and slow habituation to repetitive sounds (Cloninger, 1978). This pattern of findings is opposite to that typically reported among primary psychopaths (Hare, 1978).

The NE model posits that individuals with high levels of antisocial behavior tend to experience elevated levels of NE as a consequence of their impulsive and reckless actions (Tellegen & Waller, 1994; Watson & Clark, 1984). NE is a pervasive distress or emotional maladjustment dimension that reflects a propensity to experience negative emotions of all kinds, including anxiety, guilt, irritability, and mistrust. Fowles (1987) similarly argued that antisocial behavior leads to high NE in some individuals, because physically and socially dangerous behaviors often create life difficulties and distress for the individual (see also Tellegen, 1978/1982). Indeed, there is evidence that antisocial behavior is associated with elevated levels of NE in both adults (Lilienfeld, 1994) and children (Russo & Beidel, 1993). Unlike the behavioral disinhibition model, the NE model can readily explain the high levels of autonomic activity reported among SD patients (Cloninger, 1978), because elevated autonomic activity is associated with anxiety (Katkin & Hastrup, 1982).

NE, in turn, is associated with a propensity to experience multiple somatic symptoms (Watson & Pennebaker, 1989), presumably because (1) NE renders individuals more susceptible to actual health problems, (2) NE leads individuals to interpret ambiguous bodily sensations as indicative of somatic symptoms, or both. Watson and Pennebaker (1989), who favored the latter explanation, described NE as a "general trait of somatopsychic distress" (p. 248). Thus, the NE model posits the following causal sequence: antisocial and risk taking behavior → elevated NE → elevated levels of somatic symptoms (see Lilienfeld, 1992). This sequence has not, however, been subjected to a direct empirical test.

According to the NE model, NE mediates the association between antisocial behaviors and SD (see Baron & Kenny, 1986, for a discussion of mediation). As Lilienfeld (1992) observed, the NE model leads to the predictions that "SD would be positively correlated with ASPD, but negligibly (or perhaps even negatively) correlated with primary psychopathy" and that "the association between ASPD and SD would no longer hold after NE levels are controlled for statistically" (p. 654). Because measures of secondary psychopathy tend to be more highly correlated with ASPD than measures of primary psychopathy do (Harpur et al., 1989), the NE model also predicts that SD should be positively correlated with secondary psychopathy. Moreover, from this perspective, secondary rather than primary psychopathy is likely to be the diathesis underlying ASPD and SD.

Wilson, Frick, and Clements (1999) recently reported evidence consistent with the NE model in a nonclinical sample. In an investigation of 199 undergraduates, they found that a composite index of primary psychopathy derived from three self-report psychopathy measures was significantly and negatively correlated with a measure of somatization, whereas a composite index of secondary psychopathy—which largely assessed a history of antisocial behaviors—was significantly and positively correlated with a measure of somatization. Contrary to the sex differentiation hypothesis (Lilienfeld, 1992), however, Wilson et al. found no significant differences between males and females in the correlations between secondary psychopathy characteristics and somatization symptoms. Wilson et al. suggested that their results were consistent with the hypothesis that the relation between secondary psychopathy characteristics and somatization is mediated by NE. Because they did not include a measure of NE, however, this hypothesis could not be tested directly.

The present study represents an attempt to test the sex differentiation hypothesis of ASPD and SD, as well as the NE model of their association. The principal hypotheses tested in the present investigation were fourfold. These hypotheses were tested using three different self-report measures of psychopathy (see also Wilson et al., 1999),

providing us with a built-in test of the generalizability of our findings across different assessment instruments.

(1) The relation between psychopathic traits and somatization symptoms should be attributable entirely to secondary psychopathy features (see Lykken, 1995, for a similar prediction). In contrast, the association between primary psychopathic traits and somatization symptoms should be negligible or perhaps even negative. This pattern of findings would replicate the results of Wilson et al. (1999).

(2) The relation between secondary psychopathy and somatization symptoms should be moderated by biological sex. Specifically, because the sex differentiation hypothesis predicts that the traits of psychopathy (specifically, secondary psychopathy) should be more highly correlated with somatization symptoms in females than in males, this hypothesis predicts a significant moderator (i.e., interaction) effect (Stone, 1988). As a consequence, moderated multiple regression analyses (Jaccard, Turrisi, & Wan, 1990) should reveal the relation between secondary psychopathy features and somatization symptoms to be significantly more pronounced in magnitude among females than males (cf. Wilson et al., 1999).

(3) The relation between secondary psychopathy features and somatization symptoms should be fully mediated (Baron & Kenny, 1986) by NE. In other words, controlling statistically for the effects of NE should render the relation between secondary psychopathy features and somatization symptoms nonsignificant, as predicted by the NE model.

(4) An alternative hypothesis, which although not favored by the present authors was examined here, is that features of both primary (but not secondary) psychopathy and somatization should be negatively correlated with a measure of behavioral inhibition. This finding would furnish support for the behavioral disinhibition model and would be consistent with the possibility that behavioral disinhibition is a shared diathesis underlying primary psychopathy, SD, and perhaps other syndromes (Frick et al., 1995; Gorenstein & Newman, 1980).

These hypotheses were examined in an analogue sample consisting of undergraduates. Undergraduate samples are characterized by several potential disadvantages. For example, such samples may be marked by a restricted range of scores on measures of somatization and both primary and secondary psychopathy compared with clinical samples. In addition, if the diathesis underlying either primary or secondary psychopathy were taxonic, rather than dimensional (see Harris, Rice, & Quinsey, 1994; Lilienfeld, 1998), there could be insufficient admixture between taxon and nontaxon groups to produce sizeable correlations among either psychopathy measures themselves or between psychopathy measures and other indices. This problem would be especially likely to arise if the base rate of the putative taxon were very low (Meehl & Golden, 1982). Nevertheless, undergraduate samples have the advantage of being relatively free of severe Axis I disorders (e.g., mood and anxiety disorders), which have sometimes been found to distort the reporting of longstanding personality traits (e.g., Loranger et al., 1991). In addition, there is preliminary but promising evidence that the relations among self-report psychopathy measures in students (e.g., Lilienfeld et al., 1998) and prisoners (e.g., Poythress, Edens, & Lilienfeld, 1998) are comparable, suggesting that correlational data from the former samples may prove generalizable to the latter. Finally, certain response styles, such as malingering or random responding, may be less problematic in undergraduate samples than in certain clinical (e.g., prison) samples. Nevertheless, to exclude the possibility that our findings were attributable to systematic error in the form of a generalized social undesirability response style, we reconducted our analyses after controlling for scores on a widely used social desirability measure.

METHOD

Participants

Participants were 150 undergraduates enrolled in a large private university in the southeastern United States.[4] Of these, 33 were male and 117 female. This sex ratio, although quite imbalanced, roughly parallels the sex distribution in this university's introductory psychology participant pool. Because of missing data on some measures (measures were excluded from the analyses on a pairwise basis if any items were omitted), the total *N*s for some analyses are slightly less than 150 (see Results section).

[4]To exclude the possibility that participants' somatic complaints were attributable to physical illnesses, we asked all participants (on a demographic sheet preceding the questionnaire packet) to list any current serious medical diseases (e.g., cancer, acquired immune deficiency syndrome). Because no participants reported serious physical illnesses, however, all were retained in the analyses reported here.

Participants' mean age was 18.79 ($SD = .95$). Of the total participants, 100 were Caucasian, 23 were Asian, 14 were African American, and 5 were Hispanic; the remaining 7 participants described themselves as being of either mixed descent or another ethnicity. Information concerning ethnicity was omitted by one female participant. All participants received partial course credit in return for their participation.

Procedure

Participants completed a packet of self-report measures in group (6–24 students) settings. At least one research assistant was on hand at all times to clarify potentially ambiguous items and answer other questions. The order of questionnaire administration was counterbalanced, with half of the participants randomly assigned to receive the psychopathy measures first and the other measures second, and vice versa. Because the order of administration had no appreciable effects on the findings reported here, both orders were combined in the analyses.

Measures

Psychopathy and Antisocial Behavior

Self-Report Psychopathy Scale (SRP) (Hare, 1985). This 75-item self-report measure was designed to assess the principal features of psychopathy. The SRP was constructed empirically by identifying items that discriminated between low and high psychopathy groups as assessed by the Psychopathy Checklist (PCL), a semistructured interview used in conjunction with file information, which is the most extensively construct validated measure of psychopathy (Hare, 1991). The SRP was further refined using item analytic techniques. In the same manner as the PCL and its revision, the PCL-R, the SRP consists of two factors. Because the items on the two SRP factors were selected only if they correlated highly with one of the two PCL factors, these two factors comprise only a small portion of the total SRP item pool. SRP Factor I (SRP1; nine items) assesses callousness, grandiosity, and other core personality features of primary psychopathy (Cleckley, 1941/1988), whereas SRP Factor II (SRP2; 13 items) assesses a chronic history of antisocial behaviors. These two SRP factors were used in this study to operationalize primary and secondary psychopathy features, respectively (see also Wilson et al., 1999).

The SRP correlates moderately to highly with total scores on the PCL-R (Forth, Brown, Hart, & Hare, 1996; see Zagon & Jackson, 1994, for further validity data). The internal consistency (Cronbach's alpha) of the SRP total score in this sample was .84; the internal consistencies of SRP1 and SRP2 were .47 and .77, respectively.

Levenson Primary and Secondary Psychopathy Scales. These scales were developed by Levenson, Kiehl, and Fitzpatrick (1995) to assess self-reported features of primary and secondary psychopathy in noninstitutionalized samples. The primary (16 items) and secondary (10 items) psychopathy scales were rationally constructed by modeling items after PCL-R Factors I and II, respectively. In a sample of 487 university students, Levenson et al. (1995) reported that the Levenson primary psychopathy scale (Levenson1) was negligibly correlated with a measure of trait anxiety, whereas the Levenson secondary psychopathy scale (Levenson2) was moderately correlated with a measure of trait anxiety. In addition, both scales were positively correlated with self-reported antisocial behaviors and with the boredom susceptibility and disinhibition subscales of Zuckerman's Sensation Seeking Scale (Zuckerman, 1989). In this sample, the internal consistencies of the Levenson1 and Levenson2 scales were .84 and .62, respectively.

Psychopathic Personality Inventory (PPI). The PPI, which was developed by Lilienfeld (1990) to assess the central characteristics of psychopathy in nonclinical samples, consists of items on a 1–4 Likert-type scale. In addition to a total score, which is interpretable as an index of global psychopathy, the PPI consists of eight-factor analytically developed subscales assessing various components of psychopathy. The eight subscales of the PPI, along with one sample item from each subscale and the direction of item keying, are:

> *Machiavellian Egocentricity* ["I often tell people only the part of the truth they want to hear" (True)]
>
> *Social Potency* ["I am a good conversationalist" (True)]
>
> *Coldheartedness* ["I often become deeply attached to people I like" (False)]
>
> *Fearlessness* ["Making a parachute jump would really frighten me" (False)]
>
> *Impulsive Nonconformity* ["I've always considered myself to be something of a rebel" (True)]
>
> *Blame Externalization* ["Some people seem to have gone out of their way to make life difficult for me" (True)]
>
> *Carefree Nonplanfulness* ["I weigh the pros and cons of major decisions carefully before making them" (False)]
>
> *Stress Immunity* ["I can remain calm in situations that would make many other people panic" (True)]

The PPI total score has been found to correlate moderately to highly with self-report, structured interview, and peer-rated measures of antisocial behavior and psychopathy, including the PCL-R (Lilienfeld,

1996, Lilienfeld & Andrews, 1996; Poythress, Edens, & Lilienfeld, 1998) as well as with self-report and peer-rated measures of personality traits relevant to primary psychopathy (e.g., physical risk taking, absence of social anxiety; Lilienfeld & Andrews, 1996). The PPI total score has also been reported to exhibit incremental validity above and beyond a number of commonly used measures of psychopathy and antisocial behavior in the prediction of peer-rated and interviewer-rated Cleckley psychopathy (Lilienfeld & Andrews, 1996).

In this study, the short form of the PPI, which consists of 56 items, was administered. The PPI short form was constructed by selecting the seven items that loaded the most highly on each of the eight factors in a factor analysis of 610 undergraduates (see Lilienfeld, 1990). The PPI short form has been found to correlate $r = .90$ or above with the full form in several undergraduate samples. The internal consistency of the PPI total score in this sample was .85; the internal consistencies of the eight PPI subscales ranged from .64 to .85.

Following the principal components analyses of Wilson et al. (1999, see also Lilienfeld, 1990, for factor analyses yielding similar results), the eight PPI subscales were assigned to primary and secondary psychopathy scales. Specifically, Social Potency, Coldheartedness, Fearlessness, Impulsive Nonconformity, and Stress Immunity were combined into a PPI primary psychopathy score (PPI1), whereas Machiavellian Egocentricity, Blame Externalization, and Carefree Nonplanfulness were combined into a PPI secondary psychopathy score (PPI2). The PPI1 ($\alpha = .86$) and PPI2 ($\alpha = .82$) scales were used in the analyses reported here.

Somatization

Symptom Checklist 90-Revised (SCL-90-R) Somatization Scale. This 12-item questionnaire consists of items assessing a wide variety of somatic complaints. It has been reported to correlate moderately to highly ($r = .62$) with the experimental Somatic Depression scale of the SCL-90-R (Buckelew, Burk, Brownlee-Duffeck, & Frank, 1988). In addition, the SCL-90-R Somatization Scale has been found to distinguish women who reported illness resulting from the odors of common chemicals from normal women (Bell, Schwartz, Hardin, Baldwin, & Kline, 1998). The internal consistency of the SCL-90 Somatization Scale in this sample was .81.

Wahler Physical Symptom Inventory. This 42-item self-report measure was developed by Wahler (1973) to assess the intensity, frequency, and duration of various somatic symptoms. It has been reported to discriminate individuals reporting physical complaints, individuals undergoing physical rehabilitation, and individuals seeking financial recompense for their somatic symptoms from normals. In addition, scores on the Wahler Physical Symptom Inventory have been found to correlate highly with scores on the MMPI Hypochondriasis scale in males and females (Wahler, 1973). Finally, scores on the Wahler Physical Symptom Inventory correlate positively with both the subjective intensity of minor life stressors and self-reported stress reactivity (Waters, Rubman, & Hurry, 1993). The internal consistency of the Wahler Physical Symptoms Inventory in this sample was. 90.

Negative Emotionality (NE)

Negative Emotionality, 30 item scale (NEM-30). The NEM-30 was developed by Waller, Tellegen, McDonald, and Lykken (1996) by selecting items from the Multidimensional Personality Questionnaire (Tellegen, 1978/1982) that appeared to be especially good markers of NE. It was refined using standard item analytic techniques (e.g., corrected item–total correlations) and item response theory methods, yielding a total of 30 items. Preliminary validational analyses in a sample of twins revealed moderate and significant negative correlations with several California Psychological Inventory scales, including Social Presence, Well-Being, and Intellectual Efficiency (Waller et al., 1996). The internal consistency of the NEM-30 in this sample was .83.

Positive Affect, Negative Affect Scales (PANAS). The PANAS scales were developed by Watson, Clark, and Tellegen (1988, see also Watson & Clark, 1992) to provide markers of positive and negative affect, which appear to be the two principal dimensions of emotional temperament (Tellegen, 1985). In the analyses reported here, only the PANAS Negative Affect scale (hereafter referred to as the PANAS-Negative), which consists of 10 items, was examined. The PANAS-Negative has been found to correlate moderately to highly with several self-report indices of NE and emotional distress, including the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) and Hopkins Symptom Checklist (Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974; Watson et al., 1988). In the present study, the "general" (trait) form of the PANAS-Negative, which asks participants how they generally feel (see Watson et al., 1988, p. 1070), was administered. In conjunction with the NEM-30, the PANAS-Negative was used in this investigation as a measure of NE. The internal consistency of the PANAS-Negative Scale in this sample was .86.

Behavioral Inhibition and Activation

Behavioral Inhibition System (BIS) and Behavioral Activation System (BAS) Scales (Carver & White, 1994). These scales were rationally constructed by Carver and White (1994) to operationalize Gray's constructs (Gray, 1982) of the BIS and BAS, respectively. Although the BAS scale consists of three subscales, these subscales load on the same higher-order factor and appear to assess overlapping aspects of a latent dimension of behavioral activation (Carver & White, 1994). Hence, only total BAS scale scores were examined here.

BIS scale scores correlate positively with the amount of anxiety experienced in anticipation of punishment, whereas BAS scale scores correlate positively with the amount of happiness experienced in anticipation of reward (Carver & White, 1994). In addition, BIS scale scores correlate positively with indices of neuroticism and anxiety, whereas BAS scale scores correlate positively with indices of extraversion (Heubeck, Wilkinson, & Cologon, 1998; Jorm et al., 1999). Moreover, the BIS scale correlates negatively, although only moderately, with the Eysenck Personality Questionnaire Psychoticism Scale (Jorm et al., 1999), which measures a disposition toward impulsivity, aggressiveness, and callousness (Eysenck, Eysenck, & Barratt, 1985). The internal consistencies of the BIS (7 items) and BAS (13 items) scales in this sample were .73 and .80, respectively.

Social Desirability

Marlowe-Crowne Social Desirability Scale (MCSD). This 33-item questionnaire (Crowne & Marlowe, 1964), which consists of items assessing both desirable but rare behaviors (e.g., thoroughly investigating the qualifications of all candidates before voting) and undesirable but common behaviors (e.g., feeling jealous of others' good fortune), is a commonly used measure of social desirability. Correlational and factor analytic research suggest that it primarily assesses the component of social desirability termed as "other deception" or impression management, i.e., a largely conscious effort to tailor one's test responses to create a positive impression on others (Paulhus, 1991). Scores on the MCSD predict a susceptibility to social influence and evaluation (Crowne & Marlowe, 1964) and increase when participants' test responses are not anonymous (Paulhus, 1984). The internal consistency of the MCSD in this sample was .73.

RESULTS

The means and standard deviations for the primary variables, in both the total sample and males and females, are displayed in Table I. With the exception of PPI2, males scored significantly higher than did females

Table I. Means and Standard Deviations for Primary Measures in Total Sample, Males, and Females

Measures	Total sample	Males	Females	*t*	*df*	*d*
SRP Total score	191.65 (24.14)	212.42 (17.54)	185.58 (22.42)	6.33***	144	1.26
SRP1	24.17 (4.33)	26.61 (3.86)	23.47 (4.22)	3.83***	147	.76
SRP2	31.87 (7.81)	36.15 (6.40)	30.65 (7.77)	3.72***	147	.73
Levenson1	28.72 (7.18)	31.75 (8.09)	27.86 (6.70)	2.77**	143	.55
Levenson2	20.34 (4.27)	21.91 (3.80)	19.90 (4.30)	2.43*	146	.48
PPI Total score	118.99 (16.32)	130.94 (13.95)	115.41 (15.30)	5.14***	137	1.03
PPI1	78.08 (13.44)	88.38 (9.92)	75.14 (12.88)	5.37***	142	1.08
PPI2	40.71 (7.97)	42.27 (9.01)	40.25 (7.62)	1.29	143	.25
SCL-90-R Somatization	0.68 (.54)	.63 (.55)	.70 (.54)	−.65	148	.13
Wahler	1.16 (.55)	1.12 (.46)	1.17 (.57)	−.48	148	.09
NEM-30	10.71 (5.46)	9.88 (5.86)	10.95 (5.35)	−.98	144	.20
PANAS-Negative	21.81 (6.50)	20.21 (6.18)	22.27 (6.54)	−1.61	145	.32
BIS Scale	21.57 (3.47)	19.09 (2.70)	22.27 (3.36)	−5.00***	148	.98
BAS Scale	41.04 (4.93)	40.39 (6.45)	41.22 (4.43)	−.851	148	.17
MCSD	14.06 (4.79)	14.67 (3.38)	13.89 (5.02)	.823	148	.16

Note. *N* for total sample ranges from 139 to 150; *n* for males ranges from 32 to 33; *n* for females ranges from 112 to 117. SRP: Self-Report Psychopathy Scale; PPI: Psychopathic Personality Inventory; SCL-90-R: Symptom Checklist-90-Revised; NEM: Negative Emotionality; PANAS: Positive Affect, Negative Affect Scales; BIS: Behavioral Inhibition System; BAS: Behavioral Activation System; MCSD: Marlowe-Crowne Social Desirability Inventory. *t*: t test for significance of the mean difference between males and females; *d*: Cohen's *d*, i.e., effect size of the mean difference between males and females.

Table II. Correlations Among Primary and Secondary Psychopathy Measures

	SRP1	SPR2	Levenson1	Levenson2	PPI1	PPI2
SRP1		.14	.23**	−.13	.60***	−.03
SRP2			.48***	.51***	.47***	.58***
Levenson1				.39***	.23**	.52***
Levenson2					−.01	.71***
PPI1						.09

Note. *N*s range from 139 to 148. SRP: Self-Report Psychopathy Scale; PPI: Psychopathic Personality Inventory.
** $p < .01$.
*** $p < .001$, two tailed.

on all primary, secondary, and total psychopathymeasures. The effect sizes (Cohen's *d*s) for most of these significant differences were in the medium-to-large range (Cohen, 1988). In contrast, there were no significant differences on either the NE or somatization measures, although females scored slightly higher than did males on all of these indices. Females scored significantly higher than did males on the BIS scale, although sex differences on BAS scale were nonsignificant.

The correlations among the primary and secondary psychopathy scales are shown in Table II. These scales generally exhibited a clear pattern of convergent and discriminant validity, with primary psychopathy scales correlating more highly with other primary psychopathy scales than with secondary psychopathy scales, and vice versa. The lone exception to this pattern was Levenson1, which correlated more highly with SRP2 and PPI2 than with SRP1 and PPI1. In both cases, tests of the significance of the difference between dependent correlations (Cohen & Cohen, 1975) revealed these correlations to be significantly different from each other. For SRP2 versus SRP1, $t(141) = 2.51$ ($p < .05$), and for PPI2 versus PPI1, $t = 2.93$ ($p < .01$).

The NEM-30 and the PANAS-Negative were correlated at $r = .68$ ($p < .001$). In addition, the SCL-90 Somatization Scale and the Wahler Physical Symptoms Inventory were correlated at $r = .73$ ($p < .001$). Because of the high correlations between both the two NEM scales and the two somatization measures, these pairs of measures were combined into composite indices of NE and somatization, respectively, by standardizing them (into *z* scores) and summing them. These two composite indices, which will hereafter be referred to as the Composite NE Index and the Composite Somatization Index, respectively, were used in the remaining analyses reported here.[5]

[5]Analyses using separate measures of somatization and NE yielded results very similar to those reported here, and are available from the first author on request.

Table III presents the correlations between the psychopathy measures and the Composite Somatization Index in both the total sample and in males and females. As can be seen in this Table, the correlations between the three secondary psychopathy measures and the Composite Somatization Index were positive and significant, whereas the correlations between the three primary psychopathy and the Composite Somatization Index were either negative or negligible. In the case of SRP1, this correlation was negative and significant; in the case of Levenson1 and PPI1, this correlation was nonsignificant. Tests of the significance of the difference between dependent correlations revealed that the correlations between the three secondary psychopathy measures and the Composite Somatization Index were significantly higher (i.e., more positive in magnitude) than the corresponding correlations between the three primary psychopathy measures and the Composite Somatization Index. For the correlations between SRP2 and SRP1 and the Composite Somatization Index, *t*

Table III. Correlations Between Total, Primary, and Secondary Psychopathy Measures and Composite Somatization Index

	r		
	Total sample	Males	Females
Psychopathy measure			
SRP Total score	−.01	−.29	.09
SRP1	−.28***	−.30	−.27**
SRP2	.25**	−.06	.35***
Levenson1	.13	−.08	.20*
Levenson2	.31***	−.01	.40***
PPI Total score	.09	.03	.03
PPI1	−.15	−.15	−.11
PPI2	.42***	.23	.50***

Note. *N*s for total sample range from 145 to 150; *n*s for males range from 32 to 33, and *n*s for females range from 112 to 117. SRP: Self-Report Psychopathy Scale; PPI: Psychopathic Personality Inventory.
* $p < .05$.
** $p < .01$.
*** $p < .001$, two tailed.

(145) = 5.33, $p < .001$; for the correlations between Levenson2 and Levenson1 and the Composite Somatization Index, t (140) = 2.06, $p < .05$; and for the correlations between PPI2 and PPI1 and the Composite Somatization Index, t (136) = 5.35, $p < .001$.

As can also be seen in Table III, the correlations between the three secondary psychopathy measures and the Composite Somatization Index were in all cases higher in magnitude among females than among males, as predicted by the sex differentiation hypothesis. The correlations between the three secondary psychopathy measures and the Composite Somatization Index were low or negligible in magnitude among males.

Moderated multiple regression analyses (Jaccard et al., 1990) were performed to test the hypothesis that the relations between secondary psychopathy measures and somatization would be significantly stronger in magnitude among females than among males. In each analysis, the Composite Somatization Index was used as the dependent variable. Biological sex and each secondary psychopathy measure were forcibly entered into the first step, followed by the product of biological sex and each secondary psychopathy measure in the second step. The partialled product term represents the interaction (Stone, 1988). For SRP2, the addition of the product term at the second step was marginally significant: $R^2_{\text{change}} = .02$; F_{change} (1, 145) = 3.53, $p < .07$. For Levenson2, the addition of the product term at the second step was significant: $R^2_{\text{change}} = .02$; F_{change} (1, 144) = 4.06, $p < .05$. For PPI2, the addition of the product term at the second step was again significant: $R^2_{\text{change}} = .03$; F_{change} (1, 141) = 4.46, $p < .05$.[6]

The next set of analyses examined the hypothesis that the relation between secondary psychopathy and somatization would be mediated by NE. In these analyses, we followed the three-step procedure recommended by Baron and Kenny (1986) for testing mediation. According to Baron and Kenny, to conclude that full mediation is present, three conditions must be satisfied. First, the independent variable, in this case secondary psychopathy, must be significantly and positively correlated with the proposed mediator, in this case, NE. Inspection of zero-order correlations revealed that this correlation was fulfilled for the SRP2, Levenson2, and PPI2, whose correlations with the Composite NE Index were .19 ($p < .05$), .48 ($p < .001$), and .55 ($p < .001$), respectively. The second condition that must be satisfied in the Baron and Kenny (1986) framework is that the proposed mediator, in this case, NE, must be significantly and positively correlated with the dependent measure, in this case, somatization. This condition was satisfied, as the Composite NE Index was positively and significantly correlated with the Composite Somatization Index ($r = .62$, $p < .001$).

The third and final condition to establish full mediation according to Baron and Kenny (1986) is that controlling statistically for the proposed mediator should reduce the correlation between the independent and dependent variables to nonsignificance. In two of three cases, this condition was met. Specifically, controlling for the NE Composite Measure reduced the correlations between the Levenson2 and PPI2 on the one hand, and the Composite Somatization Index on the other, from .31 ($p < .001$), and .42 ($p < .001$), respectively (i.e., the zero-order correlations shown in Table III), to .02 (*ns*), .and .13 (*ns*), respectively. Controlling for the NE Composite Measure reduced the correlation between the SRP2 and the Composite Somatization Index from .25 ($p < .01$) to .17, although the latter partial correlation remained significant ($p < .05$).

To test the predictions of the behavioral disinhibition model, we examined the correlations between the BIS and BAS scales and measures of total, primary, and secondary psychopathy as well as the Composite Somatization Index. These correlations are displayed in Table IV. As predicted by the behavioral disinhibition model, BIS scores were significantly and negatively correlated with all primary

Table IV. Correlations Between BIS and BAS Scales and Measures of Psychopathy and Somatization

Psychopathy or somatization measure	BIS scale	BAS scale
SRP Total score	−.58***	.20*
SRP1	−.54***	.06
SRP2	−.29***	.27***
Levenson1	−.17*	.21*
Levenson2	−.09	.05
PPI Total score	−.47***	.22*
PPI1	−.56***	.18*
PPI2	−.02	.17*
Composite Somatization Index	.14	.04

Note. *N*s range from 139 to 150. BIS: Behavioral Inhibition System; BAS: Behavioral Activation System; SRP: Self-Report Psychopathy Scale; PPI: = Psychopathic Personality Inventory.
* $p < .05$.
** $p < .01$.
*** $p < .001$, two tailed.

[6] We also compared the correlations between the secondary psychopathy measures and the Composite Somatization Index in males and females, using tests of the significance of the difference between independent correlations (Shavelson, 1996). In the case of the SRP2 and Levenson2, the male and female correlations were significantly different (zs = 1.97 and 2.09, respectively, both *ps* < .05); in the case of the PPI2, the difference between male and female correlations was not significant ($z = 1.51$, *ns*).

psychopathy measures. The correlations between the BIS scale and secondary psychopathy measures were close to zero in the case of Levenson2 and the PPI2, although the correlation between the BIS scale and SRP-2 was negative and significant. BIS scores were not significantly associated with scores on the Composite Somatization Index. The correlations between the BAS scale and the primary and secondary psychopathy measures were less clear cut. The BAS scale was significantly correlated with Levenson1 and not Levenson2, but was significantly correlated with SRP2 and not SRP1. In contrast, the BAS scale was significantly correlated with both PPI1 and PPI2, although both correlations were low in magnitude. The BAS scale was negligibly correlated with the Composite Somatization Index.

Finally, to exclude the possibility that the positive correlations among secondary psychopathy measures, the Composite NE Index, and the Composite Somatization Index were attributable to the influence of a generalized social undesirability response style, we recomputed all of the previously reported correlations after controlling for scores on the MCSD. The correlations among measures remained very similar, although several decreased slightly in magnitude. In all cases, however, the secondary psychopathy scales remained more highly (positively) related than the three primary psychopathy scales to the Composite Somatization Index. In addition, controlling for MCSD scores produced no substantive changes in the relations between the BIS and BAS scales, on the one hand, and the primary and secondary psychopathy measures and Composite Somatization Index, on the other.[7]

DISCUSSION

The covariation between ASPD and SD has long been one of the most puzzling comorbidities in descriptive psychopathology (Cloninger, 1978; Lilienfeld, 1992). Although some authors (e.g., Carothers, 1975) have contended that SD is associated with the personality traits comprising primary psychopathy, others (e.g., Guze et al., 1971) have emphasized the relation between SD and ASPD. Our findings provide support for the contention that somatic complaints, at least in a nonclinical sample, are associated largely or entirely with secondary psychopathic features and negligibly and perhaps even negatively associated with primary psychopathic features. These results, which hold across three different self-report measures of psychopathy and replicate those of Wilson et al. (1999), are consistent with the possibility that secondary psychopathy is a diathesis underlying SD and ASPD. Because the secondary psychopathy scales on the measures examined here, particularly the SRP2 and Levenson2, tend to be associated with a chronic propensity toward antisocial behavior, both our findings and those of Wilson et al. (1999) suggest that somatic symptoms are associated almost entirely with the antisocial lifestyle component of psychopathy, rather than with the core affective traits (e.g., guiltlessness, lack of empathy) traditionally believed to comprise primary psychopathy (Cleckley, 1941/1988). Interestingly, total scores on the SRP and PPI (the Levenson scales are not summated to produce a total score) were negligibly and nonsignificantly correlated with somatization, suggesting that the use of total psychopathy scores may obscure the relations between primary and secondary psychopathy and at least some important psychopathological variables.

Our findings offered no support for the hypothesis that somatic symptoms are associated with low levels of behavioral inhibition (cf. Frick et al., 1995; Lilienfeld, 1992, pp. 650–653). Although replication of these negative findings among samples exhibiting greater variance in somatic symptoms is necessary, it should be noted that the nonsignificant correlation between BIS scale scores and somatization was in the opposite direction from that predicted by the behavioral disinhibition model. One potential explanation for this finding is that the BIS scale is primarily a measure of NE or trait anxiety rather than of behavioral inhibition (i.e., Constraint; see Tellegen, 1978/1982). Indeed, subsidiary analyses not presented here revealed that the BIS scale was moderately and significantly correlated ($r = .45$, $p < .001$) with the Composite NE Index. Nevertheless, additional analyses revealed that the PPI Fearlessness subscale, which like most fearlessness measures appears to be a (reversed) psychometric marker of Gray's behavioral inhibition system (see Lilienfeld & Andrews, 1996), was negligibly and nonsignificantly correlated ($r = .04$) with the Composite Somatization Index.

By using moderated multiple regression techniques, we found that the relations between secondary psychopathy measures and somatic complaints were significantly (in the case of the SRP, marginally significantly) stronger in magnitude among females than among males. As noted earlier, these moderator effects are consistent with the sex differentiation hypothesis (Lilienfeld, 1992), which predicts more pronounced correlations between secondary psychopathy measures and somatization in females than in males. These findings run counter to those of Wilson et al. (1999), who reported that the relation between secondary psychopathy and somatic complaints did not differ

[7] The results of these analyses are available from the first author on request.

in males and females. Again, the fact that our results held across three different measures of psychopathy lends some degree of credence to their verisimilitude. The reasons for the disrepancy between our findings and those of Wilson et al. (1999) are unclear, however, and will be important to ascertain in future research.

Our significant findings using both moderated multiple regression analyses and tests of the significance of the difference between independent correlations (see Footnote 6) are especially impressive given the relatively small sample of males, which would otherwise militate against detecting statistically significant moderator effects. Moreover, because there were no marked variance differences in any of these measures between sexes, our findings cannot be attributed to differential range restriction in males and females. Nevertheless, the increments in variance accounted for by the interaction term were in each case quite small in magnitude (2–3% of the variance), suggesting that biological sex may exert only a relatively modest influence on the phenotypic expression of secondary psychopathic traits. In future research, it will be important to test the complementary prediction that the relation between secondary psychopathic traits and ASPD features is stronger in magnitude among males than among females.

Contrary to expectation, there were no significant sex differences in mean levels of somatization. Moreover, the effect sizes for these differences were small (.09–.13; see Table I), rendering low statistical power a somewhat unlikely explanation for these results. These findings are difficult to reconcile with the sex differentiation hypothesis, which posits that ASPD and SD are sex-typed alternative manifestations of a shared underlying diathesis (e.g., Cloninger, 1978). Nevertheless, it is conceivable that somatization symptoms are heterogeneous in etiology, with only certain of these symptoms bearing a causal relation to secondary psychopathy. Using admixture analysis, Cloninger, Sigvardsson, von Knorring, and Bohman (1984) reported that somatic complaints in a large sample of female adoptees appeared to be underpinned by two discrete taxa. "High frequency somatization" (which resembles the *DSM-IV* category of hypochondriasis) was characterized by intense complaints in one or two bodily areas, whereas "diversiform somatization" (which resembles the *DSM-IV* category of SD) was characterized by less intense complaints in a wide variety of bodily areas. Because we did not explicitly assess the diversity of participants' bodily complaints, the distinction between these two putative subtypes of somatization could not be examined.

Our results provide provisional support for the hypothesis that the relation between secondary psychopathy and somatic symptoms is mediated by NE. In the case of the Levenson2 and PPI2, controlling for NE reduced the correlations with somatic symptoms to nonsignificance. In contrast, the correlation between the SRP2 and somatic symptoms remained significant after controlling for NE, although this correlation decreased in magnitude. These findings are broadly consistent with the NE model of the association between ASPD and SD (Lilienfeld, 1994), although the significant residual correlation between the SRP2 and somatic symptoms suggests that additional unmeasured personality or response style variables may account for this covariation. Because some authors have posited that secondary psychopathy is etiologically heterogeneous (Lilienfeld, 1994), it will be important in future investigations to examine the possibility that the relation between secondary psychopathy and somatic complaints is attributable to only one or two putative secondary psychopathy subtypes (e.g., see Lykken's 1995, discussion of the "hysterical psychopath" subtype).

Our results should be interpreted in light of several caveats. First, our findings will need to be replicated and extended to samples, such as psychiatric patients and prisoners, which exhibit higher levels of both psychopathic traits and somatic symptoms. Such studies would permit us to determine whether our findings can be applied to more severely affected samples and generalized from dimensional measures of psychopathy/ASPD and SD to categorical diagnoses of these syndromes.

Second, because all of our results were derived from self-report indices, at least some of our correlational findings may be partly attributable to method covariance. It should be noted, however, that because the items on secondary psychopathy measures and somatization indices differ substantially from one another phenotypically, our positive findings cannot readily be attributed to essentially tautological content overlap across questionnaires (cf., Nicholls, Licht, & Pearl, 1982). In addition, both the PPI and the SRP have been found to exhibit moderate to high levels of criterion-related validity with semistructured interview diagnoses of psychopathy (e.g., Forth et al., 1996; Lilienfeld et al., 1998; Poythress et al., 1998). Although the use of self-report questionnaires to assess psychopathy has been criticized on the grounds that many psychopathic individuals are prone to dissimulation (Hare, 1985), analyses controlling for scores on a measure that assesses primarily the impression management component of social desirability (viz., the MCSD; see Paulhus, 1991) produced no substantive changes in our correlational findings. Nevertheless, it will be necessary to constructively replicate (Lykken, 1968) our findings using alternative modes of assessing psychopathy, such as semistructured interviews (e.g., the PCL-R; Hare,

1991) and observer (e.g., peer) reports (e.g., Lilienfeld & Andrews, 1996; Reise & Oliver, 1994). Because the version of the PCL-R designed for use in nonclinical samples, viz., the Psychopathy Checklist: Screening Version (Hart, Cox, & Hare, 1995), may not possess a clear two-factor structure in college students (Forth et al., 1996), alternative interview measures may need to be developed to assess the two psychopathy factors in nonclinical samples.

Third, although the primary and secondary psychopathy indices generally exhibited a relatively clear-cut pattern of convergent and discriminant validity with one another, Levenson1 significantly correlated more highly with secondary psychopathy measures than with other primary psychopathy measures. Wilson et al. (1999, see p. 228) similarly found that Levenson1 was associated primarily with the antisocial lifestyle component of psychopathy rather than with its core affective and personality deficits. This anomalous finding renders unclear the implications of the finding that Levenson2 correlated more highly than did Levenson1 with the Composite Somatization Index, because Levenson1 appears to be nearly as strong a marker of secondary psychopathy as Levenson2 (see Table II). Studies examining our hypotheses with additional measures of the two psychopathy factors, particularly the PCL-R (Hare, 1991), should assist in ascertaining the robustness of our findings regarding the differential correlates of these two factors.

Fourth, the internal consistency of the SRP1 (alpha = .47) was low and may have attenuated correlations between this scale and other measures. Nevertheless, because the correlation between SRP1 and the Composite Somatization Index was negative, it is unlikely that this low reliability accounts for the differential relation between the two SRP scales and somatization. After correcting both SRP scales for attenuation due to unreliability, the correlations between the Composite Somatization Index and SRP1 and SRP2 became more divergent in magnitude (−.40 and .29, respectively).

If our findings concerning the differential manifestation of secondary psychopathy features in males and females can be replicated, they leave unanswered the question of what variables might account for these sex differences in phenotypic expression. Among these potential variables are biological influences, such as testosterone and serotonin, both of which exhibit sex differences and may be correlated (testosterone positively and serotonin negatively) with risk for overt aggression (Dabbs & Morris, 1990; Steiner, LePage, & Dunn, 1997; but see Sapolsky, 1997, for findings calling into question the causal role of testosterone in human aggression). In addition, sociocultural influences, such as differential sex role socialization (Cloninger, 1978; Lilienfeld, 1992), may be partly responsible for influencing the extent to which aggression is expressed directly versus indirectly (Eagly & Steffen, 1985). Future investigations of the relations between secondary psychopathic traits and somatization should incorporate measures of both biological indices and gender roles (e.g., the Bem Sex Role Inventory; Bem, 1974) so that the underlying causes of the potentially different manifestations of these traits in males and females can be identified.

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