Several authors have proposed that antisocial personality disorder (ASPD) and histrionic personality disorder (HPD) are sex-typed manifestations of the same underlying predisposition, namely psychopathy. In a sample of 180 undergraduates (90 males, 90 females), we tested three hypotheses: (1) psychopathy underlies both ASPD and HPD traits; (2) the relation between psychopathy and both ASPD and HPD traits is moderated by biological gender, with psychopathic males tending to exhibit an antisocial pattern and psychopathic females a histrionic pattern; and (3) the relation between psychopathy and both ASPD and HPD traits is moderated by gender roles, with psychopathic individuals possessing stereotypically "masculine" features tending to exhibit an antisocial pattern and psychopathic individuals possessing stereotypically "feminine" features tending to exhibit a histrionic pattern. The latter hypothesis was tested both across and within gender. Structural equation modeling applied to self-report measures of psychopathy, gender roles, and ASPD and HPD traits provided support for the first two hypotheses but not the third. Implications of these findings for the differential expression of psychopathy in males and females, as well as future directions for research, are discussed.

Considerable controversy surrounds the classification and diagnosis of personality disorders (Grove & Tellegen, 1991; Perry, 1992; Zimmerman, 1994). Two problems in this domain are especially vexing. First, there is extensive covariation or "comorbidity" (but see Lilienfeld, Waldman, & Israel, 1994, for a critique of this term) among many personality disorders (Grove & Tellegen, 1991). This covariation has led some authors (e.g.,
Carson, 1993) to call into question the assumption that these disorders represent independent entities. Second, a number of personality disorders exhibit sex differences in prevalence (Ford & Widiger, 1989; Kass, Spitzer, & Williams, 1983). Some authors (e.g., Kaplan, 1983; Tavris, 1992) have conjectured that these sex differences are attributable to gender biases in the criteria for these disorders, whereas others (e.g., Warner, 1978) have suggested that these criteria are sometimes applied by diagnosticians in a biased fashion. Alternatively, these differences may reflect genuine disparities in the etiological factors (e.g., sex hormones) relevant to certain personality disorders (Widiger & Spitzer, 1991).

Antisocial personality disorder (ASPD) and histrionic personality disorder (HPD) illustrate both of these phenomena. Lilienfeld, Van Valkenburg, Larntz, and Akiskal (1986) reported that ASPD and HPD co-occur within individuals considerably more often than expected by chance. In addition, Luisada, Peele, and Pittard (1974) reported a high rate of antisocial behaviors among histrionic males.

ASPD and HPD also appear to exhibit sex differences in prevalence. ASPD has consistently been found to be more common among males than females; population prevalence data indicate a rate of approximately 3% among males and 1% among females (American Psychiatric Association—APA, 1994). HPD, in contrast, has generally been found to be more common among females than males (Ford & Widiger, 1989; Kass et al., 1983; cf. Reich, 1987), particularly in clinical samples (APA, 1994). Some dispute remains, however, concerning the sex distribution of HPD in the population at large; Nesdadt et al. (1990), for example, reported a population prevalence rate for HPD of approximately 2% among both males and females.

How can the comorbidity and gender differences in these two personality disorders be explained? Similarities between ASPD and HPD are consistent with the hypothesis that they are manifestations of the same underlying syndrome, namely psychopathy. Cleckley (1941/1982) conceptualized psychopathy as consisting of such characteristics as superficial charm, lack of anxiety, guiltlessness, egocentricity, dishonesty, and sexual promiscuity. Cloninger (1978) and others (e.g., Lilienfeld, 1992) have conjectured that psychopathy is often manifested differently in males than in females, with psychopathic males generally exhibiting an antisocial pattern and psychopathic females a histrionic pattern. Thus, biological gender may tend to channel the expression of psychopathy into either ASPD or HPD. In a related vein, Chodoff (1982) suggested that ASPD and HPD represent caricatures of gender role stereotypes. Pursuing this reasoning, adherence to traditional gender roles may, like biological gender, tend to channel the expression of psychopathy into either ASPD or HPD.

ASPD AND HPD: LINKS TO PSYCHOPATHY

ASPD and HPD, although differing from psychopathy in important ways, bear a number of intriguing similarities to this syndrome. ASPD differs from psychopathy in that the former is defined by a history of antisocial, criminal, or otherwise irresponsible behaviors, whereas the latter is defined primarily
in terms of personality traits (Lilienfeld, 1994). ASPD has generally been found to overlap moderately, but by no means completely, with Cleckley’s (1941/1982) conception of psychopathy. In a study of forensic patients, Hart and Hare (1989) reported that psychopaths had high rates of ASPD, but that individuals with ASPD had relatively low rates of psychopathy. Moreover, Harpur, Hakstian, and Hare (1989) found that a factor representing the core personality features of psychopathy correlated approximately $r = .5$ with a factor representing the antisocial behaviors typical of ASPD, and that these two factors exhibited markedly different personality, intellectual, and socioeconomic correlates.

HPD is defined by such features as self-centeredness, self-dramatization, attention seeking, emotional lability, reactivity to minor events, shallow affect, and sexual provocativeness (APA, 1994). HPD has been reported to be significantly correlated with an interview-based measure of psychopathy (Hart & Hare, 1989). Although several of the characteristics of HPD (e.g., emotional lability) are not traditionally considered central to psychopathy, several others, such as shallow emotions and seductiveness, are commonly observed among psychopaths (Cleckley, 1941/1982). Moreover, although individuals with HPD are often initially perceived as charming and personable, people who know them well often see them as manipulative and dishonest (Chodoff & Lyons, 1958). Thus, HPD and psychopathy appear to share a number of important features.

**GENDER ROLES**

Several authors (e.g., Bem, 1981) have hypothesized that adherence to gender roles influences the development of both adaptive and maladaptive personality traits. A gender role can be defined as the combination of attitudes, behaviors, and emotions that are more commonly associated with one gender than the other. Several researchers have developed instruments to assess the degree of adherence to gender roles, most of which involve the classification of individuals as traditionally “masculine” or “feminine” (Bem, 1974).

Other researchers have focused on identifying individuals who adhere to extreme gender role stereotypes. Mosher and his colleagues (e.g., Mosher & Sirkin, 1984), for example, proposed the existence of a construct reflecting identification with extreme “masculine” gender roles (i.e., “hypermasculinity”). Hypermasculine men, who exhibit a “macho” behavioral style, enjoy power, danger, and violence, and perceive women as dominion (Mosher, 1991). Murmen and Byrne (1991) conceptualized “hyperfemininity” as the female analogue of hypermasculinity, and argued that hyperfemininity is a manipulative power strategy used by women to exert control in relationships.

**HYPOTHESES**

Because we have utilized a nonclinical sample (viz., undergraduates), we operationalize the constructs of ASPD and HPD in terms of dimensions (i.e.,
number of symptoms) rather than categories (i.e., diagnoses). Drawing on the research relating psychopathy, gender, and gender roles, on the one hand, to ASPD and to HPD on the other, we propose the following hypotheses.

Hypothesis 1. Individuals with high levels of psychopathy will be more likely to exhibit features of both ASPD and HPD than individuals without high levels of psychopathy. This relation will be demonstrated by significant zero-order correlations between indicators of psychopathy and indicators of ASPD and HPD traits. In addition, by means of path analysis we expect to find satisfactory model fit and significant path coefficients from the construct of psychopathy to those of ASPD and HPD traits.

Hypothesis 2. The relation between the construct of psychopathy and those of both ASPD and HPD traits will be moderated by biological gender. Specifically, given high levels of psychopathy, males will be more likely to exhibit features of ASPD than females, and females will be more likely to exhibit features of HPD than males. Thus, the addition of biological gender as a moderator will significantly increase model fit compared with the model specified in Hypothesis 1.

Hypothesis 3. The relation between the construct of psychopathy and those of both ASPD and HPD traits will be moderated by gender role adherence. Specifically, given high levels of psychopathy, traditionally "masculine" individuals will be more likely to exhibit features of ASPD than either traditionally "feminine" or "nonmasculine" individuals. In addition, traditionally "feminine" individuals will be more likely to exhibit features of HPD than either traditionally "masculine" or "nonfeminine" individuals. Thus, the addition of gender role adherence as a moderator will significantly increase model fit compared with the model specified in Hypothesis 1. Hypothesis 3 will be tested both across and within biological gender.

METHOD

SUBJECTS

Data were collected from 96 men and 90 women, who were recruited from the introductory psychology subject pool in partial fulfillment of their course research requirement. Six males were excluded from the analyses based on extreme scores on the Deviant Responding (DR) and Variable Response Inconsistency (VRIN) validity scales of the Psychopathic Personality Inventory (PPI—see Measures). The final sample thus consisted of 90 men and 90 women, whose ages ranged from 17 to 49 (M = 19.3). Sixty-four percent of the subjects were Caucasian, 11% African-American, 11% Asian, and the remaining 12% either Latino or other.

MEASURES. Subjects completed a battery of self-report measures, which are described below.

PPI. The PPI (Lilienfeld, 1990) is designed to measure the primary personality traits of psychopathy and consists of items measured on a 4 point Likert-type scale. Unlike most self-report measures of psychopathy, such as the MMPI Psychopathic deviate scale (Harpur, Hakstian, & Hare, 1989), the PPI appears to assess many of the personality features deemed by Cleckley (1941/1982) to be relevant to psychopathy. In constructing the PPI, items assessing overt antisocial behaviors were excluded in order to provide a relatively "pure" measure of the personality traits of
psychopathy. In addition to a total score, which is interpretable as a global index of psychopathy, the PPI consists of eight subscales assessing such traits as fearlessness, guiltlessness, and egocentricity (see Lilienfeld, 1990). The PPI also contains two validity scales. The first, DR, consists of statements with extremely low endorsement frequencies (e.g., “I occasionally forget my name”) and is designed primarily to detect careless responding or malingering. The second, VRIN, identifies item pairs whose content is homogeneous within each pair but varies greatly across pairs (Tellegen, 1988). High scores on VRIN indicate that subjects are not responding consistently to statements with similar content, and typically reflect careless responding.

Lilienfeld (1990) reported an internal consistency of .91 for the PPI total score among undergraduates. Twenty-six day test-retest reliability for the PPI total score was excellent (r = .95). The PPI total score has been found to correlate moderately to highly (rs ranged from .61 to .91) with Hare’s (1985) Self-Report Psychopathy Scale, a measure designed to assess the principal personality features of psychopathy, as well as with interviewer and peer ratings of Cleckley psychopathy (rs = .60 and .45, respectively). In addition, the PPI total score demonstrates good discriminant validity from measures of depression, neuroticism, psychosis proneness, and social desirability response styles (Lilienfeld & Andrews, 1994).

Personality Diagnostic Questionnaire—Revised (PDQ-R) ASPD and HPD Scales. These measures (ASPPDQ and HPPDQ—Hyler & Rieder, 1987) were rationally constructed to assess the DSM-III-R (APA, 1987) criteria for ASPD and HPD, respectively. These scales have shown moderate levels of concordance with structured interview diagnoses of ASPD and HPD (Hyler, Skodol, Kellman, Oldham, & Rosnick, 1990).

DSM-III MMPI Personality Disorder Scales for ASPD and HPD (Morey, Waugh, & Blashfield, 1985). These scales (ASPMMPi and HPMMPI) were developed from the MMPI item pool by means of a combined rational and empirical strategy. Their items were targeted to assess the DSM-III (APA, 1980) criteria for ASPD and HPD. ASPMMPI and HPMMPI have shown a promising pattern of convergent and discriminant validity with the standard MMPI clinical scales (Morey et al., 1985) and have been reported to discriminate clinical diagnoses of ASPD and HPD, respectively, from those of other personality disorders (Morey, Blashfield, Webb, & Jewell, 1988). In this study, the non-overlapping versions of these scales were used to provide more content-pure measures of ASPD and HPD traits.

Bem Sex Role Inventory (BSRI). The BSRI (Bem, 1974) is designed to assess traditional masculine and feminine gender roles, and consists of masculine and feminine trait descriptors selected on the basis of sex-typed social desirability. The BSRI yields a fourfold classification: masculine, feminine, androgynous (high on both masculinity and femininity), and undifferentiated (low on both masculinity and femininity). Analyses of the BSRI (Bem, 1974) indicate that the masculine and feminine subscales are essentially orthogonal. Bem (1981, 1984) reported that masculine and feminine sex-typed individuals were better at recalling sex-typed words than non-sex-typed individuals. Moreover, in a test of recall, sex-typed individuals were more likely to erroneously attribute “masculine” statements to males and “feminine” statements to females (Bem, 1984). The BSRI is used to test Hypothesis 3 across both biological genders (the sample sizes are not large enough to permit a meaningful fourfold classification within each gender).

Hypermasculinity Inventory (HMI). The HMI (Mosher & Sirkin, 1984), which is designed for male respondents only, is a forced-choice questionnaire designed to assess characteristics of the “macho” personality constellation (Mosher, 1991). The HMI has been found to correlate positively with self-reported frequency of aggressive behaviors while intoxicated and various forms of sexual aggression and coercion (Mosher & Sirkin, 1984). In addition, scores on the HMI are correlated with lower
levels of disgust and other negative emotions during imagined rape scenes (Mosher & Anderson, 1986). The HMI, like the HFS (see below), is used to test Hypothesis 3 within each biological gender.

Hyperfemininity Scale (HFS). The HFS (Murnen & Byrne, 1991), which is designed for female respondents only, is a forced-choice scale patterned after the HMI. Murnen and Byrne (1991) found that HFS scores were correlated with less negative perceptions of, and more numerous experiences with, sexual coercion. Furthermore, compared with other subjects, hyperfeminine subjects report lowered levels of negative reactions to depictions of coercion (Murnen, Perot, & Byrne, 1988).

MCSD. The MCSD (Crowne & Marlowe, 1960) consists of statements designed to measure the extent to which subjects respond to items in a socially conventional manner and was administered to examine the extent to which social desirability influenced the relations among measures. The MCSD is highly correlated with other self-report measures of social desirability (Paulhus, 1984). In addition, the MCSD has been reported to attenuate correlations between a measure of gender roles (viz., the HFS) and other variables (Murnen & Byrne, 1991).

Analyses. Although we report both zero-order and partial (corrected for social desirability) correlations among measures, our principal hypotheses were tested by path analysis using Linear Structural Relations (LISREL—Jöreskog & Sorbom, 1993; see also Loehlin, 1992). LISREL is a program based on structural equations that enables researchers to construct models from correlational data in a manner similar to multiple regression analysis (MRA). The major differences between LISREL and MRA are that LISREL allows researchers to obtain estimates of measurement error (i.e., unreliability) or to construct a theoretically driven error theory. Neither of these options is available in MRA, in which overall perfect model fit (i.e., no measurement error) is assumed. Additionally, LISREL allows multiple dependent variables to be addressed in the same analysis, allowing for a more powerful analysis at the same alpha level. Thus, findings that are not statistically significant in MRA will often be statistically significant in LISREL.

LISREL may also be used to estimate fit of models involving moderator variables, as specified in Hypotheses 2 and 3. When the moderators are continuous, an interaction term is created in a manner analogous to moderated MRA (Stone, 1988). When the variables are categorical, as is the case with biological gender and gender role classification according to the BSRI, HMI and HFS, this approach is no longer feasible. Instead, LISREL offers a multiple group solution using categorical groups.

Four indices of goodness-of-fit will be reported for the LISREL analyses: the $\chi^2$ test, the goodness of fit index (GFI), the comparative fit index (CFI), and the adjusted goodness of fit index (AGFI), the last three of which are adjusted for the number of parameters included in the model. These three indices range from 0 to 1, with a generally accepted level for good fit being .90 or higher. Because LISREL does not provide the AGFI for the multiple group solution, the three other goodness of fit indices are reported for the tests of Hypotheses 2 and 3.

RESULTS

Descriptive Statistics and Gender Differences. Means and standard deviations for the major variables for the total sample, males, and females are

1. It is possible to score all of these three variables dimensionally, as seen in Table 1, although this practice is not typical in the gender role literature (e.g., Bem, 1984). The lack of multiple indicators and the need to specify some paths in the lambda x matrices (to define a metric for the underlying construct) made it impossible to construct the interaction terms congruent with the limitations outlined in Jöreskog and Sorbom (1993).
shown in Table 1. None of the differences between males and females is significant and, as is evident by inspection of Cohen's d (an index of effect size), all are relatively small in magnitude. Nonetheless, all of the differences on the personality disorder measures are in the predicted direction, with males exhibiting higher scores on the the ASPD measures and females exhibiting higher scores on the HPD measures.

**Correlational Analyses.** Prior to examining the zero-order correlations among measures, we tested for equivalence of the covariance matrices between males and females. Although Box's M was significant [(F(21, 116533.7) = 2.47, p < .001)], pairwise t tests for differences between the correlations in each sample indicated that only 1 of 15 comparisons was significant at p < .05. Following Bonferroni correction for the number of comparisons, this difference was no longer significant. Thus, we combined males and females for the correlational analyses.

Table 2 displays the zero-order and partial correlations among variables. Consistent with Hypothesis 1, significant positive correlations were found

<table>
<thead>
<tr>
<th>Variable</th>
<th>Overall</th>
<th>Women</th>
<th>Men</th>
<th>t</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td>PPI</td>
<td>378.83</td>
<td>369.57</td>
<td>388.09</td>
<td>.47</td>
<td>.07</td>
</tr>
<tr>
<td>ASPMMP</td>
<td>11.36</td>
<td>10.22</td>
<td>12.50</td>
<td>.70</td>
<td>.10</td>
</tr>
<tr>
<td>HPMMP</td>
<td>14.66</td>
<td>15.08</td>
<td>14.23</td>
<td>.24</td>
<td>.04</td>
</tr>
<tr>
<td>ASPPDQ</td>
<td>3.13</td>
<td>2.08</td>
<td>4.19</td>
<td>.68</td>
<td>.10</td>
</tr>
<tr>
<td>HPPDQ</td>
<td>3.09</td>
<td>3.40</td>
<td>2.78</td>
<td>.30</td>
<td>.04</td>
</tr>
<tr>
<td>BSRIMAS</td>
<td>101.59</td>
<td>98.67</td>
<td>104.52</td>
<td>.46</td>
<td>.07</td>
</tr>
<tr>
<td>BSRIFEM</td>
<td>94.92</td>
<td>99.61</td>
<td>90.23</td>
<td>.91</td>
<td>.14</td>
</tr>
<tr>
<td>HMI</td>
<td>11.76</td>
<td>—</td>
<td>11.76</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>HFS</td>
<td>7.31</td>
<td>7.31</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>MCSD</td>
<td>14.94</td>
<td>16.01</td>
<td>13.87</td>
<td>.39</td>
<td>.06</td>
</tr>
</tbody>
</table>

*Note. Overall N = 180, n (men) = 90, n (women) = 90. Standard deviations are given in parentheses. PPI = Psychopathic Personality Inventory; ASPMMP = the Antisocial Personality Disorder measure of the DSM-III MMPI Personality Disorder Scales; HPMMP = the Histrionic Personality Disorder measure of the DSM-III MMPI Personality Disorder Scales; ASPPDQ = the Antisocial Personality Disorder measure of the Personality Diagnostic Questionnaire—Revised; HPPDQ = the Histrionic Personality Disorder measure of the Personality Diagnostic Questionnaire—Revised: BSRIMAS = the sum of the masculine items of the Bem Sex Role Inventory; BSRIFEM = the sum of the feminine items of the Bem Sex Role Inventory; HMI = Hypermasculinity Inventory; HFS = Hyperfemininity Scale; MCSD = Marlowe–Crowne Social Desirability Scale.*
### TABLE 2. Zero-Order Correlations and First-Order Partial Correlations, Controlling for Scores on the MCSD, Among Variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. PPI</td>
<td>—</td>
<td>.40***</td>
<td>.50***</td>
<td>.55***</td>
<td>.11</td>
<td>.51***</td>
<td>.42***</td>
<td>.51***</td>
<td>.12</td>
</tr>
<tr>
<td>2. ASPMMPI</td>
<td>.48***</td>
<td>—</td>
<td>.26***</td>
<td>.49***</td>
<td>.25***</td>
<td>-.18*</td>
<td>.08</td>
<td>.41***</td>
<td>.16</td>
</tr>
<tr>
<td>3. HPMMPI</td>
<td>.47***</td>
<td>.21**</td>
<td>—</td>
<td>.12</td>
<td>.17*</td>
<td>.31***</td>
<td>-.07</td>
<td>.31**</td>
<td>.17</td>
</tr>
<tr>
<td>4. ASPPDQ</td>
<td>.59***</td>
<td>.56**</td>
<td>.10</td>
<td>—</td>
<td>.02</td>
<td>.34***</td>
<td>-.24**</td>
<td>.44***</td>
<td>.11</td>
</tr>
<tr>
<td>5. HPPDQ</td>
<td>.16*</td>
<td>.16*</td>
<td>.16*</td>
<td>.08</td>
<td>—</td>
<td>-.10</td>
<td>.09</td>
<td>.04</td>
<td>.30**</td>
</tr>
<tr>
<td>6. BSRIMAS</td>
<td>.50***</td>
<td>.24***</td>
<td>.31***</td>
<td>.33***</td>
<td>-.09</td>
<td>—</td>
<td>-.16**</td>
<td>.27**</td>
<td>-.11</td>
</tr>
<tr>
<td>7. BSRIFEM</td>
<td>-.47***</td>
<td>-.29***</td>
<td>-.05</td>
<td>-.30***</td>
<td>.03</td>
<td>-.17*</td>
<td>—</td>
<td>-.28**</td>
<td>.10</td>
</tr>
<tr>
<td>8. HMIb</td>
<td>.53***</td>
<td>.43**</td>
<td>.30**</td>
<td>.46***</td>
<td>.07</td>
<td>.27**</td>
<td>-.31**</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>9. HFS</td>
<td>.19</td>
<td>.27**</td>
<td>.15</td>
<td>.19</td>
<td>.33***</td>
<td>-.10</td>
<td>.01</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>10. MCSD</td>
<td>-.30**</td>
<td>-.46***</td>
<td>.04</td>
<td>.32***</td>
<td>-.19*</td>
<td>-.04</td>
<td>.29***</td>
<td>-.15</td>
<td>-.29**</td>
</tr>
</tbody>
</table>

**Note.** Overall N = 180, n (men) = 90, n (women) = 90. Zero-order correlations appear below the diagonal and partial correlations, controlling for scores on the MCSD, above the diagonal. PPI = Psychopathic Personality Inventory; ASPMMPI = the Antisocial Personality Disorder measure of the DSM-III MMPI Personality Disorder Scales; HPMMPI = the Histrionic Personality Disorder measure of the DSM-III MMPI Personality Disorder Scales; ASPPDQ = the Antisocial Personality Disorder measure of the Personality Diagnostic Questionnaire-Revised; HPPDQ = the Histrionic Personality Disorder measure of the Personality Diagnostic Questionnaire-Revised; BSRIMAS = the sum of the masculine items of the Bem Sex Role Inventory; BSRIFEM = the sum of the feminine items of the Bem Sex Role Inventory; HMI = Hypermasculinity Inventory; HFS = Hyperfemininity Scale; MCSD = Marlowe-Cronne Social Desirability Scale.

**A two-tailed test of significance was used for both the zero-order correlations and the first-order partial correlations.**

**Because men respond to the HMI and women respond to the HFS, significance values for correlations involving these scales are based on n as of 90 and correlations among HMI and HFS scores cannot be calculated.**

*p < .05; **p < .01; ***p < .001.
between the PPI and the indicators of ASPD and HPD traits. The correlation
between scores on the HPPDQ and scores on the PPI, however, was relatively
low. A significant, but moderate, positive correlation was found between the
two measures of ASPD traits (ASPPDQ and ASPMMPI). Although the two
measures of HPD traits (HPPDQ and HPMMPI) were significantly positively
correlated, this correlation was relatively weak.

Scores on the masculine items from the BSRI (BSRIMAS) were signifi-
cantly positively correlated with scores on the PPI, ASPPDQ, and ASPMMPI.
Scores on the feminine items from the BSRI (BSRI FEM) were negatively
correlated with scores on the PPI, ASPPDQ, and APSMMPI. Further support
for the link between masculinity and ASPD traits is evidenced by significant
positive correlations among scores on the HMI, ASPMMPI, and ASPPDQ.

BSRIFEM scores were not significantly positively correlated with scores
on the HPMMPI or HPPDQ, nor were scores on the BSRIMAS significantly
negatively correlated with scores on the HPMMPI or HPPDQ. Instead, a
significant positive correlation was observed between BSRIMAS and
HPMMPI. Scores on the HFS were significantly positively correlated with
the HPPDQ and ASPMMPI.

We next examined the partial correlations among variables while control-
ling for MCSD scores. Examination of this matrix reveals that social
desirability as assessed by the MCSD had minimal effects on the relations
among the variables.

Path Analyses. As noted earlier, our primary hypotheses were tested
by path analysis using LISREL (specifically, LISREL8). We generated
several path analytic models beginning with a model in which psycho-
pathy was posited to underlie both ASPD and HPD traits, as predicted
by Hypothesis 1.

The model for Hypothesis 1 was evaluated using the sample covariance
matrix as input. All latent constructs were assessed via multiple observed
variables; those chosen to define the metric of the latent construct (i.e., the
corresponding path in the lambda x matrix was constrained to equal 1.0)
are underlined in Figure 1. The PPI was split into two measures (PPITOT1
and PPITOT2) through a partition of odd and even items, with DR scale
items excluded. There are no prima facie reasons for choosing either
PPITOT1 or PPITOT2 to define the metric of the psychopathy construct; for
the analyses reported here, we arbitrarily selected PPITOT1.2 We chose
the ASPPDQ to define the metric of the construct of ASPD traits and the HPPDQ
for HPD traits because these subscales (unlike the ASPMMPI and HPMMPI)
directly assess the DSM criteria for their respective disorders.

Goodness-of-fit for the Hypothesis 1 model showed a significant departure
from expected fit ($\chi^2(7) = 19.57, p < .05$). Although this statistic indicates
that the observed findings differ significantly from those generated by the
hypothesized model, the $\chi^2$ statistic is substantially influenced by sample
size (Hays, 1988). The other fit indices were satisfactory: GFI = .96, CFI =
.97, and AGFI = .90. The standardized path coefficients between psycho-
pathy, on the one hand, and ASPD and HPD, on the other, were .74 and

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2. Using PPITOT2 to define the metric of psychopathy yielded models with identical summary
statistics and path coefficients.
PSYCHOPATHY AND GENDER

FIGURE 1. Structural equation model for Hypothesis 2: The relation between psychopathy and ASPD and HPD moderated by biological gender (variables chosen to define the metrics of their respective constructs are underlined).

.73 (both significant at $p < .05$), indicating that psychopathy is a strong predictor of both ASPD and HPD traits, as predicted by Hypothesis 1.

In addition, we tested a second version of the Hypothesis 1 model using partial correlations [i.e., controlling for MCSD] as the input matrix. LISREL8 generated similar estimates of model fit for this version ($\chi^2(7) = 21.60$, $p < .05$; GFI = .95; CFI = .96; AGFI = .89) and similar path estimates to ASPD traits (.71) and HPD traits (.63). Both path estimates were significant at $p < .05$, and were not significantly different from those generated from zero-order correlations.

To test for the effects of biological gender as a moderator, it was necessary to show that each group within the categorical moderator variable fits equal form (i.e., the same model holds for each group). Thus, we compared the separate analyses of the male and female samples to each other while specifying the basic model form for each group, resulting in $\chi^2(14) = 31.21$, $p < .01$; GFI = .95; CFI = .96. Again, although the $\chi^2$ statistic is significant at conventional levels, the other fit indices are satisfactory and suggest that the female and male samples fit the same model form.

The basic model for the moderator variable analysis using biological gender is shown in Figure 1 (the remaining moderator variable analyses followed the same model form, with BSRI, HMI, and HFS used as moderators instead of biological gender). Using this model, we tested for equality of the paths from psychopathy to ASPD and HPD traits across males and

3. Substituting either BSRIMAS or BSRI/FEM for psychopathy in the Hypothesis 1 model yielded roughly similar goodness-of-fit indices to those reported here, although the path coefficients to ASPD and HPD traits (.37 and .23, respectively, for BSRIMAS; and -.40 and -.05, respectively, for BSRI/FEM) were substantially lower.

4. Additionally, model estimates using partial correlations (controlling for the MCSD) were obtained for the moderator models for biological gender, BSRI, HMI, and HFS. In each case, only minor and nonsignificant differences in model fit from the models using zero-order correlations were found.
females. To do this, the same multiple group solution as above was run twice, once with the path from psychopathy to ASPD traits constrained to equality (across males and females), and once with the path from psychopathy to HPD traits constrained to equality. The summary statistics can be compared with those above representing equal model form without constrained path estimates, with the difference in the $\chi^2$ (also distributed as $\chi^2$) providing an estimate of the significance of the difference. The psychopathy-ASPD constrained model had $\chi^2(15) = 40.83, p < .001$; GFI = .93; CFI = .94. Comparative fit between the two nested models was estimated by $\chi^2(1) = 9.62, p < .01$. The psychopathy-HPD constrained model had $\chi^2(15) = 40.03, p < .001$; GFI = .93; CFI = .94. Comparative fit was estimated by $\chi^2(1) = 8.82, p < .01$. These estimates indicate that there are significant differences in the path coefficients between psychopathy and ASPD and HPD traits between sexes, although these differences are relatively small in magnitude. Separate inspection of the zero-order correlation matrices for males and females (available from the first author upon request) confirms that, as predicted by Hypothesis 2, the relation between psychopathy and ASPD traits is stronger among males, whereas the relation between psychopathy and HPD traits is stronger among females.

The same procedure was followed to test for the effects of gender role classification according to the BSRI, the HMI, and the HFS as moderators. The four categories of the BSRI (masculine, feminine, androgynous, undifferentiated) had equal model form ($\chi^2(28) = 55.50, p < .001$; GFI = .94; CFI = .93). Contrary to Hypothesis 3, the BSRI did not moderate the relation between psychopathy and ASPD and HPD traits for either psychopathy-ASPD ($\chi^2(3) = 2.76, \text{NS}$) or psychopathy-HPD ($\chi^2(3) = 0.64, \text{NS}$). The two categories of the HMI also had equal model form ($\chi^2(14) = 28.92, p < .001$; CFI = .92; GFI = .91). The HMI did not moderate the relation between psychopathy and ASPD and HPD traits for either psychopathy-ASPD ($\chi^2(1) = 0.06, \text{ns}$) or for psychopathy-HPD ($\chi^2(1) = 0.30, \text{ns}$). The two categories of the HFS again fit into equal model form ($\chi^2(14) = 19.76, \text{ns}; GFI = .92; CFI = .97$), but did not moderate the relation between psychopathy and ASPD traits ($\chi^2(1) = 1.69, \text{ns}$) or HPD traits ($\chi^2(1) = 0.65, \text{ns}$). Thus, Hypothesis 3 received no support from these analyses.

DISCUSSION

In this study, we hypothesized that the expression of psychopathy would be differentially channeled by both biological gender and gender roles into ASPD and HPD features. Although these hypotheses are not new (e.g., Cloninger, 1978; Lilienfeld, 1992; see also Nuckolls, 1992), they have not been subjected to direct empirical tests. Consistent with Hypothesis 1, we found that psychopathy was a strong predictor of both ASPD and HPD traits. In addition, we found that, consistent with Hypothesis 2, the relation between psychopathy and both ASPD and HPD traits was moderated by biological gender; psychopathic males tended to exhibit ASPD characteristics, whereas psychopathic females tended to exhibit HPD characteristics.
If this finding can be replicated, it would be consistent with the possibility that two superficially different syndromes—ASPD and HPD—are sex-differentiated expressions of a shared diathesis, namely psychopathy. Research efforts should then be focused on the identification of biological variables such as sex hormones, and personality traits such as dependency (Cloninger, 1987) and aggression, which may be responsible for the differential manifestation of psychopathic characteristics in males and females. In addition, our results may hold important implications for the classification of personality disorders. If ASPD and HPD are expressions of the same latent predisposition, this finding might imply that our current taxonomic schemes for personality disorders have focused on superficial differences at the expense of underlying commonalities (Vaillant, 1984).

Nevertheless, several limitations of our results must be underscored. First, our use of an analogue sample (undergraduates) renders the interpretation of some of our findings problematic, because the features of ASPD and HPD may have not had sufficient variance to provide an adequate test of our hypotheses. Consequently, our failure to detect significant moderator effects for the two measures of gender roles, as predicted by Hypothesis 3, should be interpreted with caution. Alternatively, our failure to corroborate Hypothesis 3 could indicate that the expression of psychopathy is influenced primarily by variables (e.g., sex hormones) that exhibit large differences between genders but small differences within each gender.

Moreover, because the base rates of the DSM-III-R diagnosis of ASPD (as assessed by the PDQ-R) were relatively low (14.4% among males and 3.3% among females), we were unable to use ASPD as a categorical variable for the LISREL analyses. Because the question of whether personality disorders differ from personality traits in degree or in kind remains unresolved (Widiger, 1991), our results should be replicated in a sample permitting ASPD and HPD to be operationalized as diagnoses as well as dimensions.

Second, our exclusive reliance on self-report measures raises the possibility that at least some of our findings are attributable to method covariance. Although we find it implausible that method covariance could explain the significant interaction effect for biological gender, our conclusions would be strengthened by the incorporation of information from alternative sources, such as direct interview, file data, and observer ratings. In addition, self-report and structured interview measures of personality disorders tend to correlate weakly or at best moderately (Perry, 1992), highlighting the need for replication of our findings using interview data. In future research, we intend to use the Psychopathy Checklist—Revised (Hare, 1990), a well-validated measure incorporating both interview and file information, to operationalize psychopathy and to supplement self-report

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5. The base rates of DSM-III-R defined HPD, in contrast, were considerably higher (27.8% among males and 42.2% among females). These extremely high figures are consistent with findings indicating that the PDQ-R has a very high false positive rate and is best viewed as a screening, rather than diagnostic, measure for personality disorders (Hyler, Skodol, Olham, Kellman, & Doldge, 1992). Because of the relatively low base rates of DSM-III-R defined ASPD, however, analyses operationalizing HPD at the diagnostic level were not conducted, as comparisons between DSM-III-R defined ASPD and HPD could not be performed. Because the DSM-III MMPI Personality Disorder scales do not map directly onto DSM-III criteria, these scales cannot be used to derive diagnoses of either ASPD or HPD.
indices of ASPD and HPD traits with interview measures and ratings from significant others.

Third, the construct validity of BSRI has been called into question by some authors. Helmreich, Spence, and Wilhelm (1981), for example, have argued that BSRIMAS and BSRIFEM are better construed as indices of "instrumentality" and "expressivity," respectively, than as omnibus measures of gender roles. This interpretation of BSRIMAS may help to explain a puzzling finding in the correlational analyses: the significant positive correlation between BSRIMAS and HPMMPI (although it should be noted that the correlation between BSRIMAS and HPPDQ was close to zero). Given that histrionic individuals are often noted for their manipulative and controlling qualities (Chodoff & Lyons, 1958), this correlation is consistent with the interpretation of BSRIMAS as a measure of instrumentality.

Fourth and finally, the principal focus of our study was on convergent, rather than discriminant, validity. In other words, we included only measures of personality disorders (viz., ASPD and HPD) that we expected to be closely associated with psychopathy, but did not include measures of personality disorders, such as schizotypal personality disorder, that we expected to be largely or entirely unrelated to psychopathy. In addition, we did not include measures of borderline personality disorder (BPD), which covaries extensively with both ASPD and HPD (Pope, Jonas, Hudson, Cohen, & Gunderson, 1983). Because BPD appears to be highly etiologically heterogeneous (Akiskal et al., 1985), however, it may not represent an ideal disorder for examining the differential expression of psychopathy. Nonetheless, the incorporation of measures of additional personality disorders would provide a more stringent test of our hypotheses by permitting us to ascertain the specificity of our findings to ASPD and HPD.

REFERENCES


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