Sex differences in psychopathy and antisocial personality disorder
A review and integration

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Abstract

Although the correlates and causes of psychopathy and antisocial personality disorder (ASPD) have been the subject of extensive investigation, researchers in this area have until recently focused almost exclusively on males. As a consequence, relatively little is known about psychopathy and ASPD in females. In this paper, we review the empirical literature on sex differences in the base rates, mean symptom levels, correlates, and factor structure of psychopathy and ASPD. In addition, we discuss the potential sex-differentiated phenotypic expressions of psychopathy and ASPD (e.g., somatization disorder [SD]) as well as sex differences in the developmental trajectories of these conditions. There is suggestive evidence that these conditions may be differentially expressed across biological sex, although further investigation of this issue is warranted. We conclude with recommendations for future research in this area, including suggestions for embedding the study of sex differences in psychopathy and ASPD within a construct validational framework.

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1. Introduction

The classification and etiology of psychopathy and antisocial personality disorder (ASPD) are among the foremost challenges to researchers in the field of personality disorders today
Researchers who have attempted to clarify the correlates and causes of these conditions have traditionally focused largely or almost exclusively on males (e.g., Hare, 1982; Harris, Rice, & Quinsey, 1994; Hart & Hare, 1989). Consequently, little is known about the causes, assessment, and diagnosis of psychopathy and ASPD in females (Mulder, Wells, Joyce, & Bushnell, 1994; Salekin, Rogers, & Sewell, 1997; Salekin, Rogers, Ustad, & Sewell, 1998). Because the prevalence, correlates, and phenotypic manifestations of these conditions may differ in males and females, a better understanding of sex differences in psychopathy and ASPD is of considerable theoretical and practical importance.

2. Psychopathy and ASPD: conceptual and assessment issues

Most conceptualizations of psychopathy and ASPD have been either personality-based or behavior-based (Lilienfeld, 1994, 1998). Hervey Cleckley (1941/1988) provided the first comprehensive description of the psychopath’s personality in *The Mask of Sanity*. In this work, Cleckley specified 16 criteria for psychopathy, including superficial charm, lack of anxiety, unreliability, deceitfulness, lack of remorse, inadequately motivated antisocial behavior, failure to learn from punishment, egocentricity, poverty of affect and emotional bonds, lack of insight, and failure to plan ahead. With this constellation of primarily personality features, Cleckley provided the most influential personality-based approach to this condition (Lilienfeld, 1994). The diagnosis of “antisocial personality” was also primarily personality-based in the second edition of the American Psychiatric Association’s (APA) *Diagnostic and Statistical Manual of Mental Disorders* (DSM-II; APA, 1968), which emphasized selfishness, irresponsibility, impulsivity, lack of loyalty, lack of remorse, and failure to learn from punishment in its description of this condition. Both Cleckley and DSM-II noted that chronic antisocial behavior was neither necessary nor sufficient for a diagnosis of psychopathy.

Because the DSM-II criteria for antisocial personality were believed by some to be subjective and inferential, alternative and ostensibly more reliable operationalizations that emphasized chronic antisocial behavior were developed. DSM-III (APA, 1980) and DSM-III-R (APA, 1987) described ASPD as characterized by a history of delinquent and irresponsible behaviors (prior to age 15) that endure into adulthood, heralding a shift toward a more behavior-based conceptualization (Lilienfeld, 1994). The DSM-IV (APA, 1994; see also DSM-IV-Text Revision, APA, 2000) criteria for ASPD are similar to the DSM-III and DSM-III-R criteria in their behavioral emphasis.

Since the 1960s, Robert Hare and colleagues have investigated the conceptualization and assessment of psychopathy. A major achievement of this line of research has been the development of the Psychopathy Checklist (PCL; Hare, 1985b), its revision, the Psychopathy Checklist—Revised (PCL-R; Hare, 1991), and a briefer version, the Psychopathy Checklist: Screening Version (PCL:SV; Hart, Cox, & Hare, 1995). These semistructured interviews, which typically incorporate file information, include many aspects of the Cleckley criteria while assessing aspects of the DSM criteria for ASPD. Factor-analytic studies of the PCL and its progeny reveal that these measures operationalize a two-factor structure of psychopathy, with Factor 1 items assessing core personality features and Factor 2 items assessing antisocial
behaviors and poor impulse control (Hare, 1991). Measures of these two factors overlap moderately (Harpur, Hare, & Hakstian, 1989) and appear to correspond closely to the personality-based and behavior-based approaches, respectively (Lilienfeld, 1994).

When interpreting results of psychopathy and ASPD research, it is imperative to consider the specific measures used, whether they be primarily personality-based (e.g., PCL-R Factor 1 items) or more behavior-based (e.g., DSM criteria). For example, the Minnesota Multiphasic Personality Inventory (MMPI) Psychopathic Deviate (Pd) scale (McKinley & Hathaway, 1944) has often been used to assess psychopathy (Hare, 1985a), and the California Psychological Inventory (CPI) Socialization (So) scale (Gough, 1960), scored in reverse, was originally presumed to assess the role-taking deficits characteristic of psychopathy (Megargee, 1972). Research evidence suggests, however, that these two self-report measures correlate moderately with PCL-R Factor 2 but negligibly with Factor 1 (Harpur et al., 1989), and thus assess antisocial behaviors rather than the core personality features of psychopathy. For the remainder of this review, we use the term psychopathy to refer to the classical construct described by Cleckley and ASPD to refer to the DSM-IV and related conceptualizations of this syndrome.

3. Reviewing the literature on sex differences in psychopathy and ASPD

As noted earlier, little is known regarding sex differences in psychopathy and ASPD. Cleckley (1941/1988) described 15 adult patients, two of whom were female, as exhibiting “full clinical manifestations” of psychopathy, thereby acknowledging that this condition is found in both men and women. DSM-IV (APA, 1994, 2000) also noted that ASPD is both a male and female disorder in clinical and nonclinical adult populations. Although numerous authors have studied female criminality, the investigation of Cleckley psychopathy in women until recently has been largely neglected (cf. Salekin et al., 1997, 1998). Here, we summarize and integrate the empirical literature on sex differences in psychopathy and ASPD. In this review, we highlight empirically substantiated conclusions as well as inconsistent and provisional findings. Given the large number of studies on this topic, we organize them based on important methodological issues presented in order of increasing complexity. First, we address sex differences in categorical and dimensional assessments of psychopathy and ASPD. Second, we review sex differences in the correlates and factor structure of psychopathy and ASPD measures. Third, we discuss the literature on differential phenotypic manifestations of psychopathy and ASPD, as well as sex differences in the developmental trajectories of these conditions. We conclude with recommendations for future research in this area.

4. Sex differences in categorical and dimensional assessments

4.1. Categorical differences

Few studies of sex differences in psychopathy have assessed this condition categorically. One controversy regarding the conceptualization of psychopathy concerns whether this
condition is a taxon (i.e., a nonarbitrary class existing in nature) that differs in kind from normal functioning (Lilienfeld, 1998). Harris et al. (1994) applied taxometric techniques to PCL-R scores and criminality measures in a sample of male inmates and reported preliminary evidence for a psychopathy taxon. However, their evidence for a taxon derived entirely from PCL Factor 2 scores, but not Factor 1 scores, and no other published studies have directly addressed the taxonicity of psychopathy. Until further investigation of this issue is conducted, the taxonicity of psychopathy, particularly Factor 1 traits, should be considered unresolved.

Only one published study has examined PCL-R psychopathy base rates in an incarcerated female sample. Salekin et al. (1997) administered the PCL-R to 103 female inmates, and found, when using a cut-off score of 29 on the PCL-R, that 15% were psychopaths. This figure is relatively low compared with percentages ranging between 15% and 30% in male correctional samples (Hare 1991, 1996, 1998). There appear to be no other published studies examining sex differences in categorical assessments of psychopathy (but see Louks, 1995; Neary, 1990; Strachan, 1993, for unpublished data on PCL and PCL-R psychopathy base rates in female inmates ranging from 11% to 31%).

In contrast to studies of psychopathy, most studies of sex differences in ASPD have assessed this condition categorically. The last three editions of the DSM (APA, 1980, 1987, 1994) stated that ASPD is diagnosed more frequently in males than in females. In the general population, 3% of men and 1% of women meet criteria for ASPD (APA, 2000). Salekin et al. (1997) also examined DSM-III-R ASPD in their female inmate sample and found its prevalence to be 56%. Although this prevalence is lower than what is typically found in male inmate samples (e.g., Hare, 1991), it is similar to that found in some male samples (e.g., Hare et al., 1990). Salekin et al. noted that further research is necessary to determine whether there is a clear sex difference in ASPD diagnoses among prisoners.

Numerous studies have examined sex differences in ASPD diagnoses in alcohol and substance abuse settings. In a sample of 231 male and 90 female alcoholics, Hesselbrock, Meyer, and Keener (1985) reported that 49% of men and 20% of women met DSM-III ASPD criteria. In samples of alcohol and drug abusers, Flynn, Craddock, Luckey, Hubbard, and Dunteman (1996) reported that men were twice as likely as women to receive a DSM-III-R ASPD diagnosis, whereas Brown and Nixon (1997) reported nonsignificant sex differences in DSM-III-R ASPD diagnoses. It is important to note that the latter study was potentially limited by a relatively small sample, increasing the likelihood of a Type II error. Cottler, Price, Compton, and Mager (1995) found that 44% of male and 27% of female injecting drug users met DSM-III-R ASPD criteria. In methadone patients, Darke, Swift, and Hall (1994) reported that men were 2.2 times more likely than women to receive DSM-III-R ASPD diagnoses, and Rutherford, Alterman, Cacciola, and Snider (1995) similarly found that men had higher rates of DSM-III and DSM-III-R ASPD than women. In contrast, Rutherford, Alterman, Cacciola, and McKay (1998) found nonsignificant sex differences in DSM-IV ASPD diagnoses for a sample of 397 male and 121 female substance abusers.

Two studies have investigated sex differences in ASPD diagnoses in depressed outpatients. Golomb, Fava, Abraham, and Rosenbaum (1995) assessed DSM-III-R personality disorder diagnoses in depressed individuals and found that men (N=99) were significantly more likely than women (N=189) to meet criteria for ASPD on the DSM-III-R Personality Diagnostic
Questionnaire—Revised (PDQ-R; Hyler & Rieder, 1987). In contrast, they found that among those who were administered the Structured Clinical Interview for DSM-III-R Personality Disorders (SCID-II, Spitzer, Williams, & Gibbon, 1987) (48 men, 69 women), men and women did not differ significantly in the prevalence of diagnoses of ASPD. The ability to detect sex differences in structured interview ASPD diagnoses, however, may have been limited by relatively small samples. Even so, in a larger sample of depressed outpatients (99 men, 126 women), Carter, Joyce, Mulder, Sullivan, and Luty (1999) also found non-significant sex differences in SCID-II ASPD diagnoses. Potential sex differences in help-seeking behavior may account for these inconsistent findings in ASPD diagnoses (Carter et al., 1999). More specifically, men may be less inclined than women to request the help of psychiatric outpatient services and thus experience more internalizing symptoms (e.g., depression, anxiety) than females before seeking help. Consequently, men in psychiatric settings may not be representative of other populations of men, which may be more characterized by externalizing (e.g., antisocial) symptoms.

Studies of noncriminal, nonpsychiatric samples provide further information concerning sex differences in ASPD base rates. Spalt (1980) examined ASPD diagnoses in a sample of 318 male and 242 female undergraduates and found that men were more likely than women to receive a “definite” or “probable” ASPD diagnosis, as indicated by responses on self-report questionnaires. In primary care samples, Barry, Fleming, Manwell, and Copeland (1997) and Smith, Golding, Kashner, and Rost (1991) found that men were more likely than women to meet DSM-III and DSM-III-R criteria for ASPD. However, because Smith et al.’s results derive from a sample of individuals who all met criteria for somatization disorder (SD), these results may not be generalizable to nonselected samples. North, Smith, and Spitznagel (1993) assessed DSM-III-R ASPD in homeless individuals and reported results that both included and did not include one criterion that is potentially biased against the homeless (i.e., failure to plan ahead as indicated by lack of a fixed address). They found (with and without the potentially biased criterion, respectively) that 25–23% of men and 10–7% of women met DSM-III-R ASPD criteria.

Consistent with reports of ASPD prevalence in the overall population (see APA, 2000), Mulder et al. (1994) found that the overall ASPD lifetime prevalence was 3.1% for a general population survey sample and that the ASPD rate for men (4.2%) was higher than the ASPD rate for women (1.9%), although this difference was nonsignificant. In a sample of 75 male and 75 female undergraduates, Forth, Brown, Hart, and Hare (1996) found that 21.3% of men and 1.3% of women met DSM-III-R criteria for ASPD. However, because the sample was selected on the basis of meeting DSM-III-R criteria for conduct disorder [CD] (thus, likely accounting for the high ASPD prevalence for men), it is not representative of undergraduate populations at large.

4.2. Dimensional differences

Studies of sex differences in dimensional assessments of psychopathy and ASPD have examined these conditions in substance abuse and undergraduate samples. Rutherford et al. (1998) assessed 397 male and 121 female patients in drug abuse treatment and reported that PCL-R scores were significantly higher in males than females. Cooney, Kadden, and Litt
(1990) administered various psychopathy- and ASPD-related measures, including the PCL, to a sample of 79 male and 39 female inpatient alcoholics. An overall multivariate analysis of variance (MANOVA) revealed nonsignificant sex differences in all psychopathy and ASPD measures, also indicating that the men and women did not differ significantly in mean PCL total scores.

In their sample of 150 undergraduates, Forth et al. (1996) found that men scored significantly higher than women on total PCL-R:SV scores and on almost all PCL-R:SV items. Although this finding suggests that men score higher than women across psychopathy features, this study may be limited by an interviewer sex bias as all the PCL-R:SV interviewers were female. Zagon and Jackson (1994) administered the Self Report Psychopathy Scale-II (SRP-II; Hare 1991), a self-report measure modeled after the PCL-R, to 48 male and 101 female undergraduates and reported that males obtained significantly higher psychopathy scores.

The Psychopathic Personality Inventory (PPI) is a self-report measure developed to assess the psychopathic personality features delineated by Cleckley that has been found to correlate highly with the SRP-II in undergraduate samples (Lilienfeld & Andrews, 1996). Hamburger, Lilienfeld, and Hogben (1996) administered the PPI to undergraduates and found that PPI scores for 90 men were nonsignificantly higher than for 90 women. In contrast, Lilienfeld and Andrews (1996) found that undergraduate males scored significantly higher than females on PPI total scores (Cohen’s $d=.97$). Men scored significantly higher than women on six PPI subscales: Machiavellian Egocentricity (Cohen’s $d=.53$), Coldheartedness (Cohen’s $d=.73$), Fearlessness (Cohen’s $d=.79$), Impulsive Nonconformity (Cohen’s $d=.52$), Stress Immunity (Cohen’s $d=.74$), and Blame Externalization (Cohen’s $d=.19$), but there were no significant sex differences for the Social Potency and Carefree Nonplanfulness subscales. With the exception of Blame Externalization, all of the effect sizes for the scales with significant sex differences were in the medium-to-large range. [See also Cale and Lilienfeld, 2000, for a study of 75 nonclinical, nonincarcerated adults, in which men scored higher than women on PPI total scores (Cohen’s $d=.84$) but not peer ratings of Cleckley psychopathy (Cohen’s $d=.37$).]

Wilson, Frick, and Clements (1999) administered the SRP-II and the Levenson Psychopathy Scales (LPS Primary and Secondary scales; Levenson, Kiehl, & Fitzpatrick, 1995), a measure designed to assess Factors 1 (Primary) and 2 (Secondary) of psychopathy, to 91 male and 108 female undergraduates. The authors reported that men scored significantly higher than women on SRP-II Factor 1 and Factor 2 and LPS Primary and Secondary scale scores. Additionally, in a sample of 33 male and 117 female undergraduates, Lilienfeld and Hess (2001) reported that men scored significantly higher than women on SRP-II Factor 1 and Factor 2, LPS Primary and Secondary, and PPI total scale scores. The researchers also conducted analyses on PPI Factor 1 and Factor 2 scale scores, which roughly correspond to other Factor 1 and Factor 2 conceptualizations of psychopathy. They found that PPI Factor 1 scores were significantly higher for men than women, but that PPI Factor 2 scores were not significantly different across biological sex.

Few studies have examined sex differences in dimensional measures of ASPD. In their sample of inpatient alcoholics discussed earlier, Cooney et al. (1990) administered a shortened version of the MMPI Pd scale, the CPI So scale, and the ASPD section of the NIMH Diagnostic Interview Schedule (DIS; Robins, Helzer, Croughan, & Ratcliff, 1981).
Again, the overall MANOVA revealed nonsignificant sex differences across all psychopathy and ASPD measures. Hamburger et al. (1996) also assessed ASPD symptoms with the PDQ-R ASPD scale and the DSM-III MMPI Personality Disorder Scale for ASPD (ASPMMP; Morey, Waugh, & Blashfield, 1985). Although males exhibited higher scores than females on both measures, these findings were nonsignificant. (See also Cale & Lilienfeld, 2002, who reported that men scored significantly higher than women on two self-report measures, but not peer ratings, of ASPD.)

4.3. Summary and discussion

Most studies of psychopathy and ASPD indicate that men score higher than women on both categorical and dimensional operationalizations of these conditions. Given the paucity of studies of incarcerated females, however, additional studies that directly compare categorical and dimensional assessments in male and female criminals are needed to better determine whether this sex difference generalizes to such settings. In alcohol, substance abuse, and psychiatric settings, most studies have reported a higher ASPD prevalence in men, although in some psychiatric settings where clinical interviews were used to assess ASPD, findings are mixed, perhaps due to sex differences in help-seeking behaviors, the measures used (i.e., self-report vs. interview), or both. With few exceptions, studies of nonincarcerated and nonclinical individuals support the assertion that men have higher base rates and mean symptom levels of psychopathy and ASPD than women.

Additional concerns involving the characteristics of alcohol and drug-abusing samples merit further discussion. Although ASPD is one of the most frequent DSM diagnoses in alcohol and drug abusers and the overall prevalence of ASPD is higher in substance abuse settings than in the general population (APA, 2000), there are limitations in using alcohol and drug-abusing samples, particularly when investigating ASPD. Gerstley, Alterman, McLellan, and Woody (1990) argued that assessing ASPD in substance abusers may be problematic because DSM criteria focus on overt criminal behavior such as drug use, and there is no diagnostic requirement that antisocial behavior exists independently of such abuse. In addition, for these populations, both the high prevalence of ASPD and its co-occurrence with other psychological difficulties such as substance abuse and depression (e.g., see Luther, Glick, Zigler, & Rounsaville, 1993) cast doubt on their relevance to nonclinical populations, further complicating interpretations of sex differences in ASPD. As a first step in elucidating such methodological ambiguities, future research should incorporate personality-based (i.e., psychopathy) assessments in studies of alcohol and drug-abusing samples.

5. Sex differences in correlates and factor structure

5.1. Psychopathy correlates

Although the literature suggests that males score higher than females on both categorical and dimensional measures of psychopathy, less is known about how, if at all, males and
females differ in their manifestations of psychopathic features. A few researchers have examined sex differences in various psychopathological and behavioral correlates of psychopathy. The PCL-R diagnosis of psychopathy is a moderately strong predictor of recidivism in male offenders (e.g., Hart, Kropp, & Hare, 1988), and Salekin et al. (1998) confirmed this association, albeit less strongly, in their sample of female inmates. However, only Factor 1 characteristics were significantly correlated with recidivism ($r = .26$) in females, whereas both Factor 1 and Factor 2 scores were predictive of recidivism in prior studies of males. Salekin et al. did not, however, report whether the correlations between Factors 1 and 2 and recidivism were significantly different in men and women.

In Zagon and Jackson’s (1994) undergraduate sample, there were no substantial sex differences in correlations between SRP-II scores and measures of narcissism, anxiety, empathy, social desirability, and lying. They did find, however, that the negative association between SRP-II total scores and empathy was significant for women only. As a whole, Zagon and Jackson’s findings are limited in that no analyses were conducted to examine whether these correlations were significantly different in men and women. From the data presented, however, we were able to examine this issue by testing the significance of differences between the reported correlations (Cohen, 1982). These calculations indicated that the correlations between SRP-II total scores and other psychological measures were not significantly different in men and women.

5.2. ASPD correlates

Similar to the literature on psychopathy’s correlates, relatively little is known how, if at all, males and females differ in their manifestations of ASPD. As individuals with ASPD tend to have a higher prevalence of alcohol abuse and dependence than individuals without ASPD (Hesselbrock, Weiderman, & Reed, 1985; Lewis & Bucholz, 1991), a number of investigators have examined potential correlates of ASPD in alcohol abusing populations. Hesselbrock, Weiderman, et al. (1985) found that ASPD interacted statistically with biological sex to predict performance on intelligence and neuropsychological measures. Specifically, the authors reported that the association between DSM-III ASPD and Wechsler Adult Intelligence Scale (WAIS) Block Design scores was stronger for males than females and that the association between ASPD and Halstead–Reitan Neuropsychological Test Category errors was stronger for females than males. Considering that both tests require rapid problem-solving skills, the researchers suggested that this interaction may indicate that ASPD males need to be “streetwise” and make quick decisions in order to survive their antisocial lifestyles, whereas ASPD females may have different experiences demanding different cognitive abilities. Nevertheless, this interpretation is speculative and should be interpreted with caution pending replication of the unpredicted test by sex interaction. Moreover, because the ASPD diagnosis does not require that antisocial behavior exist independently of such abuse, it is difficult to know whether or not Block Design or Category error scores correlated with overall levels of ASPD or with levels of alcoholism for this sample.

Windle, Windle, Scheidt, and Miller (1995) examined abuse history and DSM-III ASPD in 481 male and 321 female inpatient alcoholics. Logistic regression analyses indicated that
patient experiences of sexual abuse, physical abuse, and both sexual and physical abuse were significant predictors of ASPD for males, whereas sexual abuse, both sexual and physical abuse, but not physical abuse alone were significant predictors of ASPD for females. Nevertheless, analyses examining biological sex as a moderator were not conducted to determine whether these associations differed significantly across biological sex. Moreover, given the absence of causal modeling in these statistical analyses, it would be premature to suggest that either sexual or physical abuse might play a causal role in ASPD (see also Zlotnick, 1999, for evidence suggesting that sexual and physical abuse histories do not predict ASPD in females). Thus, the relation between early abuse history and ASPD in males and females warrants further examination.

A number of researchers have investigated ASPD as a risk factor for alcoholism. Hesselbrock, Meyer, et al. (1985) found that 110 of 112 DSM-III ASPD male alcoholics and 17 of 18 DSM-III ASPD female alcoholics exhibited alcohol abuse symptoms secondary to the onset of ASPD, although these proportions were not significantly different. Stabenau (1984, 1990) examined the associations among various risk factors (e.g., biological sex, ASPD, family history of alcoholism) and alcoholism. In a sample of 156 male and 54 female inpatient alcoholics, he found that DSM-III ASPD did not interact with biological sex to predict an early onset of alcohol abuse. Similarly, Stabenau (1990) reported that DSM-III ASPD diagnoses predicted alcoholism in 98 male and 121 female adult offspring of alcoholics and nonalcoholics and that the association between ASPD and alcoholism was not significantly different across biological sex. Overall, these data suggest that ASPD is an equivalent risk factor for alcoholism in males and females. In contrast, Lewis and Bucholz (1991) found that biological sex and DSM-III ASPD interacted to predict alcoholism. For their sample of 1008 males and 1564 females, logistic regression analyses revealed that ASPD was significantly more associated with alcoholism in females than in males.

Drug abuse samples (i.e., individuals who abuse drugs other than, or in addition to, alcohol) have also been used to study differential correlates of ASPD. Sutker, DeSanto, and Allain (1983) administered the Adjective Checklist (Gough & Heilbrun, 1980) to 54 male and 25 female chronic drug abusers diagnosed with DSM-III ASPD. The authors found that females tended to be more negative than males in self-descriptions and, more specifically, that females viewed themselves as confused, sentimental, and worrisome. These findings suggest that ASPD females differ from ASPD males in their self-descriptions. However, these results may not be specific to ASPD because females may provide more negative self-descriptions regardless of diagnosis. For example, because females typically score higher than males on measures of Negative Emotionality (NE; Tellegen, 1978/1982), they are likely to be more self-critical in general than males.

With a few exceptions, there is limited literature on sex-differentiated ASPD correlates in noncriminal or nonclinical populations. Kosson, Steuerwald, Newman, and Widom (1994) examined the correlates of CPI So scale (which, as noted earlier, serves as a reversed measure of ASPD features) scores in 107 male and 199 female undergraduates. Negative correlations between So scale scores and number of arrests, hallucinogen use, and marijuana use were significantly stronger for males than females, and negative correlations between So scores and stealing, vandalism, barbiturate use, cocaine use, and alcohol use were nonsignificantly
stronger for males than females. In a large general population sample, Mulder et al. (1994) examined behavioral manifestations of ASPD males and females. Chi-square analyses revealed that ASPD men were more likely to engage in unlawful behavior and have more traffic offenses than ASPD women, whereas ASPD females were more likely to have relationship difficulties and exhibit lying than ASPD males. However, log-linear analyses, which would have enabled the authors to examine whether the associations between ASPD and these symptoms differed significantly in men and women, were not conducted.

5.3. Factor structure

A few studies have investigated sex differences in the factor structure of psychopathy symptoms in both incarcerated and nonclinical samples. Salekin et al. (1997) conducted exploratory factor analyses on PCL-R scores for 103 female offenders, and compared their findings to previously collected data on PCL-R factor structure in a male offender sample (see Hare et al., 1990). Salekin et al. found a two-factor structure for PCL-R scores, whereby Factor 1 items assessed core affective personality features and Factor 2 items assessed an antisocial lifestyle in females. Nevertheless, their sample exhibited more overlap among Factor 1 and Factor 2 items than has been reported for male offenders. The items assessing promiscuous sexual behavior loaded substantially (±.40) on Factor 2 for females, but these items had previously been found to load substantially on Factor 1 for males (Hare, 1991). If this finding proves replicable, the extent to which it can be explained by women’s relatively higher rates of prostitution (which might also produce higher variances of sexual promiscuity in women than in men) remains to be ascertained. Salekin et al. also reported that items assessing need for stimulation, poor behavioral controls, impulsivity, and irresponsibility loaded substantially on Factor 1 for females, whereas these items had previously been found to load on Factor 2 for males (Hare, 1991).

Salekin et al. (1997) suggested that the factor structure used to conceptualize psychopathy in males may not be applicable to females in either forensic or nonclinical populations, but this interpretation should be tempered in light of several caveats. First, some of the sex differences in factor loadings they reported appear to reflect more of an overlap between Factor 1 and Factor 2 for females than males, rather than sex-differentiated manifestations of psychopathic features. Specifically, items assessing need for stimulation, poor behavior controls, lack of realistic goals, impulsivity, and irresponsibility appeared to load substantially (≥.34 for each item) on both Factor 1 and Factor 2 for females. Second, the relatively small female sample size may have limited the stability of the PCL-R’s factor structure. When replicating factor structures in new samples, it is recommended that a minimum of five individuals be assessed per item analyzed and that the total sample consist of at least 100 individuals (Gorsuch, 1983). Depending on the communalities among items and how well determined the factors are, some recommend sample sizes ranging from 200 to 500 (MacCallum, Widaman, Zhang, & Hong, 1999). A conservative interpretation of Salekin et al.’s factor analysis is that only promiscuous sexual behavior loaded differently in females and males. Further complicating Salekin et al.’s findings is the fact that factor analyses were conducted by combining African Americans and Caucasians (Vitale & Newman, 2001), and there is some preliminary evidence (e.g., see...
Kosson, Smith, & Newman, 1990) that the factor structure of the PCL differs across these two races (but see Windle & Dumenci’s, 1999 confirmatory factor analysis of 740 inpatient alcoholics, in which item–factor relations for PCL-R Factor 1 and Factor 2 were supported across African Americans, Puerto Ricans, and Caucasians).

Because Hare et al. (1990) and Salekin et al. (1997) provided individual factor loadings for each PCL-R item in their respective samples, we examined the similarity of the PCL-R factor structure in males and females. Coefficients of congruence (see Cattell, 1978) between samples’ factor loadings were calculated to compare the psychopathy factor structures in males and females. For Hare et al.’s and Salekin et al.’s samples, the congruence coefficients between the PCL-R factors were moderately high (.859 and .856 for Factors 1 and 2, respectively), suggesting that Factors 1 and 2 assess similar attributes in males and females. Admittedly, the use of congruence coefficients when comparing factor structures is limited because there is no method of interpreting the statistical significance of such coefficients (see Pinneau & Newhouse, 1964). Nevertheless, our supplementary analyses do not support the claim that the factor structure of psychopathy differs markedly in males and females.

Cooney et al. (1990) conducted a factor analysis on psychopathy scores in their sample of 118 inpatient alcoholics to determine whether various measures of psychopathy and ASPD assess a similar construct in males and females. A one-factor solution was found for four psychopathy-related measures (i.e., PCL, MMPI Pd scale, CPI So scale, DIS ASPD symptoms), although the factor loadings of individual PCL items were not reported. The order of factor loadings for the individual scales did not differ across biological sex. Cooney et al. also conducted separate factor analyses for men (N=79) and women (N=39). Although the factor loadings were similar for both men and women, these samples are quite small for factor analysis and are likely to result in unstable factor structures (see Gorsuch, 1983; MacCallum et al., 1999).

Several authors have reported factor analyses of psychopathy measures in nonclinical samples. Lilienfeld and Andrews (1996) conducted factor analyses on male and female undergraduate psychopathy scores, and reported no clear evidence for sex differences in factor structure. Wilson et al. (1999) conducted principal axis factor analyses on various personality (i.e., “Factor 1”) and behavioral (i.e., “Factor 2”) indices of psychopathy in male and females undergraduates, and found that the indices loaded similarly across biological sex. Forth et al. (1996) obtained a one-factor solution for PCL-R:SV scores in both male and female undergraduates and found that this factor accounted for less variance in females than in males. Again, however, this study’s small sample sizes of men (N=75) and women (N=75) and the requirement that all participants meet criteria for CD render these findings difficult to interpret. Furthermore, all these nonclinical samples exhibited relatively low variances (and thus, covariances) in scores, which limit the ability to replicate factor structures (MacCallum et al., 1999).

5.4. Summary and discussion

Few conclusions can be drawn from studies of the correlates and factor structure of psychopathy and ASPD. There is provisional evidence that ASPD correlates differently in
males and females with neuropsychological variables, although further research is needed to replicate and clarify these findings. There is relatively little evidence that the behavioral, psychological, and abuse history correlates of psychopathy and ASPD differ in males and females. In addition, findings are mixed as to whether ASPD predicts alcoholism more strongly in males than females and whether ASPD correlates with different types of criminal behavior in males and females. There is no convincing evidence that the factor structure of psychopathy differs in males and females, although factor analyses on larger samples are needed to better evaluate this issue.

Issues regarding sample selection should be considered when examining the correlates and factor structure of psychopathy and ASPD. As discussed earlier in reference to categorical and dimensional measures, it is critical to attend to the problems associated with using alcohol and substance abuse samples in such investigations. When examining potential correlates of ASPD, studies of alcohol and drug abusers have typically not controlled for ASPD criterion endorsement due to alcohol or drug abuse. Substance abusers almost certainly comprise both psychopathic and nonpsychopathic individuals (Gerstley et al., 1990), and psychopathy has been assessed less frequently than ASPD in studies of sex-differentiated correlates in substance abusers. For these reasons, we advise researchers to administer both psychopathy and ASPD measures in these samples.

Both incarcerated and clinical samples may be useful in examining differential correlates and factor structure because, compared with nonclinical samples, these samples may exhibit greater variances of psychopathy and ASPD features (Lilienfeld, 1998). It may be more useful to use dimensional measures than categorical measures of ASPD when examining ASPD correlates in these samples because dimensional assessments better allow the investigation of correlates within only mildly antisocial samples (Strain, 1995). In addition, restricted variances of these features may limit the investigation of factor structure in undergraduate samples. On the other hand, because undergraduate samples are potentially useful in that findings may be generalizable to other nonclinical samples, we therefore recommend that, whenever possible, researchers examine the factor structure of psychopathy measures in both noninstitutionalized and institutionalized samples.

6. Sex differences in phenotypic manifestations

Some authors have hypothesized that males and females possess the same underlying personality features of psychopathy, but differ in their overt behavioral manifestations (e.g., Hamburger et al., 1996). This issue is relevant to McCrae and Costa’s (1995) distinction between basic tendencies and characteristic adaptations. According to this distinction, basic tendencies are underlying core features of personality, whereas characteristic adaptations are overt behaviors that result from the transaction between these core features and the environment. Because quite disparate phenotypic manifestations may reflect the same underlying basic tendencies (Harkness & Lilienfeld, 1997), biological sex may shape the personality dispositions of psychopathy into different overt conditions. This possibility is particularly relevant to understanding psychopathy and ASPD because a number of authors
have conjectured that SD, histrionic personality disorder (HPD), and possibly borderline personality disorder (BPD) (Paris, 1997; see also Hudziak, Boffeli, Battaglia, Stanger, & Guze, 1997; Morgenstern, Langenbusher, Labouvie, & Miller, 1997) are predominantly female manifestations of underlying psychopathic tendencies, whereas ASPD is a predominantly male manifestation of such tendencies. Although these syndromes (i.e., SD, HPD, and ASPD) are superficially different from each other, such differences may mask fundamental similarities in their etiologies.

SD’s and HPD’s roots extend back to early descriptions of “hysterical neurosis,” which included conversion, somatization, dissociation, and histrionic features. Today, SD is classified as a somatoform disorder in which individuals exhibit multiple physical symptoms, with no identified organic causes, across various bodily systems. HPD is a personality disorder characterized by attention seeking, seductiveness, and overemotionality (APA, 2000). SD and HPD tend to covary across individuals (e.g., Lilenfeld, Van Valkenburg, Lamntz, & Akiskal, 1986). SD is found more frequently in females than in males, perhaps due to gender role influences. Moreover, in clinical settings, HPD is diagnosed more in females than males, although this sex difference may result from the greater ratio of females than males in mental health settings (APA, 2000).

Some markers of SD and HPD appear to overlap considerably with those of ASPD. SD and ASPD share such correlates as family histories of both SD and ASPD (e.g., Cloninger, Reich, & Guze, 1975a, 1975b) and a chronic and largely unremitting course (Lilenfeld, 1992). Individuals with HPD and ASPD share a propensity towards impulsivity, superficiality, excitement seeking, recklessness, seductiveness, and manipulativeness (APA, 2000), all of which reflect aspects of the prototypical Cleckley psychopath. In addition, HPD and ASPD are both included in the DSM-IV Cluster B personality disorders, which are characterized by dramatic, emotional, and erratic behaviors (APA, 2000).

It is plausible that males and females differ in their manifestations of antisocial behaviors rather than in the core affective and interpersonal features of psychopathy. Some researchers have argued that SD and HPD may be different but overlapping manifestations of the same underlying diathesis toward psychopathy, whereby males are diagnosed more often with ASPD and females are diagnosed more often with SD or HPD. These arguments are especially intriguing because SD and HPD exhibit characteristics (e.g., conversion symptoms, overemotionality) that are ostensibly quite different from those of prototypical psychopathy and ASPD (e.g., lack of remorse, criminality).

Several studies provide suggestive evidence that SD and HPD are female-typed manifestations of underlying psychopathic propensities. In his undergraduate sample, Spalt (1980) found that the base rate of ASPD among SD females was significantly higher than the base rate of ASPD among non-SD females (22.8% vs. 8.1%, respectively). Similarly, the base rate of SD among ASPD females was significantly higher than the base rate of SD among non-ASPD females.
ASPD females (46.1% vs. 20.6%, respectively). In contrast, base rates of ASPD were not significantly different between SD and non-SD males (34.1% vs. 36.1%, respectively), and base rates of SD were not significantly different between ASPD and non-ASPD males (12.3% vs. 13.2%, respectively). Although these findings suggest a significant association between ASPD and SD symptoms in females only (Spalt, 1980), there are reasons to question this interpretation. The self-report questionnaires used to assess SD and ASPD symptoms yielded unusually high prevalence rates of these disorders compared with population norms (see APA, 2000). In addition, log-linear analyses were not conducted to examine whether the associations between SD and ASPD differed significantly across biological sex.

Wilson et al. (1999) examined correlations among measures of psychopathy and somatic complaints in male and female undergraduates. The researchers reported that in both males and females, LPS Primary scores and SRP-II Factor 1 scores correlated negatively with somatic complaints, whereas LPS Secondary scores and SRP-II Factor 2 scores correlated positively with somatic complaints. However, the correlations were significant for the LPS scales only, and the authors did not conduct analyses to examine whether the correlations between psychopathy factors and somatic complaints differed significantly in males and females. We were able to test the significance of the differences in correlations between psychopathy scores and somatic complaints in males and females, and found that correlations between LPS Primary, LPS Secondary, SRP-II Factor 1, and SRP-II Factor 2 scores and somatic complaints were not significantly different across biological sex.

Lilienfeld and Hess (2001) also examined correlations among measures of psychopathy and somatic complaints. They found that “Factor 2” psychopathy indices (i.e., SRP-II Factor 2, LPS Secondary, and PPI2 scale scores) were significantly positively correlated with somatization scores in female, but not male, undergraduates and that these three correlations were significantly stronger for females than males. Moderated multiple regression analyses revealed that the interaction between secondary psychopathy and biological sex was significant for the LPS Secondary and PPI2 scores and marginally significant ($P = .07$) for SRP-II Factor 2 scores. Given that the male sample was small ($N=33$) and that moderator effects tend to be difficult to detect because of low statistical power (Jaccard, Turrisi, & Wan, 1990), this study provides fairly consistent support for the claim that the behavioral features of psychopathy are positively related to somatic complaints, and that these associations are stronger for females than males. Lilienfeld and Hess also found weak and inconsistent associations between “Factor 1” psychopathy indices (i.e., SRP-II Factor 1, LPS Primary, and PPI1 scale scores) and somatization scores.

Female criminals have been used to examine the overlap among SD, HPD, and ASPD. Cloninger and Guze (1970a, 1970b) examined the prevalence of DSM-II diagnoses of 66 female felons who were either on probation or parole. They found that 39% were diagnosed with ASPD, 15% were diagnosed with SD, and 26% were diagnosed with both ASPD and SD. Of those with SD features, 60% manifested HPD features. However, their small sample and absence of a male comparison sample precluded further testing of biological sex as a moderator.

There is relatively little research on SD and HPD in males. Luisada, Peele, and Pittard (1974) identified 27 male psychiatric patients diagnosed with DSM-II HPD and reported that
most of the sample had abused alcohol or drugs and had been charged for impulsive crimes such as drunkenness or robbery. Based on these findings, the researchers suggested that HPD males are more likely to commit antisocial acts than HPD females, whereas HPD females are more likely to have histories of surgical procedures. Clearly, this study was marked by several important limitations, including a small sample size and an absence of a comparison group of either HPD females or non-HPD males. Lilienfeld et al. (1986) examined the associations among DSM-III ASPD, SD, and HPD diagnoses in a sample of 250 patients from mental health institutions, and found that the three disorders covaried extensively across individuals. More specifically, the authors found that ASPD was significantly related to both SD and HPD in males and females, and that ASPD was significantly more strongly related to HPD than SD for the entire sample.

In a study of 90 male and 90 female undergraduates discussed earlier, Hamburger et al. (1996) applied structural equation modeling to self-report data and found that psychopathy was significantly associated with both ASPD and HPD features. In addition, biological sex moderated the relations between psychopathy and other personality disorders, such that there was a significantly stronger association between psychopathy and ASPD for males, whereas there was a significantly stronger association between psychopathy and HPD for females. Nevertheless, these differences were relatively weak in magnitude. Hamburger et al.’s findings provide provisional evidence that biological sex moderates the relations between psychopathic features and their presumed antisocial and histrionic manifestations. These findings warrant replication in clinical samples using alternative (i.e., non-self-report) measures of psychopathy, ASPD, and HPD.

In an attempt to explain putative sex differences in the behavioral expressions of an underlying predisposition toward ASPD, Cloninger and colleagues (e.g., Cloninger et al., 1975a, 1975b) reported evidence consistent with a shared etiology for ASPD and SD, whereby males and females differ in their thresholds for manifesting ASPD and SD symptoms. Cloninger (1978) reviewed the literature linking ASPD and SD, and noted that ASPD and SD tend to aggregate within the same families (see also Frick, Kuper, Silverthorn, & Cotter, 1995; Lilienfeld et al., 1986, for evidence that SD individuals tend to report elevated levels of ASPD in their first-degree relatives). He presented the multifactorial model of disease transmission to explain the pathogenesis of these disorders. This model allows for genetic, familial environmental, and nonfamilial environmental factors to contribute to one’s threshold for manifesting symptoms of a disorder. Thus, those who have a higher transmitting load of these features have a lower threshold for exhibiting the disorder themselves. With regard to ASPD and SD, Cloninger and colleagues investigated whether males and females with either SD or ASPD differed in their familial load of these disorders.

Cloninger and colleagues found evidence in support of the multifactorial model’s account of familial influences in the development of ASPD and SD. Cloninger et al. (1975a) assessed 58 ASPD males and 28 ASPD females and found that a two-threshold multifactorial model supported the hypothesis that ASPD females are more severely affected than ASPD males. Specifically, ASPD females were found to have more first-degree relatives with either ASPD or SD than were ASPD males. Cloninger et al. (1975b) also assessed a sample of 800 individuals, which included ASPD males, ASPD females, and SD females. They found that a
three-threshold multifactorial model supported the hypothesis that ASPD females are more severely affected than both SD females and ASPD males because ASPD females had more ASPD and SD relatives. Considering the associations between SD and HPD discussed earlier, this finding is potentially consistent with the hypothesis of a shared etiology for ASPD and HPD features, whereby ASPD in females is a more severe manifestation of this etiology. No published studies, however, have tested the multifactorial model in explaining sex differences in ASPD and HPD manifestations. In addition, this model has not been used to determine whether SD or HPD males are more severely affected than SD or HPD females.

6.1. Summary and discussion

There appears to be considerable evidence that SD and HPD features correlate with ASPD features among individuals, with some provisional evidence that the covariation among these conditions is more consistent in females than males. Such findings suggest that there may be a shared etiology among these disorders, whereby their overt manifestations differ across biological sex. Researchers have recently begun examining whether biological sex moderates the relations between an underlying propensity toward psychopathy and overt ASPD, SD, and HPD manifestations, such that psychopathic males tend to have more ASPD features than females whereas psychopathic females tend to have more SD and HPD features than males. Future studies using self-report, interview, and family history measures, and examining biological sex as a moderator variable, should help to elucidate these issues.

It is worth noting that other authors have posited that sex differences in the prevalences of psychopathy, ASPD, and other conditions (e.g., SD, HPD) are due to sex bias in diagnosis. Several researchers have found that mental health professionals are more likely to diagnose males with ASPD and females with HPD even when the individual case descriptions are identical or nearly identical (see Belitsky et al., 1996; Ford & Widiger, 1989; Hamilton, Rothbart, & Dawes, 1986; Warner, 1978). On the other hand, Ford and Widiger (1989) found that sex bias did not appear to be operating when psychologists rated individual criterion symptoms as characteristic of either ASPD or HPD for males and females. In light of these potentially conflicting findings, clinicians should consider guarding against possible bias, perhaps by using structured clinical interviews in conjunction with self-report measures (Pfohl, 1995).

There are other issues to consider with regard to the potential problem of diagnostic sex bias. For example, Funtowicz and Widiger (1999) tested the hypothesis that DSM-IV diagnoses of some personality disorders are biased against women because these diagnoses require less dysfunction to be diagnosed with a female-typed disorder (e.g., HPD), but they found no support for this contention. It is also important to bear in mind that if there are genuine sex differences in the base rates of ASPD and HPD, then clinicians’ diagnoses of these conditions may reflect the inherent probabilities (i.e., base rates) of individuals diagnosed with either ASPD or HPD, rather than sex bias (Ford & Widiger, 1989). To clarify this issue, researchers should examine sex differences in individual criterion endorsement as well as in overall base rates. More broadly, when designing studies, researchers should first consider whether their selected methods of assessing psychopathy, ASPD, and HPD are potentially sex-biased.
7. Sex differences in developmental trajectories

7.1. Childhood antisocial behavior

Before the age of 18, individuals with CD exhibit a pattern of violating others’ rights, actual or threatened harm of others, stealing or destroying property, deceitfulness, and serious violation of rules (APA, 2000). DSM-IV delineated two classifications of CD: childhood-onset (before age 10) and adolescent-onset (after age 10), and noted that males are more frequently diagnosed with CD than females (APA, 2000; see also Cottler et al., 1995). In addition, some researchers have found that males and females differ in their adult manifestations of specific antisocial and criminal behaviors. Here, we review literature on sex differences in psychopathy and ASPD manifestations at different ages and discuss whether sex-specific criteria for these syndromes are needed.

Although the prevalence of CD has been increasing in females over time, males tend to have higher rates of CD than females. In addition, the sex ratio for CD decreases in adolescence (Silverthorn & Frick, 1999). Silverthorn and Frick (1999) reviewed this literature and concluded that (1) before age 5, rates of conduct problems are similar across biological sex; (2) after age 5, females have fewer conduct problems than males; and (3) CD symptoms increase for both males and females in adolescence (see also Rounds-Bryant, Kristiansen, Fairbank, & Hubbard, 1998).

There are a few studies of sex differences in the relations between ASPD and CD features. In a sample of methadone patients, Rutherford et al. (1998) found that the correlation between PCL-R Factor 1 symptoms and DSM-III-R CD symptoms was significant among males, but not females. Nevertheless, the difference between these correlations was nonsignificant. In a sample of DSM-III-R ASPD alcoholics and substance abusers, Brown and Nixon (1997) found significant correlations between adult ASPD symptoms and CD symptoms, as assessed by the Childhood Behavior Disorders Checklist (Tarter, McBride, Buonpane, & Schneider, 1977), among males but not females. However, analyses were not conducted to examine whether these associations were significantly different across biological sex. In their sample of drug abusers with DSM-III-R ASPD, Goldstein et al. (1996) reported adjusted odds ratios of the proportions of individuals meeting each ASPD criterion. They found no sex differences in the age of onset for CD. Analyses of CD criteria indicated that as children, ASPD females ran away significantly more than ASPD males and that ASPD males used weapons, were cruel to animals, set fires, and vandalized significantly more than ASPD females.

Sex differences in ASPD–CD relations have also been examined in nonpsychiatric individuals. In their sample of homeless individuals, North et al. (1993) used logistic regression analyses to determine which specific ASPD criteria were predicted by total number of CD symptoms and found that for males but not females, CD symptoms significantly predicted later employment or financial difficulties. They also found that CD symptoms predicted lack of remorse in females but that the association fell short of significance for males. North et al. did not test whether these associations differed significantly between males and females.

Several researchers have examined sex differences in early externalizing (i.e., conduct disordered, oppositional) behaviors, which are associated with adult ASPD (APA, 2000). In
samples of children and young adults, Crick and colleagues (e.g., Crick, 1997; Werner & Crick, 1999) have distinguished between two types of aggressive behavior. Compared with overt forms of aggression (i.e., behaviors that harm others via physical damage or threats of physical damage) such as fighting and fire setting, relational aggression includes behaviors whereby relationships, rather than overt forms of aggression, serve as the means for a youth’s antisocial behavior (e.g., threatening classmates, spreading rumors about others) (Crick, 1997). Crick (1995) found that boys and girls between the ages of 9 and 12, who engaged in gender nonnormative forms of aggression (i.e., overt aggression in girls and relational aggression in boys), were significantly more maladjusted socially and psychologically than children who engaged in gender normative behavior. Others (e.g., APA, 2000; Rutherford et al., 1995) have suggested that rather than engaging in aggressive behaviors, young girls may engage in minor norm-breaking behaviors and assume adult roles, perhaps by stealing or finding ways to obtain money, clothes, or drugs. Alternatively, these sex differences may be artifacts of sex-biased CD criteria.

A few studies have focused on sex differences in adolescent CD manifestations. This line of research is particularly relevant to the diagnosis of ASPD because a number of late-onset CD individuals may display adult ASPD symptoms but not be diagnosed with ASPD because they do not exhibit CD symptoms before age 15. For example, Zoccolillo (1993) reviewed studies that showed no sex differences in CD rates for adolescents, whereas sex differences in CD were evident in studies of preadolescents. In a longitudinal study of 1254 male and 1157 female adolescents, Windle (1990) examined sex differences in antisocial behaviors in early (i.e., ages 14 and 15) and late (i.e., ages 18 and 19) adolescence. Although males were found to commit more property and violent crimes and engage more in substance use than females, there was no sex difference in males’ and females’ running away from home. Windle also found that although antisocial behaviors more highly correlated with substance use among early adolescent males than females, most correlations among antisocial behaviors were not significantly different across sex.

Several authors have posited and tested theories attempting to explain sex-typed developmental pathways for CD. Keenan and Shaw (1997) hypothesized that sex differences in problem behaviors from infancy to school age are indicative of early problem behaviors being channeled into predominantly externalizing disorders for boys and predominantly internalizing disorders for girls. In a review of the literature, Zoccolillo (1993) concluded that for boys, but not girls, externalizing disorders at age 11 predict externalizing disorders at age 15. He also found that at ages 11 and 15, females are less likely than males to manifest criminal, particularly aggressive, behaviors and are more likely than males to manifest SD symptoms alone or in conjunction with externalizing behaviors. Zoccolillo further argued that the sex difference in behaviors among CD individuals may be due to additive, but not interactive, effects of CD and biological sex, so that the correlations between CD and both aggression and internalizing disorders are similar in boys and girls.

Silverthorn and Frick (1999) suggested that traditional conceptualizations of CD development (i.e., childhood-onset and adolescent-onset; APA, 2000; Moffitt, 1997) do not apply to females, and proposed a unique, female “delayed-onset” developmental ASPD pathway. They noted that although girls do not tend to manifest antisocial behaviors until adolescence,
they show many of the same pathogenic mechanisms that are associated with the childhood-onset pathway in boys (e.g., processes that consistently lead to failed interactions with others). In addition, the authors proposed that girls share similar vulnerabilities with early-onset CD boys but do not manifest severe CD behavior until adolescence, when there are sex-specific biological and social changes. As females tend to have later onsets of CD than males, females with early-onset CD are especially antisocial, even through adulthood. More specifically, ASPD females who meet criteria for CD are atypical and more deviant than other antisocial (i.e., adult-only ASPD) females and males with CD. Overall, these suggestions (e.g., Keenan & Shaw, 1997; Silverthorn & Frick, 1999; Zoccolillo, 1993) warrant further empirical investigation.

Given the relatively low correlations between childhood CD and adult ASPD criteria in females, several authors (e.g., Rutherford et al., 1995; Zoccolillo, 1993) have questioned the relevance of some CD criteria for assessing ASPD in females. Moreover, the CD criteria have changed progressively with DSM revisions. For example, some of the CD criteria that were dropped in the revision of DSM-III were arguably the most useful in assessing CD in females (e.g., running away, poor school attendance and performance). Widiger and Corbitt (1995) argued that some DSM-IV CD criteria (e.g., forced sexual activity, which is rarely found in CD females) are not especially valid for assessing CD in females. Instead, some criteria that are not presently used to assess CD (e.g., sexual promiscuity, prostitution) could be more specific and diagnostic of CD in females (Widiger & Corbitt, 1995). Zoccolillo (1993) further suggested that because the CD criteria may fail to diagnose girls at age 11, sex-specific CD criteria, consistent with sex differences in base rates of aggression and antisociality in children, should be used. Zoccolillo, Tremblay, and Vitaro (1996) found that using altered, sex-specific CD criteria improved the sensitivity of assessing CD in females, whereas specificity remained unchanged.

To date, there is relatively little theoretical or empirical evidence supporting the use of sex-specific CD criteria (see also Zahn-Waxler, 1993, for arguments against using sex-specific CD criteria). Silverthorn and Frick (1999) argued that the assumption that CD is measured improperly across biological sex does not explain why some girls manifest CD symptoms like boys and why there are sex-differentiated changes in CD manifestations across ages. They also contended that there is little evidence that sex-specific CD criteria would identify the same construct in both sexes. Although such issues have been discussed in reference to the assessment of ASPD and CD, they also apply to whether psychopathy should be assessed differently in boys and girls. Before sex-specific criteria are implemented, a better understanding of (1) which criteria are most valid in assessing CD in males and females, and (2) the potentially different developmental trajectories of CD in males and females is needed.

7.2. Adult antisocial behavior

As previously discussed, many researchers have found sex-differentiated correlates of ASPD, some of which are specific to adult antisocial behaviors. Rutherford et al. (1998) found that female substance abusers exhibited stronger associations between PCL-R Factor 1 scores and adult ASPD criteria than males. In their study of injecting drug abusers, Cottler
et al. (1995) found that females endorsed more adult-only DSM-III-R criteria than males. Similarly, in their sample of treatment drug abusers with DSM-III-R ASPD, Goldstein et al. (1996) reported adjusted odds ratios indicating that females endorsed more adult ASPD criteria than males. Overall, these studies provide provisional evidence that the association between psychopathy and adult antisocial behavior is stronger for females than males.

Some authors have argued that sex-specific criteria should be used when assessing adult criminals for ASPD. This contention is based partly on the fact that there appear to be male-typed offenses such as rape, robbery, and pedophilia, and female-typed offenses such as child abuse, shoplifting, and prostitution (Heidensohn, 1968; Widom, 1984; see also Eagly & Steffen, 1986, whose meta-analytic review revealed that men tend to engage in more physically aggressive and psychologically harmful acts than women). Psychopathy and ASPD appear to be valid constructs in female prostitutes (see De Schampheleire, 1990), but there are no published studies examining the validity of psychopathy or ASPD in females who have committed other female-typed crimes (e.g., child abuse, shoplifting). In a sample of adult female offenders awaiting trial for a variety of offenses, Widom (1978) conducted cluster analyses on various measures of psychopathy and ASPD and reported evidence for four distinct profile types, including primary (i.e., Cleckley) psychopathy, neurotic psychopathy, overcontrolled criminality, and normal criminality. Widom acknowledged, however, that further investigation of adult female offenders is needed.

7.3. Summary and discussion

Overall, there is evidence that males and females differ in the developmental courses of psychopathy and ASPD. Between the ages of 5 and adolescence, males manifest more externalizing symptoms than females, whereas females manifest more internalizing symptoms than males, although this sex difference diminishes in adolescence. Moreover, boys and girls differ in what types of antisocial behaviors and aggression they exhibit. Differences in types of antisocial and criminal behaviors also seem to extend to adulthood, although few studies have examined such differences. To date, there is no compelling evidence to support the claim that psychopathy or ASPD criteria should be tailored specifically to assessing either male or female adults. To clarify this issue, researchers should investigate whether sex-specific criteria assess these constructs better than non-sex-specific criteria, such that sex-specific criteria correlate better than non-sex-specific criteria with other indicators of these conditions (e.g., putative laboratory, biological, family history, and natural history correlates of psychopathy and ASPD).

8. Conclusion and integration

In this review, we have brought together the existing empirical literature on sex differences in psychopathy and ASPD. In this concluding section, we wish to highlight recommendations for future research. The most consistent finding across studies is that psychopathy and ASPD are more prevalent in males than in females. However, the magnitudes of these sex
differences are uncertain, and the extent to which these magnitudes differ across sample characteristics is unknown. We first recommend that researchers design studies using sufficient sample sizes so that categorical and dimensional indicators of psychopathy and ASPD can be systematically compared in males and females. In addition, to better determine the generalizability of sex differences in psychopathy and ASPD, researchers should ascertain whether findings extend to forensic, clinical, substance abuse, and noninstitutionalized populations. Sex differences in psychopathy have more often been examined in forensic and undergraduate samples than in clinical and substance abusing samples, whereas sex differences in ASPD have more often been examined in clinical and substance abuse than in forensic and undergraduate samples. Findings in incarcerated female samples should be compared with those of nonincarcerated female samples because, for example, cognitive or demographic factors (e.g., intelligence, socioeconomic status) may account for differences in the behavioral manifestations of these samples. In addition, because psychopathy may be manifested differently in non-Caucasians than in Caucasians (e.g., see Kosson et al., 1990), future studies should examine sex differences in the correlates of psychopathy across race.

In addition, researchers should not examine sex differences in these conditions using categorical methods of assessment only. Even if psychopathy was found to be taxonic, this would not imply that a categorical approach to assessment is warranted. We recommend a dimensional framework given that dimensional measurements can be used to assess categories, but not vice versa (Grove & Tellegen, 1991). Moreover, dimensional measures tend to provide more valid assessments than categorical measures of taxonic constructs (Gangestad & Snyder, 1991). Also, a dimensional model may also be more useful for assessing psychopathic and ASPD features in noninstitutionalized samples because the psychopathology of individuals in these samples is relatively mild and often does not severely disrupt social functioning, and consequently, the base rates of categorically defined psychopathy and ASPD tend to be low (Forth et al., 1996; Gunderson, Links, & Reich, 1991). Furthermore, analyses conducted at the item level on measures of psychopathy and ASPD, such as item response theory (IRT) analyses (Cooke & Michie, 1997), may reveal items that are more discriminating of these syndromes in males or females. If individual criteria are found to be more discriminating of these disorders in males or females, further research may focus on these specific sex-differentiated features.

The possibility of specific sex-differentiated manifestations of these disorders merits greater consideration. We recommend that researchers test hypotheses concerning sex differences in the putative phenotypic manifestations of psychopathy and ASPD (e.g., SD and HPD) by examining whether biological sex moderates the phenotypic expressions of these disorders. Determining whether associations between these conditions and other potential phenotypic expressions differ in males and females remains an important issue. Future research should examine sex differences in other potential phenotypic expressions of psychopathy and ASPD, including BPD (see Hudziak et al., 1997) and Type II alcoholism (see Cloninger, 1987). In addition, hypotheses should focus on sex-differentiated developmental trajectories of these conditions (e.g., a “delayed onset” ASPD pathway in females; Silverthorn & Frick, 1999) to better elucidate differences in the course of psychopathy and ASPD in males and females.
More broadly, we suggest that researchers adopt a construct validational approach toward investigating psychopathy and ASPD, which in turn, should clarify many of the issues regarding sex differences we have raised. In other words, researchers should design studies by embedding psychopathy and ASPD within a “nomological network” (i.e., an interlocking system of laws that link a construct to observable variables as well as to other constructs; see Cronbach & Meehl, 1955). Robins and Guze’s (1970) guidelines for diagnostic validation comprise five phases (i.e., clinical description, laboratory studies, delimitation from other disorders, follow-up study, and family study), which altogether provide a construct validational perspective for examining empirically the commonalities between these disorders in males and females.

Consistent with Robin and Guze’s (1970) approach, which includes examining clinical features and differences across demographic variables (e.g., sex, race, age), researchers should examine familial prevalence rates of psychopathy, ASPD, and related conditions. Although decades ago, Cloninger et al. (1975a, 1975b) tested the multifactorial model’s account of the transmission of ASPD and SD in males and females, this area has since been neglected, especially with regard to the transmission of psychopathic and HPD features in relatives. Although the multifactorial model makes no assumptions regarding the relative primacy of either genetic or environmental factors (Cloninger, 1978), it proposes testable hypotheses regarding the patterns of sex-differentiated familial transmissions of psychopathy and ASPD.

We further recommend that researchers incorporate putative laboratory measures, including both biological and behavioral indices, in their research to better ascertain the construct validity of psychopathy and ASPD in females. For example, compared with nonpsychopaths, psychopaths exhibit lowered skin conductance responses to conditioned aversive stimuli (Arnett, Howland, Smith, & Newman, 1993; Jutai & Hare, 1983). In addition, some research suggests that psychopaths exhibit poor passive avoidance learning (i.e., the failure to inhibit a previously punished response) (e.g., Lykken, 1957; Newman, Patterson, Howland, & Nichols, 1990) and deficits in response modulation (i.e., the capacity to ignore extraneous cues, including punishments, when engaged in a dominant response set; Newman, 1987; Newman, Schmitt, & Voss, 1997). Presently, there are no published studies on the relations between psychopathy and laboratory measures in females.

Because relatively little is known about the course, outcome, and treatment amenability of individuals with psychopathy and ASPD, sex differences in these variables require clarification. The long-term prognosis of psychopaths is still a matter of some dispute (Hare, 1998), and the extant literature on psychopathy’s prognosis and treatment amenability consists exclusively of studies on men. Some evidence suggests, however, that male psychopaths begin their criminal careers at relatively young ages (Hart & Hare, 1997). Moreover, male criminal offending tends to decrease around the age of 40 (Hare, 1996), although core psychopathic features appear to remain stable with age. In addition, there is no compelling evidence that male psychopaths are responsive to treatment, although further research on this issue is warranted (Hart & Hare, 1997). Given that these findings are preliminary and have yet to be examined in females, it would be premature to generalize these findings across biological sex.
We also recommend that future research address questions concerning additional variables that have been posited to influence the overt manifestations of psychopathy and ASPD across biological sex. Because aggressive behavior is a manifestation of psychopathy and ASPD, researchers have investigated the role of hormones, neurotransmitters, and other biological variables as potential mediators of sex differences in these conditions. For example, some have suggested that males’ higher levels of testosterone compared with females are related to their higher rates of antisocial behavior (Lilienfeld, 1992). In addition, there is provisional evidence suggesting that levels of serotonin and monoamine oxidase (MAO) activity are negatively associated with psychopathy, criminality, and impulsivity and tend to be lower in males than in females (Ellis, 1991; Steiner, Lepage, & Dunn, 1997). These findings notwithstanding, the mediating roles of these biological variables in sex-differentiated manifestations of psychopathy and ASPD are not well understood (see Mazur, 1983; Sapolsky, 1997).

Some authors have also argued that gender roles (i.e., the attitudes, behaviors, and emotions typically associated with a particular sex; Bem, 1974) also mediate the manifestations of antisocial behaviors, whereby males are socialized to be independent and aggressive and females are socialized to be dependent (Lilienfeld, 1992; Nichols, 1996; Nuckolls, 1992; Widom, 1984; see also Forth et al., 1996). Gender role socialization may also affect the types of aggression in which individuals engage (Crick, 1997; Werner & Crick, 1999). For example, there is evidence that physiological aggression correlates more with ASPD symptoms in males than in females, perhaps because of sex differences in perceived consequences learned via gender roles (Magdol et al., 1997). Nevertheless, it is quite possible that ostensibly male- and female-typed crimes may reflect preexisting sex differences in the types of crimes men and women commit independently of gender role socialization.

Biological variables and gender role socialization are two primary sets of influences that have been hypothesized to mediate the associations between underlying psychopathic conditions and aggressive behaviors. Researchers have not, however, explicitly examined whether biological and social variables moderate the relations between psychopathy and overt phenotypic expressions, whereby such variables interact statistically with psychopathy to differentially affect the magnitude of these associations across biological sex. Thus, the variables we have outlined as potential mediators may be further tested as potential moderators of sex-typed psychopathy and ASPD manifestations.

A substantial body of research on sex differences in psychopathy and ASPD has accumulated over the past few decades. Nevertheless, researchers have only recently begun to extend their work beyond examining sex differences in the base rates and mean symptom levels of these conditions. From a construct validational perspective, examining personality variables, psychopathological characteristics, and behavioral patterns across biological sex should enable researchers to further investigate hypotheses for these conditions’ etiology. Moreover, we recommend that researchers construct and test models of psychopathy and ASPD’s etiology by positing specific biological and psychosocial moderators of putative sex-differentiated expressions of psychopathy and ASPD. The testing of such models should help advance our understanding of the etiology of psychopathy and ASPD, as well as of their potentially diverse phenotypic manifestations.
References


