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A psychometric investigation of gender differences and common processes across Borderline and Antisocial Personality Disorders

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Abstract

The comorbidity between Borderline Personality Disorder (BPD) and Antisocial Personality Disorder (ASPD) is well-established, and the two disorders share many similarities. However, there are also differences across disorders: most notably, BPD is diagnosed more frequently in females and ASPD in males. We investigated if a) comorbidity between BPD and ASPD is attributable to two discrete disorders or the expression of common underlying processes, and b) if the model of comorbidity is true across sex. Using a clinical sample of 1400 drug users in residential substance abuse treatment, we tested three competing models to explore whether the comorbidity of ASPD and BPD should be represented by a single common factor, two correlated factors, or a bifactor structure involving a general and disorder-specific factors. Next, we tested whether our resulting model was meaningful by examining its relationship with criterion variables previously reported to be associated with BPD and ASPD. The bifactor model provided the best fit and was invariant across sex. Overall, the general factor of the bifactor model significantly accounted for a large percentage of the variance in criterion variables, whereas the BPD and AAB specific factors added little to the models. The association of the general and specific factor with all criterion variables was equal for males and females. Our results suggest common underlying vulnerability accounts for both the comorbidity between BPD and AAB (across sex), and this common vulnerability drives the association with other psychopathology and maladaptive behavior. This in turn has implications for diagnostic classification systems and treatment.

General scientific summary—This study found that, for both males and females, borderline and antisocial personality disorders show a large degree of overlap, and little uniqueness. The commonality between BPD and ASPD mainly accounted for associations with criterion variables. This suggests that BPD and ASPD show a large common core that accounts for their comorbidity.

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Keywords

borderline personality disorder; antisocial personality disorder; substance use disorders; comorbidity

The high comorbidity between borderline personality disorder (BPD) and antisocial personality disorder (ASPD) is well-established. Rates of comorbidity range between 5.6% and 27% in community samples, and are as high as 57% in clinical and forensic settings (Black et al., 2007; Black, Gunter, Loveless, Allen, & Sieleni, 2010; Blackburn, Logan, Donnelly, & Renwick, 2003; Grant et al., 2008; McGlashan et al., 2000; Tadic et al., 2009). Unsurprisingly, multiple studies indicate that BPD and ASPD share etiology and developmental course. The two disorders have similar prevalence rates in both community (~1–2% for BPD, ~1–3% for ASPD) (Torrens, Gilchrist, Domingo-Salyany, & PsyCoBarcelona, 2011) and clinical (between 10–30% for both BPD and ASPD) samples (Black et al., 2007; Kessler et al., 1994; Lenzenweger, Lane, Loranger, & Kessler, 2007; Trull, Jahng, Tomko, Wood, & Sher, 2010). A meta-analysis of seven family studies of BPD found that the median prevalence of ASPD in relatives of BPD probands was 7%—twice that in the general population (White, Gunderson, Zanarini, & Hudson, 2003). Results from quantitative genetic studies indicate that BPD and ASPD symptoms have common genetic and nonshared environmental influences (Hunt, Bornovalova, & Patrick, 2015; Kendler et al., 2011) even after accounting for other cluster B personality disorders (Torgersen et al., 2008). Likewise, BPD and ASPD show similar temperamental vulnerabilities of emotion dysregulation, weak inhibitory control, and social cognition deficits (Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009; Beeney et al., 2015; Hicks, Vaidyanathan, & Patrick, 2010; Scott et al., 2013) and are associated with similar environmental precursors, including but not limited to childhood trauma, maladaptive parenting, and stressful life events (Eitle & Turner, 2002; Hicks et al., 2010; Stepp, Olino, Klein, Seeley, & Lewinsohn, 2013; Zanarini et al., 1997). While many of these risk factors also appear in other psychopathology, certain aspects of social cognition (i.e., ability to predict someone else's behavior based on their belief state, mentalization) are unique to BPD and ASPD (Beeney et al., 2015). Finally, the two disorders have similar developmental course: a steep increase in adolescence, a decrease or plateauing in young adulthood, and a "burning out" by late middle age (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2006; Bornovalova, Hicks, Iacono, & McGue, 2009).

Striking as these similarities may be, it is important to highlight several important differences between the two disorders. For instance, BPD gives considerably more emphasis to the construct of affective instability and/or emotion dysregulation, identity diffusion, and difficulties in interpersonal relationships (APA, 2013). In a cross-sectional multi-trait, multi-method study, Scott and colleagues (2013) reported that emotion dysregulation and preoccupied attachment were robustly related to both BPD and ASPD; however, both emotion dysregulation and preoccupied attachment shared significantly more variance with BPD than APSD. Likewise, disinhibition – although common to both disorders – frequently takes different forms. A recent study modeled the relationships between different aspects of impulsivity (namely, urgency, perseverance, premeditation, and sensation-seeking) with

BPD and ASPD simultaneously. Urgency and lack of perseverance were uniquely related to BPD, whereas lack of premeditation and excitement seeking were uniquely related to ASPD (DeShong & Kurtz, 2013; see also Fossati, Krueger, Markon, Borroni, & Maffei, 2013). Most relevant to the current study, there are marked sex differences in the severity and prevalence of the disorders. In clinical samples, approximately 2/3 to 3/4 of all BPD diagnoses are in females (Johnson et al., 2003). In contrast, ASPD is three times more common in males (Alegria et al., 2013).

To date, there are at least two gaps in the literature on sex, BPD, and ASPD. First, do BPD and ASPD have a common underlying core (Beauchaine et al., 2009; Skodol & Bender, 2003)? Second, after accounting for the common variance between the two disorders, do sex differences (if any) stem from true differences in symptom severity or biased diagnostic indicators (Skodol & Bender, 2003)? In other words, is the relationship between the disorders the same for males and females?

The first question– that BPD and ASPD share core etiological vulnerabilities that are expressed differently for males and females – is not a new one. Most recently, a very similar idea is the foundation for DSM-5 Section III (Models and Measures for Further Study) framework for describing personality disorders. This alternative model of personality pathology should, in principle, account for both commonalities and unique characteristics of each disorder (APA, 2013). Likewise, the BPD-ASPD-sex question has been explored numerous times in conceptual (Beauchaine et al., 2009; Paris, 1997; Paris, Chenard-Poirier, & Biskin, 2013; Skodol & Bender, 2003) and, to a lesser degree, in empirical (Hunt, Bornovalova, Kimonis, Lilienfeld, & Poythress, 2015; Johnson et al., 2003; Sprague, Javdani, Sadeh, Newman, & Verona, 2012; Verona, Sprague, & Javdani, 2012; Zlotnick, Rothschild, & Zimmerman, 2002) literature. However, solid empirical tests of this question are scarce – in part, because it is surprisingly difficult to test if two separate constructs are actually the same construct. One way is to simultaneously model the relationship between putative vulnerabilities, BPD, and ASPD (across sex). If BPD and ASPD represent the same underlying psychopathology, then the relationships between putative vulnerabilities and each disorder should be equal. Taking it a step further, if the general vulnerability is colored differently by sex, then the relationships between putative vulnerabilities and ASPD should be stronger among males, while the same relationships with BPD should be stronger among females. Although the Scott et al (2013) study we described above did not examine the relationships by sex, it is one of the few published studies that simultaneously modeled the relationships between putative vulnerabilities, BPD, and ASPD.

Another way of approaching the BPD-ASPD-sex question is to fit different factor analysis models to the covariances of BPD and ASPD indicators and test the invariance of the best fitting model across sex groups. The benefit of this approach is its ability to test competing hypotheses. If BPD and ASPD reflect the same underlying form of psychopathology, then all BPD and ASPD diagnostic indicators should load on a single common factor. In contrast, if the disorders are truly separable, then a correlated two-factor model should fit best. Finally, if BPD and ASPD have distinguishing characteristics but also a common core, then a bifactor model involving a general psychopathology factor and a factor specific to each disorder will fit best (Reise, 2012). A benefit of this model is the ability to determine the

proportions of variance associated with common and specific factors. To our knowledge, these competing hypotheses have not been empirically examined.

For the most part, work addressing the second question – that of potential measurement bias versus true sex differences – is also scant. Studies relying primarily on content review found little evidence of sex bias (Anderson, Sankis, & Widiger, 2001; Morey, Warner, & Boggs, 2002; Sprock, Crosby, & Nielsen, 2001). More recent work has used measurement invariance (MI) models which can separate observed sex differences into a component reflecting true differences and a component reflecting bias that results from interpreting diagnostic indicators differently for males and females. This work focuses almost exclusively on BPD, and suggests that few items show evidence of bias (Aggen, Neale, Roysamb, Reichborn-Kjennerud, & Kendler, 2009; Hoertel, Peyre, Wall, Limosin, & Blanco, 2014; Jane, Oltmanns, South, & Turkheimer, 2007; Sharp et al., 2014). Item by item invariance analysis in these studies provides interesting information; however, without information about the overall model fit, it is difficult to tell if the latent mean sex differences are meaningful. As ASPD has been modeled almost exclusively in the context of other externalizing psychopathology (see Jane et al, 2007 for exception), tests of this disorder alone are limited. More importantly, MI testing of BPD and ASPD does not take into account the high comorbidity between the two disorders. In sum, ambiguities in the MI BPD literature and the dearth of studies involving ASPD indicators suggests that more research is needed to determine whether sex differences in prevalence rates are artifactual or substantive.

Current Study

In summary, research is needed to explore the covariance structure of BPD and ASPD, as well as measurement invariance of this structure across sex. We chose a large clinical sample (N=1400 substance users in residential treatment) to model these relationships. Notably, both clinical and epidemiological samples have their own strengths and weaknesses. Epidemiological samples make use of systematic sampling techniques and easily generalize to the population “at large”; however, the relatively low base rates of BPD and ASPD (~1–4%) (Hamdi & Iacono, 2014; Trull et al., 2010) and resulting restricted variance limit the type of models that can be fit. In clinical and forensic samples – despite their limited generalizability – the prevalence and comorbidity of BPD and ASPD is considerably higher (Black et al., 2007; Black et al., 2010; Blackburn et al., 2003; Grant et al., 2008; Zanarini et al., 1998), and the disorders are particularly relevant to maladaptive outcomes (Kokkevi, Stefanis, Anastasopoulou, & Kostogianni, 1998; Torrens et al., 2011). Finally, we examined the predictive utility of the BPD and AAB factor scores with respect to other clinically relevant outcomes: major depressive disorder, anxiety disorders, substance use disorders, history of psychiatric and drug treatment, lifetime length of incarceration, and age of onset for drugs and alcohol (Trull et al., 2010; Zanarini, Frankenburg, Hennen, Reich, & Silk, 2004). Many of these criterion variables are associated with both BPD and AAB; however, others are unique to the specific disorders. For example, age of drug and alcohol onset as well as history of incarceration/criminal behavior are more closely linked with ASPD, whereas lifetime psychiatric treatments are more typically associated with BPD (Cloninger, Sigvardsson, & Bohman, 1996; Cohen, Chen, Crawford, Brook, & Gordon, 2007; Franken

& Hendriks, 2000; Johnson, Cloninger, Roache, Bordnick, & Ruiz, 2000; Zanarini, Frankenburg, Khera, & Bleichmar, 2001).

Method

Patients (N = 1400; 481 = female; 919 = male) were sequential admissions into an inpatient substance use treatment facility. The mean age of the sample was 42.91 (SD = 10.64). The majority of the sample was court-mandated to treatment (67.5%). The sample was primarily African American (88%). 39.1% of the sample reported less than a high school education; 50.6% reported a high school diploma or equivalent; and 10.3% reported some college or above. Participants were required to submit a negative urine drug screen prior to admission; those with positive drug screens completed medically assisted detoxification before admission to the facility. Drug-testing occurred on a weekly basis; use was grounds for immediate discharge.

Recruitment and consent

Intake assessments were conducted by doctoral level graduate students/senior research staff upon patient's first week in treatment. The assessments served two purposes: (1) to provide diagnostic information to treatment staff at the center, and (2) to gather data for the current study. Patients were invited to participate in research following the intake assessment and were provided details regarding how information collected during the assessment would be used. Data for the current study includes only cases where informed consent was obtained (< 5% of patients declined to provide informed consent). The study protocol was reviewed and approved by the Institutional Review Board.

Measures

Assessment of Borderline and Antisocial Personality Disorders—Information regarding ASPD was gathered using the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders IV, SCID-IV (First, 2002). The Diagnostic Interview for Personality Disorders (DIPD) was used to assess Borderline Personality Disorder (BPD), as it has been argued to be a more comprehensive measure of BPD than the SCID-IV (Zanarini, Frankenburg, Chauncey, & Gunderson, 1987). All BPD and ASPD items were administered to all patients. Notably, although the criteria for DSM-IV ASPD includes a diagnosis of conduct disorder (CD) prior to age 15, our analyses focused specifically on the adult criteria of ASPD (adult antisocial behavior, AAB) because CD and AAB are traditionally treated as separate scales. In addition, including CD items would have developmental implications that are inconsistent with the diagnostic description of BPD, and that could obscure our interpretations of factor structure. For both AAB and BPD, the indicator-level data were dichotomous responses provided by clinicians to indicate whether a patient exhibited or did not exhibit a symptom.

Diagnostic interviewers were extensively trained and comprehensively supervised to ensure the accuracy of diagnoses. Training included viewing the full video protocol for the SCID-IV, conducting two mock interviews using the SCID-IV and the DIPD, observing two full interviews by experienced interviewers, conducting a final certification practice interview,

being observed while conducting two real interviews at the treatment center (with the SCID-IV and the DIPD), and participating in weekly supervision led by a clinical psychologist. When disagreements occurred, discussion continued until consensus was reached and changes were made (Lechner et al., 2013).

Criterion Variables—Information regarding other comorbid psychopathology was collected via the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders IV (SCID-IV; First et al., 1995), including major depressive disorder, panic disorder, social phobia, generalized anxiety disorder, and various substance dependence (see below). All disorders were assessed for current and ever/past status, with the exception of generalized anxiety disorder which was assessed for the last six months only. Diagnostic status, rather than symptom counts was used for all psychopathology, as all interviews (with the exception of personality disorders) were administered with “skip-outs”. All psychopathology was coded to be lifetime (i.e., disorder endorsed either currently or ever/past) absent or present. All anxiety disorders were combined into a single dichotomous “lifetime anxiety disorder” variable, as we did not expect different patterns of results across individual disorders. Interviewers attended to the timeline of substance dependence, and psychopathology diagnoses were made only when symptoms could not be tied directly to acute substance intoxication or the effects of withdrawal from a substance. For SUDs, we only assessed substance dependence (not abuse, given the severity of the sample), and we report here on alcohol, cannabis, opioid, hallucinogen/PCP, and crack/cocaine dependence, as these were the most frequently used substances in this sample. As with non-substance psychopathology, substance use disorders were assessed dichotomously and coded to be lifetime absent or present. All drug categories were left separate (as opposed to combining into larger “drug” and “alcohol” categories) given the rich literature surrounding personality, personality disorders, and drug choice (Bornovalova, Daughters, Hernandez, Richards, & Lejuez, 2005; Conway, Kane, Ball, Poling, & Rounsaville, 2003; Hopwood, Baker, & Morey, 2008). Lifetime rates of psychopathology across sex are reported in Table 1.

Additional criterion variables were collected via a self-report treatment, substance use, and legal history questionnaire (Banducci et al., 2013). Specifically, we collected data on: age of onset for alcohol and other drugs; lifetime number of days spent in jail; lifetime history of drug or alcohol treatment, and lifetime history of psychiatric treatment. Lifetime number of days in jail was coded as: none (0); 30 days or less (1); 31–90 days (2); 91 days–one year (3); between 1–5 years (4); greater than 5 years (5). Lifetime drug or alcohol and lifetime psychiatric treatment were coded dichotomously (absent/present).

Statistical Analyses

All factor analysis models were estimated using a robust weighted-least-squares estimator (WLSMV) in Mplus 7.11 (Muthén & Muthén, 1998–2015). Although item-level MI analyses for BPD and AAB is not the main focus of this paper, the methods and results for these analyses may still be informative for many researchers and are provided in the supplemental materials.

Modeling the Comorbidity between BPD and AAB—To answer the research questions regarding the covariance of BPD and AAB and invariance of factor structure across males and females, we fit three increasingly complex confirmatory factor analysis models for the combined sample: (1) a unidimensional model, (2) a correlated two-factor model, and (3) a bifactor model. The goodness of fit of each model was assessed using the comparative fit index (CFI) and the root-mean-square error of approximation (RMSEA). Values ≥ .95 and values ≤ .08 respectively for CFI and RMSEA indicate reasonable model-data fit (Hu & Bentler, 1999). For these nested model comparisons, we used the likelihood ratio test (i.e., $\Delta\chi^2/\Delta df$). Because $\Delta\chi^2$ cannot be directly employed for nested models when the WLSMV estimator is used, the $\Delta\chi^2$ was computed using the DIFFTEST procedure in Mplus.

MI testing for AAB and BPD factor structure—After identifying the best-fitting model based on the combined sample, we tested for MI across sex groups. Females were defined as the reference group, and males were defined as the focal group. We followed the procedure for MI testing with binary data recommended by Muthén and Muthén (1998–2015) which involves a comparison of configural and scalar invariance models. To allow every item to be compared across reference and focal groups, we identified the scale of measurement by constraining the variance of the general and group-specific factors to 1 in both groups. In the configural model, we fixed the residuals to 1 and the latent means to 0 in both groups for identification, while factor loadings and thresholds were free to vary. In the scalar model, we constrained the loadings and thresholds to be equal across groups while freely estimating the focal group's residuals and latent means. We compared the goodness of fit statistics for the configural and scalar models using three criteria: likelihood ratio test $\Delta\chi^2/\Delta df$), changes in CFI (ΔCFI) and changes in RMSEA (ΔRMSEA). In accordance with interpretive guidelines (Chen, 2007), ΔCFI ≥ .01 and ΔRMSEA ≥ .015 indicated a significant decrement in fit.

Unique contributions of general and specific factors in predicting criterion variables—As is seen in the results below, we identified the bifactor model as the best-fitting model; this model showed MI across sex. Consequently, we conducted a series of structural equation models (SEMs) to test whether the BPD and AAB specific factors would account for variance in criterion variables beyond the general factor. Each criterion was modeled separately. For each criterion variable, we fit two models. The full model estimated direct paths from both the general and specific factors on the criterion variable. In the constrained model, we fixed the path coefficients of the specific factors on the criterion variable to zero. Next, we evaluated the significance of the BPD and AAB specific factors (i.e., the decision to keep the full versus the constrained model). The formal test was done via nested model comparisons (full versus constrained) based on $\Delta\chi^2/\Delta df$. However, we also required that the change in variance accounted for (ΔR^2) should be meaningful. We defined "meaningful" by an increase in variance accounted for of at least 2.25% (Hunsley &Meyer, 2003). Thus, to proceed beyond the constrained model, the full model should demonstrate a significant $\Delta\chi^2/\Delta df$ and a $\Delta R^2 \geq .0225$.

Finally, we conducted a multi-group SEM to examine whether the structural relationships of the general and specific factors with the criterion variables were significantly different across sex. Statistical significance was evaluated using Wald tests. If the specific BPD and AAB factors failed to provide evidence for meaningful incremental contribution, sex differences were only examined for the general factor. Given the number of tests, significance was set at .01 for all SEMs.

Results

Diagnostic and comorbidity rates

The percentages of males and females exhibiting each BPD and AAB symptom are reported in Table 1. Diagnostic and comorbidity rates were as follows. Overall, 14.1% of participants (24.4% females and 10.3% males) met criteria for BPD. Likewise, 36.8% of the participants (32.9% females and 38.8% males) met criteria for AAB. Of the entire sample, 9.6% met criteria for both BPD and AAB (13.5% females; 7.6% males). Rates and mean levels of psychopathology and maladaptive behavior of interest are also reported in Table 1.

MI testing for AAB and BPD factor structure

Model fit indices for the unidimensional, correlated two-factor, and bifactor models are presented in Table 2 for the male, female, and combined samples. In each case, the bifactor model provided the best fit. All 16 AAB and BPD indicators loaded on the general factor in addition to their respective specific factors[1,2].

Table 3 shows the standardized parameter estimates for the bifactor model for males and females. Notably, after controlling for the general factor, the group factor loadings were lower in both groups. As seen in Table 4, the general factor accounted for nearly 66% of the common variance in females, and 60% in males. The specific factors – BPD and AAB – only accounted for 14% and 20% (respectively) of the common variance in females and for 23% and 18% of the common variance in males.

Additional statistics, coefficient omega hierarchical (Ω_h) and omega-subscale (Ω_s), were calculated for both sexes (Reise, 2012; Zinbarg, Revelle, Yovel, & Li, 2005)[3]. Ω_h estimates the proportion of total variance attributable to the general factor, whereas the Ω_s represents the proportion of variance accounted for by the specific factor after controlling for the general factor. Ω_h were .77 and .71 for females and males (respectively), indicating that 77% and 71% of the variance of the sum scores across the sixteen diagnostic criteria could be

[1] In all bifactor models, the general and specific factors were modeled as orthogonal.

[2] It is important to note that the loading of one of the BPD criteria (i.e., “impulsivity”) on the corresponding specific factor was fixed to zero for solving a convergence problem. To ensure that this constraint did not substantially alter our results, we repeated the analyses without the BPD impulsivity item. The factor structure and subsequent interpretations did not change.

[3] Reise (2012) illustrates how to compute variance accounted for by general and specific factors. In this case, $\Omega_h = \frac{(\sum \lambda_{iG})^2}{(\sum \lambda_{iG})^2 + (\sum \lambda_{iB})^2 + (\sum \lambda_{iA})^2 + \sum \theta_i^2}$, where G, B, and A represent the general and specific factors, and $\sum \theta_i^2$ represents the sum of the item error variances (uniquenesses) over all items. For specific factor AAB, $\Omega_{s\,(A)} = \frac{(\sum \lambda_{iA})^2}{(\sum \lambda_{iG})^2 + (\sum \lambda_{iB})^2 + (\sum \lambda_{iA})^2 + \sum \theta_i^2}$ but, in this case, $\sum \theta_i^2$ represents the sum of the error variances for the items measuring AAB (a similar calculation is used for BPD items).

attributed to variance on the general factor. Ω_s were .19 and .43 for BPD and AAB indicators, respectively, among females, and.30 and .38, respectively, among males[4].

Finally, after determining that the bifactor model provided the best fit in the combined sample, we tested for MI across sex groups. The results were as follows: configural invariance, χ^2 (df = 178) = 286.82, CFI = .98, RMSEA = .03; scalar invariance, χ^2 (df = 206) = 290.23, CFI =.99, RMSEA =.02. All three criteria supported scalar MI ($\Delta\chi^2/\Delta df$ = 27.35 (28), p >.05; ΔCFI =.00 and ΔRMSEA=.01), indicating that the comorbidity between AAB and BPD reflects equivalent processes for males and females. Also, females had higher general (.34 units) and BPD-specific (.29 units) factor means, and males had a higher mean on the AAB-specific factor (.52 units)[5].

Unique contributions of general and specific factors in predicting criterion variables

Table 4 presents fit from a series of nested SEM models examining the unique contribution of BPD and AAB specific factors to criterion variables. First, the fit of all constrained models (criterion on general factor only; criterion on BPD and AAB fixed at zero) met benchmark CFI and RMSEA criteria for overall good model fit. Each constrained model revealed that the general factor significantly predicted all criterion variables. Variance accounted for by the general factor varied a great deal across criterion variables. The largest effects were found for (in descending order): lifetime major depressive disorder (R^2=.40), lifetime anxiety disorder (R^2=.26), lifetime history of psychiatric hospitalization (R^2=.18), and lifetime alcohol dependence (R^2=.14). The variance accounted for by the general factor in age of onset, heavy substance use, and various dependence diagnoses was generally small to moderate (R^2=.02 for heroin dependence through .10 for onset of drug use). The smallest effect size was for total time spent in jail: the general factor only accounted for 1% of the variance in this criterion.

Next, the fit of all full models (criterion on general as well as BPD and AAB specific factors) also met benchmark model fit criteria, demonstrating adequate fit to the data. When comparing the full models against the constrained models, however, the full model did not fit better (as indicated by the $\Delta\chi^2/\Delta df$) in three cases (lifetime crack/cocaine, cannabis dependence; and hallucinogenic dependence). Two additional variables (lifetime heroin and alcohol dependence) failed to pass the Hunsley & Meyer (2003) benchmark criteria of meaningful variance accounted for (Hunsley & Meyer, 2003) (ΔR^2 > = .0225). In sum, for

[4]Although we focus on describing the relationship with adult AAB symptoms, we also examined whether the model would hold after accounting for childhood symptoms of conduct disorder. Results indicated that, using 22 indicators of ASPD (15 CD; 7 AAB) and 9 indicators of BPD, consistent with the main results, a bifactor model fit the best. The model fit was as follows: unidimensional model: χ^2 (df = 434) = 2811.92, CFI = .82, RMSEA = .06; correlated two-factor model, χ^2 (df = 433) = 1617.99, CFI = .91, correlated three-factor model, χ^2 (df = 431) = 1287.389, CFI = .93, RMSEA = .04; bifactor with two specific factors, ASPD and BPD, χ^2 (df = 404) = 1183.28, CFI = .94, RMSEA = .04; bifactor with three specific factors, CD, AAB, and BPD, χ^2 (df = 404) = 1237.00, CFI = .94, RMSEA = .04. We also conducted MI tests across sex. As in the main analyses, we tested for MI across sex groups: configural invariance, χ^2 (df = 808) = 1405.71, CFI = .95, RMSEA = .03; scalar invariance, χ^2 (df = 866) = 1464.48, CFI =.95, RMSEA = .03. Although likelihood ratio test was significant ($\Delta\chi^2/\Delta df$ = 123.76 (58), p <.05), ΔCFI =.00 and ΔRMSEA=.00 were not.

[5]We examined a more exploratory (and thereby less restrictive) version of the bifactor model as well: the bifactor ESEM. As with CFA, the bifactor ESEM fit well [χ^2 (df = 75) = 149.21, CFI = .99], the general factor accounted for >60% of the common variance among the indicators, and the conclusions about the factor structure did not change. We also explored MI of the ESEM model. There was evidence of scalar invariance as indicated by all three criteria ($\Delta\chi$2 (Δdf), ΔRMSEA, ΔCFI) [configural: (χ^2 (df = 150) = 228.65, CFI = .99, RMSEA = .03; scalar: χ^2 (df = 186) = 241.56, CFI = .99, RMSEA = .02;$\Delta\chi^2/\Delta df$ = 33.78 (36), p >.05; ΔCFI =.00; ΔRMSEA=.01. The latent mean gender differences were nearly the same, with women scoring higher on the general factor, and men scoring higher on the AAB specific factor.

all drug and alcohol use disorders, the BPD and AAB specific factors failed to add incremental variance above the general factor, suggesting that most of the effect is accounted for by the common variance between BPD and AAB.

On the other hand, for three criterion variables (namely, total time spent in jail; lifetime major depressive disorder; and lifetime history of psychiatric treatment), criterion variables, the specific factors accounted for a large percentage of incremental variance (ΔR^2= .12–.17). Unsurprisingly, the effect on total time spent in jail was driven by the AAB specific factor, whereas the effect on lifetime major depressive disorder and lifetime history of psychiatric treatment was driven by the BPD specific factor.

For the remaining four criterion variables, the specific factors accounted for a small to moderate proportion of the variance (ΔR^2=.03–.05). The association with age of onset of drug and alcohol use was driven by the AAB specific factor (although the BPD factor also predicted age of onset of drug and alcohol use to a smaller degree). The association with lifetime anxiety disorder was driven by the BPD specific factor. Both BPD and AAB contributed to the with lifetime history of drug and alcohol treatment.

Multi-group bifactor SEM models to test sex differences in structural relationships

Table 5 presents results from multi-group SEM models testing sex differences in the overall effects reported above. Wald tests indicated the association of the general factor with each of 12 criterion variables was equal for males and females, suggesting that the relationship between the general and specific factors with criterion variables is not sex-specific.

Discussion

The current study investigated if BPD and AAB have a substantial common underpinning, in addition to characteristics that uniquely differentiate the two disorders. We also examined the relative importance of the common and disorder specific variance by examining associations with other relevant clinical constructs. Finally, we examined whether the covariance structure of BPD and AAB shows evidence of sex bias by investigating MI of the models across sex, and tested whether the relationships between general and disorder-specific factors with clinical criterion variables differ across males and females. We found that a bifactor model involving a fairly strong general psychopathology factor and two disorder-specific factors provided the best description of the data. This is consistent with previous findings indicating that BPD and AAB show phenotypic, genetic, and environmental overlap, above and beyond other psychopathology (Torgersen et al., 2008). This suggests that the general factor may be a more reliable index of sex differences than either latent means of BPD or AAB. Thus, it may be more parsimonious to combine BPD and AAB into a single syndrome in diagnostic classification systems as well as studies of etiology and treatment. Likely, this factor heavily represents impulsive and disinhibited tendencies – and to a lesser degree, interpersonal dysfunction and emotion dysregulation. While this makes our general factor unlike other studies that generally extract a large factor of negative affectivity, emotional dysregulation, or interpersonal dysfunction (Hopwood et al., 2011; Jahng et al., 2011; Livesley, 2011; Pincus & Wiggins, 1990), it is reflective of both the common item content between the disorders and the empirical findings that BPD and

AAB both load on the externalizing spectrum (Eaton et al., 2011; James & Taylor, 2008; Kendler et al., 2011).

Although the variances accounted for by the disorder-specific factors were small in comparison to the general factor, it may be still worthwhile to ask what those variances represent. For BPD, across sex, the items that most strongly loaded on the disorder-specific factor represent feelings of emptiness and cognitive disturbance (stress-related paranoia/dissociation). For AAB, the remaining variance was accounted by items that represent lack of socialization/lack of conformity to rules (Hicks & McGue, 2014). It may be possible to think of these criteria as ones that "color" the expression of BPD and AAB, and possibly account for sex differences in the respective disorders.

Next, we tested the measurement invariance of this bifactor model. Notably, previous work examining MI has a) generally focused on BPD; b) took little consideration of AAB or its covariance with BPD. Although previous studies used slightly different MI methods than ours (individual-item invariance versus global model invariance), we appear to have produced findings consistent with epidemiological samples (Aggen et al., 2009; Hoertel et al., 2014; Jane et al., 2007; Sharp et al., 2014). Similar (and a very limited number, see supplemental materials) items produced item-level non-invariance. Overall, our bifactor model was invariant across sex, suggesting little evidence of bias in our data. The females demonstrated a higher mean on the general and BPD factor, whereas the males demonstrated a significantly higher AAB factor. While the sex patterns in BPD and AAB specific factors are expected, it is difficult to make much of the mean differences in the general factor at this time. The female > male mean difference may be a local phenomenon due to sample characteristics or number and type of BPD and AAB items in the model

The incremental validity analyses lined up with the rest of our results. The general factor of the bifactor model is a pivotal and sole predictor of five criterion variables: namely, all dependence diagnoses. For all alcohol and drug dependence, the general factor significantly accounted for a large percentage of the variance, whereas the BPD and AAB specific factors added little to the models. Our results strong support to the notion that a common underlying vulnerability accounts for both the comorbidity between BPD and AAB, and this common vulnerability drives the association with all substance use problems (but not necessarily age of onset of drug and alcohol use, or drug and alcohol treatment). Thus, despite the traditional clinical, diagnostic, and statistical distinction between the two disorders, the general factor is much more important for prediction of comorbidity and treatment planning of at least, drug and alcohol treatment (see below for implications of these results for interpretation of the general factor).

In several cases, however, the specific factors produced incremental prediction to the variance that could not be ignored. The BPD specific factor incrementally predicted lifetime major depressive disorder and lifetime history of psychiatric treatment, whereas the AAB factor predicted lifetime number of days in jail. While these results intuitively make sense and are consistent with previous work (Zanarini et al., 2001), it is unclear how a factor representing emptiness and cognitive distortions interacts with the liabilities representing the general factor to produce depression and psychiatric treatment history. Likewise, the

association between AAB specific factor and incarceration is not surprising. The highest factor loading on the AAB specific factor is an indicator reading “failure to conform to social norms with respect to lawful behaviors as indicated by repeatedly performing acts that are grounds for arrest” (APA, 1994, 2013); the association of this factor with incarceration may simply reflect the same construct. More notable are the associations of the AAB specific factor with drug and alcohol age of initiation; early age of alcohol initiation, for instance, has been long thought to be a marker for an alcoholism subtype marked by a high genetic loading and high criminal behavior (Cloninger et al., 1996; Johnson et al., 2000). It is possible that we are tapping into this latent dimension with the specific AAB factor.

Implications, Limitations, and Future Directions

Our results should be considered in the broader scope of personality disorder and normal personality literature. As we note in the introduction, both our questions and our results are consistent with current conceptualization of personality disorders which propose both general impairments in personality (common to all PDs), and specific manifestations of each phenotype which are captured by maladaptive trait profiles. (APA, 2013). Likewise, our results are consistent with recent reports of both normal personality (Biderman, 2013; Biderman, Nguyen, Cunningham, Chen, & Watson, 2013) and personality (and non-personality) pathology (Hengartner, Ajdacic-Gross, Rodgers, Mueller, & Roessler, 2014; Hopwood et al., 2011; Jahng et al., 2011; Scott et al., 2013; Sharp et al., 2014; Wolf, Miller, & Brown, 2011). To illustrate, a recent study by Sharp and others (2015) reported that a general underlying factor accounted for common variance across personality disorders, and six specific factors accounted unique variance in personality disorders. What is also notable is Sharp et al reported that there was no specific “factor” of BPD; rather, BPD symptoms loaded appreciably onto the general underlying factor. Other researchers have replicated this phenomenon: while the specific factors frequently differ, a general factor – usually representing interpersonal dysfunction – accounts for much of the covariance across personality pathology symptoms and traits (Hengartner et al., 2014; Jahng et al., 2011; Wolf et al., 2011).

What is not clear, at this point, is where our bifactor model of BPD-AAB falls within these frameworks. In other words, is there something unique to BPD-AAB comorbidity that is not accounted for by a common liability to all personality (or non-personality) disorders? Or, like Sharp et al, does BPD generally represent a general liability to personality pathology, and our model reflects the tendency of BPD to sink into a general factor? The first possibility is not implausible. Jahng and colleagues (2011) reported that retaining a specific factor characterized by cluster B comorbidity –representing information about behavioral disinhibition/impulsivity – significantly improved model fit over the model that included just the general factor representing interpersonal dysfunction. All other specific personality disorder factors (i.e., Cluster A and C) did not improve model fit. More importantly, the specific cluster B factor accounted for considerably more variance in alcohol, drug, and nicotine dependence than the interpersonal factor. This is highly consistent with our own results, indicating that our general factor was the sole predictor of all drug and alcohol use disorders. Unsurprisingly, we also interpret our general factor to contain information about behavioral disinhibition. The notion that a common core of behavioral disinhibition largely

accounts for the comorbidity between BPD, AAB, and substance use disorders is consistent with several twin (Distel et al., 2012; James & Taylor, 2008; Kendler et al., 2011) and neuroimaging (Sauder et al., 2016; Beck et al., 2009; Jentsch & Taylor, 1999) studies.

Nevertheless, our study is not equipped to distinguish between these two alternative hypotheses. A clear next step is to replicate the current study (using a multi-method, multi-sample approach, see below). This type of study would a) model whether there is a specific factor BPD-AAB after accounting for the covariance across all personality disorders (i.e., the interpersonal dysfunction factor) and the disorder-specific variance; b) investigate if this BPD-AAB factor is clinically meaningful, i.e., shows incremental criterion validity. In this context, it is also possible to test if this BPD-AAB specific factor simply picks up externalizing psychopathology, or some other characteristic unique to the two disorders.

Several limitations of the current paper set the stage for follow-up studies. First, our study was cross-sectional and utilized an adult sample, and therefore conclusions cannot be advanced regarding development or causality. Thus longitudinal studies examining the covariance structure of BPD and AAB are needed. Likewise, future work should examine the incremental predictive utility of the general and specific factors in predicting developmental course and response to treatment. Second, the symptoms of BPD and AAB were entirely clinician-rated. This is a non-trivial point: the clinician-rated, diagnostic indicators have, in the past, produced different sex patterns than the psychometrically-validated BPD and AAB instruments– and the clinical and self-report instruments have been shown to contribute unique information to the constructs (DeShong & Kurtz, 2013; Hopwood et al., 2008; Trull, 2001). Related to this point, the lack of trait-based instruments such as the Personality Inventory for DSM-5 is a significant limitation, as trait-based scales that are specifically designed to account for comorbidity between personality disorders may produce a different factor structure (Krueger et al, 2012). Thus, both the sex difference and psychometric structure question should be investigated using a multi-method procedure that combines a) self-report measures of BPD and AAB, b) traits hypothesized to underlie them (Few et al., 2013); c) clinical/interview measures of the two constructs.

Next, it is possible that females in a residential treatment facility had a higher “threshold” of severity than males. On the other hand, this type of ascertainment difference would have produced measurement noninvariance; yet, the model was invariant. More importantly, there is a very real possibility that the participants were selected (court-mandated into treatment) based on the behavior that was one of the primary variables of interest in our study (adult antisocial behavior). Because the resulting participants would be systematically different from the rest of the population, a clear next step is to rule out the possibility of measurement noninvariance of the current model across clinical and epidemiological samples. Third, the current set of results should be replicated in a representative epidemiological sample, as previous work shows that community samples show a very different pattern of sex differences (and lack thereof), and possibly different patterns of MI and patterns of comorbidity (Bornovalova, Hicks, Patrick, Iacono, & McGue, 2011). Finally, the current analyses should be replicated in a genetically informed (e.g., twin) sample. This in turn would allow researchers to examine whether the factor common to BPD and AAB also

reflects common etiology. Exploration of these questions will provide further insight into etiological influences on BPD and AAB as well as their comorbidity.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Percentage (%) of Participants Meeting Criteria for Each BPD and AAB Symptom by Gender

	Combined (N=1400)	Female (N=481)	Male (N=919)
BPD symptoms			
abandonment avoidance	12.0	14.9	20.3
unstable relationships	19.0	22.6	29.6
identity disturbance	9.4	10.8	13.5
impulsivity	36.2	38.6	43.3
inappropriate anger	17.3	20.1	25.5
self-harm/suicidality	12.6	15.7	21.6
affective instability	23.8	28.4	37.1
emptiness	20.6	22.3	25.6
stress-related paranoia/dissociation	10.6	12.9	17.4
AAB symptoms			
failure to conform to social norms	47.0	43.5	36.6
deceitfulness	28.9	27.5	24.8
consistent irresponsibility	35.4	33.9	31.0
impulsivity	32.1	31.1	29.2
irritability or aggressiveness	25.9	24.8	22.8
reckless disregard for safety of self or others	28.9	28.5	27.9
lack of remorse	11.9	10.8	8.8
Criterion Variables			
Major Depressive Disorder	50.5	59.3	45.9
Lifetime Anxiety Disorder	15.6	18.3	14.1
Lifetime Alcohol	42.4	43.9	43.2
Lifetime Cannabis	28.1	19.1	32.8
Lifetime Hallucinogens	23.9	24.7	23.5
Lifetime Heroin/Opiates	29.1	25.2	31.2
Lifetime Crack/Cocaine	55.5	60.9	52.7
Lifetime history of psychiatric treatment	57.9	65.9	53.6
Lifetime history of drug and alcohol treatment	69.4	68.0	70.1
Age of onset, alcohol[a]	15.25 (5.06)	16.51 (5.77)	14.62 (4.53)
Age of onset, drugs[a]	16.94 (5.89)	18.38 (6.39)	16.18 (5.46)
Total time spent in jail[a]	3.15 (1.74)	2.38 (1.62)	3.54 (1.67)

[a]For variables scored continuously (age of onset of drugs and alcohol, and total time spent in jail), means and standard deviations are presented.

Table 2

Model Fit Statistics

Model	χ^2	*df*	CFI	RMSEA	Comparison: $\Delta\chi^2(\Delta df)$
Total sample (N = 1,400)					
Model 1: Unidimensional model	937.41***	104	.88	.08	Model 1 vs. 2: 164.97*** (1)
Model 2: Correlated factor model	348.91***	103	.97	.04	Model 1 vs. 3: 460.75*** (15)
Model 3: Bifactor model	199.62***	89	.98	.03	Model 2 vs. 3: 111.02*** (14)
Female (N = 481)					
Model 1: Unidimensional model	379.25***	104	.91	.08	Model 1 vs. 2: 53.22*** (1)
Model 2: Correlated factor model	188.83***	103	.97	.04	Model 1 vs. 3: 156.80*** (15)
Model 3: Bifactor model	153.01***	89	.98	.04	Model 2 vs. 3: 36.55*** (14)
Male (N = 919)					
Model 1: Unidimensional model	544.16***	104	.88	.07	Model 1 vs. 2: 101.44*** (1)
Model 2: Correlated factor model	251.41***	103	.96	.04	Model 1 vs. 3: 270.90*** (15)
Model 3: Bifactor model	133.50***	89	.99	.02	Model 2 vs. 3: 86.04*** (14)

Note.

*** $p < .001$.

Table 3

Parameter Estimates of Bifactor Model

	Female (F)					Male (M)					|F-M|			
	τ	λ_G	λ_B	λ_A	υ^2	τ	λ_G	λ_B	λ_A	υ^2	$\Delta\tau$	$\Delta\lambda_G$	$\Delta\lambda_B$	$\Delta\lambda_A$
BPD symptoms														
abandonment avoidance	.83	.72	.28		.41	1.17	.63	.41		.44	.34	.09	.13	
unstable relationships	.54	.71	.29		.42	.89	.69	.26		.46	.35	.02	.03	
identity disturbance	1.11	.67	.39		.39	1.32	.50	.54		.46	.21	.18	.15	
Impulsivity	.18	.90	.00		.19	.35	.83	.00		.32	.18	.07	.00	
inappropriate anger	.66	.75	.27		.37	.95	.62	.25		.55	.28	.13	.02	
self-harm/suicidality	.79	.47	.43		.60	1.16	.51	.45		.54	.37	.04	.02	
affective instability	.34	.65	.43		.40	.71	.61	.46		.42	.38	.04	.03	
Emptiness	.66	.52	.60		.38	.83	.41	.69		.37	.16	.11	.09	
stress-related paranoia/dissociation	.94	.66	.40		.40	1.24	.47	.64		.37	.30	.19	.24	
AAB symptoms														
failure to conform to social norms	.35	.43		.67	.37	.09	.39		.59	.50	.26	.04		.08
deceitfulness	.69	.42		.48	.60	.56	.47		.39	.63	.13	.05		.09
consistent irresponsibility	.55	.65		.41	.41	.47	.55		.44	.51	.09	.10		.03
impulsivity	.50	.60		.43	.46	.38	.55		.43	.51	.11	.04		.01
irritability or aggressiveness	.75	.50		.51	.49	.65	.38		.57	.54	.10	.12		.06
reckless disregard for safety of self or others	.59	.55		.60	.34	.55	.54		.41	.54	.04	.01		.19
lack of remorse	1.36	.52		.45	.53	1.18	.57		.30	.59	.18	.05		.15
		Gen	BPD	AAB	υ^2		Gen	BPD	AAB	υ^2				
% Total Variance		.38	.08	.12	.42		.31	.12	.09	.48				
% Common Variance		.66	.14	.20			.60	.23	.18					
Ω_h		.77					.71							
Ω_s			.19	.43				.30	.38					

Note. τ is a threshold parameter, and λ_B, λ_A and λ_G represent the standardized factor loadings on BPD, ASPD, and general factor. υ^2 represents a uniqueness (residual) term (i.e., variance not accounted for by λ_B, λ_A and λ_G). $\Delta\tau$, $\Delta\lambda_A$, and $\Delta\lambda_B$ represent the absolute difference of each parameter estimate between female and male samples. All loadings are statistically significant. Ω_h and Ω_S represent coefficient omega hierarchical for general and specific group factors, respectively

Table 4

Bifactor SEM for each criterion variable: Model fit statistics and Structural Parameters

		Model fit statistics				Standardized estimate (*SE*)				
		χ^2 (*df*)	RMSEA	CFI	$\Delta\chi^2$ (Δdf)	General	BPD	AAB	R^2	ΔR^2
Age of onset: Alcohol	M_1	285.44***(104)	.04	.98	44.48***(2)	−.27^a (.03)			.07	.05
	M_2	211.10***(102)	.03	.99		−.11^a (.04)	−.13^a (.05)	−.30^a (.04)	.12	
Age of onset: Drugs	M_1	306.68***(104)	.04	.97	60.76***(2)	−.32^a (.03)			.10	.05
	M_2	217.83***(102)	.03	.98		−.17^a (.04)	−.07 (.06)	−.34^a (.05)	.15	
Total time spent in jail	M_1	395.86***(104)	.05	.96	80.69***(2)	.10^a (.04)			.01	.16
	M_2	223.15***(102)	.03	.98		−.03 (.04)	−.01 (.05)	.41^a (.04)	.17	
Major Depressive Disorder	M_1	332.32***(104)	.04	.97	71.10***(2)	.64^a (.03)			.40	.12
	M_2	205.58***(102)	.03	.99		.39^a (.04)	.61^a (.06)	.07 (.05)	.52	
Lifetime Anxiety Disorder	M_1	265.01***(104)	.03	.98	32.71***(2)	.51^a (.04)			.26	.03
	M_2	211.41***(102)	.03	.99		.36^a (.05)	.39^a (.06)	−.01 (.06)	.29	
Lifetime Alcohol Dependence	M_1	221.60***(104)	.03	.98	11.41***(2)	.37^a (.04)			.14	−.01
	M_2	205.96***(102)	.03	.99		.30^a (.05)	.21^a (.06)	.01 (.05)	.13	
Lifetime Cannabis Dependence	M_1	208.02***(104)	.03	.98	5.15(2)	.20^a (.04)			.04	.00
	M_2	203.95***(102)	.03	.99		.13^a (.05)	.08 (.07)	.14^a (.06)	.04	
Lifetime Hallucinogenic Dependence	M_1	223.15***(104)	.03	.98	6.01 (2)	.25^a (.05)			.07	.00
	M_2	217.59***(102)	.03	.98		.15^a (.05)	.17^a (.07)	.13^a (.06)	.07	
Lifetime Heroin/Opiates dependence	M_1	257.14***(104)	.03	.98	11.08**(2)	.13^a (.04)			.02	.02
	M_2	243.45***(102)	.03	.98		.07 (.05)	.02 (.07)	.18^a (.06)	.04	
Lifetime Crack/Cocaine	M_1	217.88***(104)	.03	.98	4.66 (2)	.30^a (.04)			.09	−.01
	M_2	215.67***(102)	.03	.98		.23^a (.05)	.08 (.06)	.13^a (.05)	.08	
Lifetime history of psychiatric treatment	M_1	324.02***(104)	.04	.97	63.13***(2)	.43^a (.04)			.18	.17
	M_2	206.26***(102)	.03	.99		.19^a (.05)	.55^a (.06)	.08 (.05)	.35	
Lifetime history of drug and alcohol treatment	M_1	235.78***(104)	.03	.98	12.69**(2)	.19^a (.05)			.04	.04

	Model fit statistics				Standardized estimate (*SE*)				
	χ^2 (*df*)	RMSEA	CFI	$\Delta\chi^2$ (Δdf)	General	BPD	AAB	R^2	ΔR^2
M_2	216.71*** (102)	.03	.98		.06 (.05)	.16[a] (.07)	.23[a] (.06)	.08	

Note. M_1 = constrained model; M_2 = Full model; General = general factor; BPD = BPD group specific factor; AAB = AAB group specific factor; R^2 = proportion of variance explained in the criterion by the model; ΔR^2 = Change in R^2; $\hat{I}^2$ = the proportion of variance uniquely counted for by the specific factors above and beyond the general factor.

*** $p < .001$

** $p < .01$.

[a] Denotes standardized estimates falling within the 95% CI.

Table 5

Multigroup Bifactor SEM for Examining Gender Difference in Structural Parameters

	Model fit indices				Standardized estimate (*SE*)			
	χ^2 (*df*)	RMSEA	CFI		General	BPD	AAB	Wald test χ^2 (*df*)
Age of onset: Alcohol	320.96 (232)***	.023	.987	Female	−.18[a] (.07)	−.16 (.08)	−.17[a] (.07)	2.55 (3)
				Male	−.12[a] (.04)	−.16[a] (.07)	−.33[a] (.06)	
Age of onset: Drugs	329.06 (232)***	.024	.986	Female	−.31[a] (.06)	−.02 (.09)	−.22[a] (.08)	7.17 (3)
				Male	−.13[a] (.05)	−.17[a] (.07)	−.35[a] (.06)	
Total time spent in jail	324.43 (232)***	.024	.987	Female	.10 (.06)	−.02 (.08)	.20[a] (.07)	7.59 (3)
				Male	−.03 (.05)	.08 (.07)	.47[a] (.06)	
Lifetime Major Depressive Disorder	312.92 (232)***	.022	.989	Female	.41[a] (.07)	.52[a] (.09)	.10 (.09)	1.92 (3)
				Male	.37[a] (.06)	.66[a] (.08)	.08 (.07)	
Lifetime Anxiety Disorder	319.14 (232)***	.023	.988	Female	.37[a] (.08)	.41[a] (.10)	.02 (.10)	.38 (3)
				Male	.35[a] (.07)	.39[a] (.09)	−.04 (.09)	
Lifetime Alcohol Dependence	337.08 (236)***	.025	.985	Female	.43[a] (.06)			1.05 (1)
				Male	.34[a] (.05)			
Lifetime Cannabis Dependence	318.22 (236)***	.022	.988	Female	.28[a] (.08)			.77 (1)
				Male	.20[a] (.05)			
Lifetime Hallucinogens Dependence	337.49 (236)***	.025	.985	Female	.19[a] (.08)			1.31 (1)
				Male	.30[a] (.06)			
Lifetime Heroin/Opiates Dependence	355.99 (236)***	.027	.983	Female	.03 (.07)			3.87 (1)
				Male	.21[a] (.05)			
Lifetime Crack/Cocaine Dependence	344.47 (236)***	.026	.984	Female	.32[a] (.07)			.28 (1)
				Male	.28[a] (.05)			
Lifetime history of psychiatric treatment	311.85 (232)***	.022	.989	Female	.31[a] (.07)	.58[a] (.09)	.23[a] (.09)	8.97 (3)
				Male	.12 (.06)	.54[a] (.09)	.06 (.07)	
Lifetime history of drug and alcohol treatment	328.92 (232)***	.024	.986	Female	.04 (.08)	.30[a] (.10)	.34[a] (.10)	3.19 (3)

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Model fit indices				Standardized estimate (*SE*)			
	χ^2 (*df*)	RMSEA	CFI	General	BPD	AAB	Wald test χ^2 (*df*)
Male				.09 (.06)	.08 (.10)	.17[a] (.08)	

Note: General = general factor; BPD = BPD group specific factor; AAB = AAB group specific factor;

*** $p < .001$

** $p < .01$.

[a] Denotes standardized estimates falling within the 95% CI.