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Antisocial (ASPD) and borderline (BPD) personality disorders (PDs) are associated with increased risk for substance use. They are "specific" risk factors among PDs in that they withstand adjusting for the other PDs, whereas the reverse does not hold. Specificity is a classic sign of causation. This empirical work addresses 3 further problems that can undermine causal inferences in personality and substance-use research: hierarchical nature of etiologic factors in psychiatry, imperfectly operationalized PD criteria, and possible genetic or environmental confounding, as seen in lack of "etiologic continuity." We used exploratory structural equation bifactor modeling and biometric models to mitigate these problems. The participants were Norwegian adult twins of ages 19–36 years ($N = 2{,}801$). Criteria for *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM–5)*, PDs were assessed using a structured interview. General substance-use risk was indicated by World Health Organization Composite International Diagnostic Interviewed alcohol use disorder and illicit drug use, and by self-reported regular smoking. A general risk factor for all criteria of both ASPD and BPD was the strongest individual correlate of general substance use and showed etiologic continuity, though just 3 specific PD criteria could predict substance use to the same extent. The findings indicate that a broad latent factor for both ASPD and BPD may be a specific and a genetically and environmentally unconfounded risk factor for substance use. Substance-use treatment research might benefit from attending to transdiagnostic models of ASPD, BPD, and related behavioral disinhibition.

Keywords: substance use disorders, population study, exploratory structural equation modeling, bifactor model, hierarchical model

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Personality disorders (PDs) inflict a huge burden on society and on individuals suffering from them. In Nordic countries, for example, PDs were associated with 13–22 year's reduction in life expectancy (Nordentoft et al., 2013). Risky behaviors, such as substance use and misuse, are one of the likely mechanisms through which PDs increase mortality and disability (Kuo et al., 2019; Lenzenweger, Lane, Loranger, & Kessler, 2007). Most PDs are associated with alcohol and substance use disorders (Gillespie, Aggen, Gentry, et al., 2018; Gillespie, Aggen, Neale, et al., 2018; Long et al., 2017; Trull, Jahng, Tomko, Wood, & Sher, 2010). PDs are highly comorbid, however, and studies that control one PD for the presence of other PDs have found that only antisocial (ASPD) and borderline (BPD) PDs independently and robustly predict substance use (Compton, Thomas, Stinson, & Grant, 2007; Gillespie, Aggen, Gentry, et al., 2018; Gillespie, Aggen, Neale, et al., 2018; Hasin, Stinson, Ogburn, & Grant, 2007; Long et al., 2017). These findings appear not to be substance specific, but rather implicate ASPD and BPD as specific PD-related risk factors for substance use in general.

In general, adverse life outcomes associated with substance use disorders are not specific to use of a given substance. Instead, a recent large study found that a one-factor model explained well intercorrelations among different substance use disorders (Franco et al., 2019). The same factor was the main predictor of their association with other adverse outcomes. The authors concluded that future work should examine the mechanisms underlying the latent factor for substance use and its relation to adverse life outcomes. In this study, we investigate specific criteria of ASPD and BPD as such potential mechanisms because they may mediate relationship between life events and substance use (Rosenström et al., 2019). Franco and colleagues (2019) did not find much specificity in associations between substance use disorders and adverse outcomes, but this does not necessarily hold for personality disorder traits (Rosenström et al., 2018). "Specificity" of an association is considered an important sign of causation (A. B. Hill, 1965), whereas understanding causation facilitates treatment and has been called for psychological treatments (Cuijpers, Reijnders, & Huibers, 2019).

ASPD and BPD constructs aggregate etiologically complex symptoms (Kendler, Aggen, & Patrick, 2012; Reichborn-Kjennerud et al., 2013; Rosenström et al., 2017), meaning that the aggregate constructs could dilute what is central to substance use risk in them, as suggested by some predictive models (Rosenström et al., 2018). Therefore, here we strive to model the full structure of ASPD and BPD criteria to disentangle specific effects in ASPD and BPD from general dispositions. Here we further consider three important insights from recent literature on psychiatric nosology, listed in this Introduction section and further expanded throughout the article.

First, nearly all psychiatric disorders appear to share some underlying common risk with each other, as discussed in the literature on "general psychopathology factor" or "p factor" (Caspi & Moffitt, 2018; Lahey, Krueger, Rathouz, Waldman, & Zald, 2017). In addition, normative and pathological personality traits may partly reflect the same general psychopathology factor (Oltmanns, Smith, Oltmanns, & Widiger, 2018; Rosenström, Gjerde, et al., 2019). Genetic overlap among PDs and other psychiatric disorders is pervasive (Kendler et al., 2011), and major ongoing efforts aim to organize all psychiatric disorders into a hierarchy of successively more specific etiologic factors (Kotov et al., 2017). This suggests that one should ask "what proportion of substance-use risk is attributable to specific *versus* non-specific factors of PDs" rather than "are there specific factors." An argument against the use of the general psychopathology factor has been that it is not of interest because it may reflect general impairment rather than a common cause (Oltmanns et al., 2018). However, a "measurement-invariant" general factor has been detected in onsets and recoveries of psychiatric disorders (Gluschkoff, Jokela, & Rosenström, 2019) and a general factor also fits with genetic and brain correlates of psychopathology (Elliott, Romer, Knodt, & Hariri, 2018; Goodkind et al., 2015; McTeague et al., 2017; Neumann et al., 2016; Wang, Gaitsch, Poon, Cox, & Rzhetsky, 2017) as well as responses to psychosocial treatment (Constantinou et al., 2019). Requiring more indicators of validity for a psychometrically derived construct than this would mean having to discount many constructs studied in clinical and personality psychology. Furthermore, neglecting possibility of shared general factors could lead to serious misinterpretations of specific traits (Caspi & Moffitt, 2018). For example, without a general factor the specific traits can act as continuous confounders for each other in multivariate analyses—a situation where both categorical measurement and measurement errors can induce statistical errors and bias (Austin & Brunner, 2004; Brunner & Austin, 2009). Here, we chose to explicitly take into account a general factor, whatever its underlying substantive interpretations might be, and consider how different levels of a hierarchy of ASPD and BPD risk factors relate to substance use risk. Bifactor models are typically used to extract a general factor but have been criticized for their high "fitting-propensity" in comparison with classic factor models (Markon, 2019). That discussion pertains to confirmatory frameworks which are not used in this study (see the following paragraph). Exploratory bifactor analysis model has the exact same fit as the exploratory factor analysis model; in fact, the models are indistinguishable on statistical grounds only (Jennrich & Bentler, 2011, 2012).

Second insight in recent literature is that the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (*DSM–5*), symptoms that make up the ASPD and BPD diagnoses may be viewed as currently best accepted but imperfect operationalizations for the PDs. They are lay language representations subject to semantic drift (changing meaning in different time periods) and multiple possible interpretations (Zandersen, Henriksen, & Parnas, 2019). As one example, Zandersen et al. (2019) discussed the BPD criterion "chronic feelings of emptiness", noting that the only additional remark *DSM–5* provides in terms of defining how this criterion characterizes individuals is: "Easily bored, they [individuals with BPD] may constantly seek something to do" (American Psychiatric Association, 2013, p. 664). This definition is general enough that it could describe addictive cravings as well as antisocial urges, among other things, whereas historically "chronic feelings of emptiness" have been associated with basic identity and a fragile sense of self-presence, characteristic of schizophrenia and other "self-disorders" (Zandersen et al., 2019). Exploratory structural equation models (ESEMs) have been developed in an attempt to mitigate consequences of fallible, nonpure, indicator variables—PD symptoms in this case—that may simultaneously reflect several latent constructs (Asparouhov & Muthén, 2009; Morin, Arens, & Marsh, 2016). Typical inferences based on the classic, confirmatory, approach to SEM are biased if for example,

"chronic feelings of emptiness" reflect both a latent factor for ASPD and another for BPD. ESEMs remove such bias, while retaining much of the flexibility of SEMs. Here, we combine ESEMs with hierarchical models of ASPD and BPD as substance-use risk factors.

Third, after putting our data to the above frameworks, we assess whether our main findings conform to *etiologic continuity* in having congruent multivariate findings for genetic and environmental variance in the observed phenotypes (Kendler et al., 2019). That is, we say PDs are etiologically continuous with risk of using various substances if the indicators of the PDs and the substances are "correlated primarily because they share etiologic influences (as opposed to genetic and environmental forces working in distinct ways on each indicator)" (Mullins-Sweatt, DeShong, Lengel, Helle, & Krueger, 2019, p. 56). Technically, etiologic continuity is assessed via "common pathway biometric model," further explained in the Method section (Franić et al., 2013; Kendler, Heath, Martin, & Eaves, 1987; Livesley, 2005; Neale & Cardon, 1992). Etiologic continuity provides evidence for specificity and causation because it supports attributing an association to observed phenotypes instead of genetic or environmental confounders working in distinct ways on each indicator (Briley et al., 2019; Rosenström, Czajkowski, et al., 2019).

The existing evidence on ASPD, BPD, and substance use suggests that efforts should be taken to implement the above three considerations in empirical works. For example, the etiologic factors of ASPD and BPD appear to form a hierarchy of highest, intermediate, and lowest level of generality, as expressed in bifactor models (Figure 1a). Diagnoses of ASPD and BPD share genetic etiology with each other and with other related PDs, but they also have independent genetic influences (Chun et al., 2017; Reichborn-Kjennerud et al., 2015; Torgersen et al., 2008). Specific criteria for ASPD (respectively BPD) share many genetic influences, but also they have specific (i.e.,

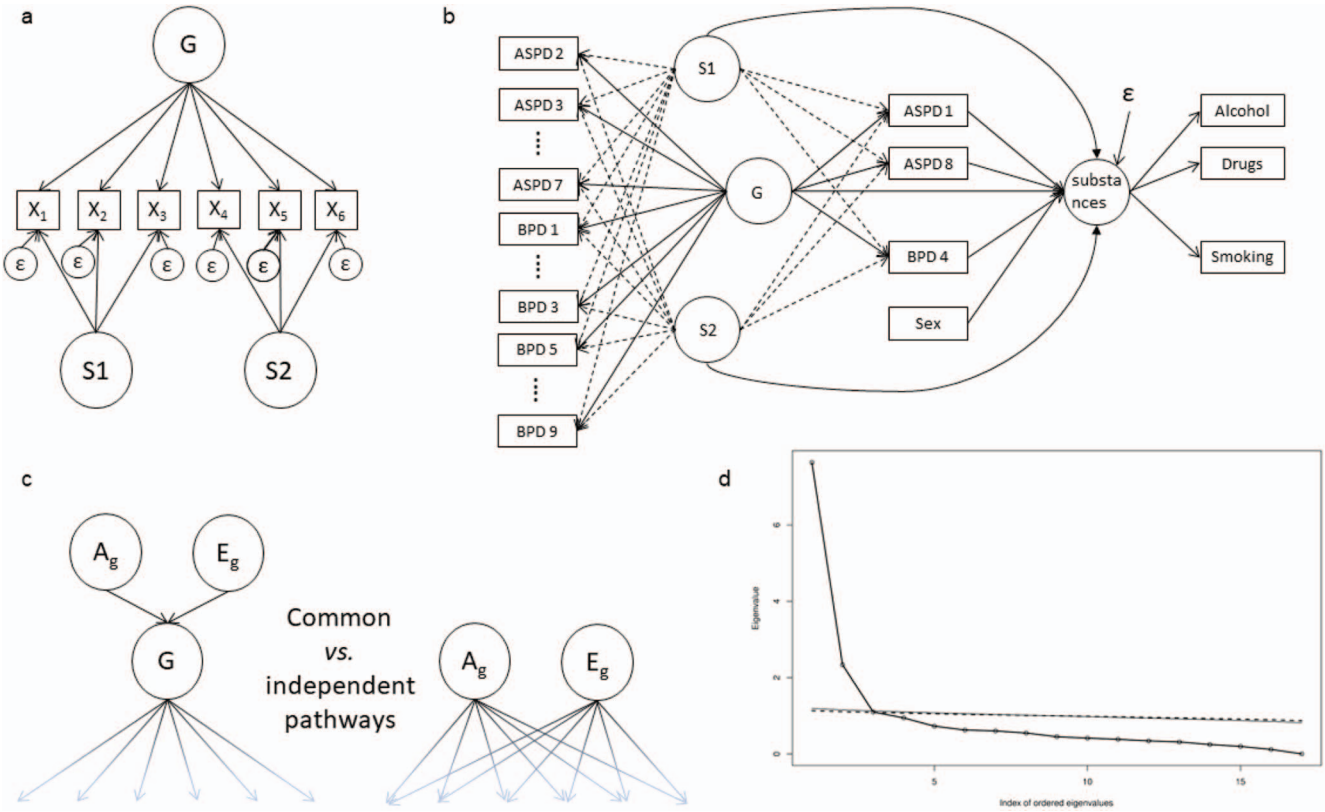

Figure 1. Path modeling. (a) A bifactor model captures a hierarchy of successively more specific unobserved etiologic factors (circles) underlying the observed variables (X; boxes): the general factor (G) affects everything, the specific factors (S1 and S2) affect subgroups of variables, and the unique variances affect just one variable (ε; these are always modeled, but often not drawn to path diagrams). (b) In exploratory bifactor models also the specific factors (S1 and S2) can "cross-load" on every variable (cf. dashed lines), but they are rotated to load maximally on just one specific factor. In our exploratory structural equation model, both the latent factors and three observed personality criteria are allowed to predict the substance use factor. (c) Additive genetic (A_g) and environmental (E_g) factors can influence variables through a common phenotypic pathway (G; left-hand side) or the phenotype may be "illusory" in that the genes and environments exert their effects through independent pathways, as in right-hand side (refer Rosenström et al., 2017, for more examples). A common-pathway model is "nested" within independent pathway model, allowing statistical null-hypothesis testing of the common-pathway model. (d) A parallel analysis plot, suggesting three phenotypic factors. See the online article for the color version of this figure.

criterion-specific) genetic and environmental influences (Kendler et al., 2012; Reichborn-Kjennerud et al., 2013; Rosenström et al., 2017).

Although ASPD and BPD are known to share genetic influences with substance use risk (Gillespie, Aggen, Gentry, et al., 2018; Gillespie, Aggen, Neale, et al., 2018; Long et al., 2017), it is unclear where substance use is situated within the hierarchy of etiologic factors for these PDs. Many researchers argue that a broad and continuous dimension of externalizing psychopathology, or that of general liability to all psychopathology, is the key trait behind ASPD, BPD, and all substance use (Caspi & Moffitt, 2018; Kotov et al., 2017; Krueger, Markon, Patrick, Benning, & Kramer, 2007; Rosenström, Gjerde, et al., 2019; Soe-Agnie, Paap, VanDerNagel, Nijman, & de Jong, 2018). Yet, there are also findings on the lowest level of risk-factor hierarchy—a level of explanation that has been frequently overlooked in personality research for technical reasons (Ashton, Paunonen, & Lee, 2014). Using modern tools that mitigate technical problems, for example, due to overfitting, we previously found that three specific PD criteria independently increased alcoholism risk over and above all the other PD criteria (80 altogether) and diagnoses (10 altogether): "failure to conform to social norms with respect to lawful behavior," "childhood conduct disorder" (ASPD Criteria 1 and 8), and "self-damaging impulsivity" (BPD Criterion 4; Rosenström et al., 2018). It remains unclear whether those "independent" effects derive from the criterias' central role in the etiology of the PDs or from their own specific etiologies, but such questions can be addressed using bifactor ESEMs.

(Bifactor) modeling of the hierarchy of etiologic factors behind ASPD and BPD does not itself solve the problem of impurely operationalized criteria (Morin et al., 2016). In addition to the problems of semantic drift and multiple interpretations discussed by Zandersen et al. (2019), one could note that PD criteria like "self-damaging impulsivity" and "conduct disorder" are themselves complex phenotypes (Aggen, Neale, Røysamb, Reichborn-Kjennerud, & Kendler, 2009; Dick et al., 2010; Kendler, Aggen, & Patrick, 2013). Most likely, it is unrealistic to assume that the *DSM–5* criteria for ASPD and/or BPD are "pure" indicators of single unidimensional constructs that will have factor cross-loadings of zero on any closely related constructs (Morin et al., 2016). Fortunately, the "exploratory" version of bifactor model relaxes the pure-indicator assumption (Morin et al., 2016) of the more restricted, confirmatory bifactor models (Eid, Geiser, Koch, & Heene, 2017; Gibbons & Hedeker, 1992).

When it comes to etiologic continuity versus confounding, mediational relationships between stressful life events, BPD, and alcohol use disorder (AUD) may be subject to genetic and/or environmental confounding (Bornovalova et al., 2013; Rosenström, Czajkowski, et al., 2019). Whatever predominant substance-use risk factor emerges from hierarchical modeling of ASPD and BPD criteria, it should ideally be subjected to a test of etiologic continuity. After all, a better understanding of etiology helps in design of prevention strategies and treatment programs. Questions could be answered regarding choice of therapies specifically for substance use versus more unified protocols covering wider sets of psychopathologies, as well as those regarding treatments directed toward overarching personality pathologies versus specific thoughts and behaviors indicated by individual diagnostic criteria.

The Current Study

Whereas many previous studies have found evidence that ASPD and BPD have strong links with general substance use risk, the exact etiology behind the link remains unclear. We know ASPD and BPD are constructs with partly overlapping etiologies that aggregate over criteria with partly distinct etiologies, which calls for a criterion-level reassessment of the findings. We know that the plausible existence of a continuous general factor can bias the typical regression-based epidemiology on categorical disorders. We know that confirmatory bifactor models, although useful and appropriate in certain applications, can lead to overfitting data, whereas the less often used explorative bifactor models are rotations with the exact same fit as for the classic factor model, reducing the reliance on the assumption of pure indicators. We suspect that few indicators are "pure" in the sense that they are completely unique in identifying a particular PD, which implies a degree of bias or interpretational confounding when fitting typical confirmatory models. And, we know that bifactor ESEMs can partly eliminate biases due to nonpure indicators, categorical formulations, measurement errors, and confirmatory bifactor modeling. What we know less about is what happens when attempts are made to reduce the effects of these known sources of bias: What kind of etiologic constructs arise from data then and how do they associate with substance use risk? This is the question this article aims to clarify.

This study aims to clarify the etiology of substance use in relation to its risk PDs, while addressing the aforementioned three complicating factors (i.e., hierarchical nature of etiologic factors in psychiatry, imperfectly operationalized PD criteria, and genetic and/or environmental confounding). To that end, we take an exploratory structural equation modeling approach. First, we assessed the joint phenotypic hierarchical structure of the ASPD and BPD criteria. Second, we investigated how the structure relates to substance use risk when minimizing the introduced modeling pitfalls (i.e., determined which levels of the hierarchy captured the predominant risk factor for general substance use). Third, we determined what genetic and environmental etiology pertains to the PD-related substance use risk (i.e., determined how genetic and environmental influences on the predominant risk factor were related to general substance use risk). Because the structural equation models used here to address the aforementioned complicating factors typically make more assumptions than many regression models and suffer more from computational and data-related bottlenecks (VanderWeele, 2012; Zahery, Maes, & Neale, 2017), we concentrate only on the core constructs of ASPD, BPD and substance use, leaving other psychiatric comorbidities for future research.

Method

Sample

The participants, 2,801 Norwegian twins, were drawn from the Norwegian Institute of Public Health's Twin Panel (Harris, Magnus, & Tambs, 2002; Tambs et al., 2009). The zygosity of the twins was determined by a combination of questionnaire items and genotyping. The sampling targeted twins born between 1967 and 1979, capturing 43.5% of those eligible. Their mean age was 28.2

years (range 19–36 years). Their *DSM–IV* Axis I and Axis II psychiatric disorders were assessed in an interview between 1999 and 2004. Although 2,284 of the twins were reinterviewed again in between 2010 and 2011 and they completed a self-report questionnaire in 2017–2018, here we concentrate on the first wave of data collection because recording of substance use has differed across the three waves, and because genetic etiology of substance use differs by age and peaks in young adults (E. M. Hill & Chow, 2002; Torvik et al., 2017). Approval for this study was received from The Norwegian Data Inspectorate and the Regional Committee for Medical and Health Research Ethics, and written informed consent was obtained from all participants.

Procedure

PDs within past 5 years were assessed using a Norwegian version of the Structured Interview for *DSM–IV* Personality (Pfohl, Blum, & Zimmerman, 1995), a comprehensive semistructured interview of all *DSM–IV* Axis II diagnoses that produced an ordinal rating of the specific *DSM–IV* criteria (0 = *not present or limited to rare isolated examples*, 1 = *subthreshold*, 2 = *present*, 3 = *strongly present*; Reichborn-Kjennerud et al., 2015; Rosenström et al., 2018, for more detail). PD diagnoses are identical in *DSM–5* and *DSM–IV* (American Psychiatric Association, 2013).

We studied general risk of substance use rather than risks for specific substances, as previous research has indicated that ASPD and BPD are nonspecific predictors of substance use and as use disorders of different substances reflect a single dimension of risks and outcomes (Franco et al., 2019). Specifically, an underlying risk factor was modeled using three available *indicators* of general substance use: AUD, illicit drug use, and smoking. The indicators for AUD and drug use were assessed using the computerized Norwegian version of World Health Organization's Composite International Diagnostic Interview (Wittchen & Pfister, 1997), whereas smoking status was assessed in a separate mailed questionnaire. Lifetime AUD was indicated by either alcohol abuse (F10.1 in International Classification of Diseases, 10th revision [ICD-10]) or dependence (F10.2). Because specific drug use disorders are rare outcomes, we combined illicit drug use into an ordinal variable with Value 0 indicating no serious use, Value 1 indicating having used illegal (nonprescribed) drugs more than 10 times, and Value 2 indicating a disorder or dependency for opioids, cannabis, sedative, cocaine, amphetamine, hallucinogens, or inhalants (as in F11–16 and F18). Smoking was indicated as the status of current regular smoking (yes/no). See previous studies for more details on the procedures (Harris et al., 2002; Reichborn-Kjennerud et al., 2015; Tambs et al., 2009). Note that smoking reflects further health hazards (e.g., lung cancer) besides its strong association with general substance use risks (Franco et al., 2019), but here we are primarily interested in shared behavioral antecedents of substance use.

Statistics

Exploratory structural equation models (ESEM) were fit to underlying liabilities for the binary and ordinal-valued indicator variables using liability-threshold modeling (Asparouhov & Muthén, 2009; Falconer, 1965; Morin et al., 2016; Neale & Cardon, 1992). Liability-threshold models estimate an underlying normally distributed continuum of risk behind each crude categorical observation item to remove the bias in ESEMs that would otherwise result from the low (i.e., ordinal) measurement precision. All the ESEM models were fitted using mean- and variance-adjusted weighted least squares estimator of Mplus software. Because members of twin pairs are more similar to each other than to the other twins, they represent clusters of dependent observations. Sandwich estimators were therefore used to correct the ESEM estimates for the dependent observations (Asparouhov, 2005; Højsgaard, Halekoh, & Yan, 2006). The underlying heritability patterns behind the phenotypes were studied using the "Open Mx" R package, "ACE" twin design, and full-information maximum likelihood estimation (Neale et al., 2016; Neale & Cardon, 1992). The twin design uses the average 100% genetic similarity of monozygotic twins and 50% similarity of dizygotic twins to estimate heritability and correlations of genetic and (shared and nonshared) environmental influences for given variables. Regarding, specific study questions, the following logic was applied.

To assess the hierarchical structure of ASPD and BPD (Research Question 1), "parallel analysis criterion" was used to determine the optimal number of underlying factors for modeling of covariance among the ASPD and BPD criteria, because previous studies support the strategy (Garrido, Abad, & Ponsoda, 2013, 2016; Hayashi, Bentler, & Yuan, 2007; Rosenström et al., 2017). Parallel analysis sorts (Mplus estimates of) eigenvalues of the polychoric correlation matrix in descending order, and then compares them to average descending eigenvalues from 1,000 simulated data sets of the same size but with no underlying factors; the estimated factor number corresponds to those eigenvalues that exceed the average sampling noise in the simulated values. Given the number of factors, the estimated bifactor loadings and their stability were investigated to interpret the underlying factors. A bifactor rotation does not affect factor number nor model fit (Jennrich & Bentler, 2011, 2012), and its robustness (Mansolf & Reise, 2016) was verified in our online supplement by rerotating from 1,000 distinct randomly generated starting rotations (Mezzadri, 2007).

To assess which levels in the hierarchical model of ASPD and BPD criteria best predict general substance use risk (Research Question 2), an ESEM described by the path diagram of Figure 1b was fit to the data (Morin et al., 2016). This model assumes a hierarchy of general, specific, and criterion-specific influences in ASPD and BPD items. It does not fix factor loadings a priori before using the members of the factorial hierarchy to predict a general substance use factor (controlling sex). Instead, all the parameters are estimated simultaneously. In a direct analogy with usual regression models, however, the ESEM model can be used to test which predictors are needed (statistically significant). The factor loadings are estimated jointly with the other model structure using an exploratory bifactor (an orthogonal "biquartimin") rotation (Jennrich & Bentler, 2011, 2012) that rotates the solution as close to a hierarchical pattern as possible (cf. Figure 1a). The ASPD Criteria 1 (failure to conform to social norms) and 8 (childhood conduct disorder) and the BPD Criterion 4 (self-damaging impulsivity) are treated similarly to the other PD criteria in the factor analysis part of the model, but are only illustrated in the middle of Figure 1b because they are also directly used in the regression part of the model, based on their previously shown independent associations with AUD (Rosenström et al., 2018).

To explore continuity of etiologic factors behind ASPD, BPD, and substance use (Research Question 3), multivariate twin models were estimated. Specifically, we tested whether the pertinent dimensions of risk reflected common versus independent genetic and environmental pathways (cf. Figure 1c; Franić et al., 2013; Kendler et al., 1987; Neale & Cardon, 1992; Rosenström et al., 2017). The most parsimonious path model was then used to partition variance to shared versus specific components, which were further partitioned to genetic versus environmental components. We used a different software (Open Mx) to assess etiologic continuity than for bifactor modeling (Mplus) because it has been developed specifically for such questions. We concentrated only on core variables (discussed in the following text) because pertinent biometric models failed to converge for all the variables. Even when they do so, achieving convergence for single models of this many variables takes several weeks and does not always provide reliable results (Kendler et al., 2019; Zahery et al., 2017).

Results

Bifactor Structure of Borderline and Antisocial Personality Disorder Criteria

According to the parallel analysis criterion, a three-factor solution was sufficient to capture the correlations between the PD criteria (Figure 1d). Although that analysis left room to argue also for a two-factor solution, we used a three-factor solution because overfactoring is generally considered less detrimental than underfactoring (Hayashi et al., 2007) and because three factors was favored also by the root mean squared error of approximation (RMSEA) fit index (RMSEA = 0.019 with 90% confidence interval [CI; 0.015, 0.023] vs. RMSEA = 0.030 with CI [0.027, 0.033]). According to a bifactor rotation (see Table 1), the three factors could be interpreted as (a) a general risk factor for both antisocial and borderline personality disorder (titled G), (b) a specific factor distinguishing affective BPD criteria from remorseless antisocial behaviors (S1), and (c) a specific factor for aggression (S2). Although all ASPD and BPD criteria loaded strongly on the general-risk factor (G in Table 1), the preselected three PD criteria had much more variance (more endorsements of nonzero categories) than the other criteria (see Table 1), meaning they were relatively well-suited for measuring a wide range of G-factor values (i.e., suffered less from "range restriction" than most PD criteria). The orthogonal bifactor rotation was preferred for this study because it was able to differentiate between hierarchies of risk factors, but see online supplemental materials for alternative classic rotations and for our sensitivity analysis suggesting the bifactor solution was robust.

Factor Structure of General Substance Use

Loadings of AUD, illicit drug use, and smoking on the general substance use factor were 0.694, 0.826, and 0.396, respectively (see Table 2). This means that the general factor explained 48%, 68%, and 16% of variance in the liabilities to endorse AUD, drug use, and smoking, respectively. Thus, smoking was clearly related to the general liability to substance use, but less so than AUD and illicit drug use.

Which Level of Personality Pathology Best Captures General Risk for Substance Use?

We then investigated the model described in Figure 1b (Model 1) and its versions where regression coefficients of substance use

Table 1
Exploratory Bifactor Loadings for Antisocial and Borderline Personality Disorder Criteria

		Factor loadings (Λ)			
DSM-5 criterion	Abbreviated criterion content	G: General	S1: BPD vs ASPD	S2: Aggression	Observed variance
ASPD1	Not conforming	**0.762**	**−0.463**	0.136	0.159
ASPD2	Deceitfulness	**0.565**	−0.157	0.015	0.070
ASPD3	Impulsivity or failure to plan	**0.697**	0.090	**0.328**	0.063
ASPD4	Irritability/repeated fights	**0.849**	−0.270	**−0.353**	0.034
ASPD5	Reckless disregard	**0.507**	−0.296	0.111	0.072
ASPD6	Irresponsibility	**0.716**	−0.028	**0.407**	0.090
ASPD7	Lack of remorse	**0.760**	**−0.451**	−0.014	0.037
ASPD8	Conduct disorder	**0.663**	−0.260	0.181	0.280
BPD1	Avoid abandonment	**0.533**	**0.362**	0.028	0.140
BPD2	Unstable relationships	**0.633**	**0.424**	−0.027	0.227
BPD3	Identity disturbance	**0.607**	**0.411**	0.113	0.032
BPD4	Self-damaging impulsivity	**0.738**	−0.195	0.264	0.317
BPD5	Suicidality or self-mutilation	**0.612**	0.302	0.037	0.135
BPD6	Affective instability	**0.605**	**0.496**	−0.172	0.369
BPD7	Feelings of emptiness	**0.488**	**0.510**	−0.028	0.213
BPD8	Inappropriate intense anger	**0.650**	0.139	**−0.626**	0.256
BPD9	Stress-related paranoia	**0.604**	0.309	0.063	0.079

Note. BPD = borderline personality disorder criterion/trait; ASPD = antisocial personality disorder criterion/trait. Factor loadings above $\sqrt{0.1}$ are highlighted with bold font. The last column shows raw observed variance per criterion to demonstrate the large differences in endorsement rates of the criteria. These numbers characterize potentially informative variance and its relationship to a latent factor. However, factor loadings were estimated using liability-threshold modeling, i.e., an underlying continuum was modeled instead of direct modeling of ordinal data.

Table 2
Prevalence of Substance Use and Factor Loadings on a General Substance Use Factor

	Observed frequency			Model estimate
Substance use variable	Not present	>10 times	Present	Factor loading
AUD	2,528	—	264	0.694
Illicit drug use	2,586	133	54	0.826
Smoking	1,174	—	492	0.396

Note. AUD = alcohol use disorder. ">10 times" refers to having used illicit drugs more than 10 times; such intermediate category was not recorded for AUD and smoking. Factor loadings were estimated using liability-threshold modeling, i.e., an underlying continuum was modeled instead of direct modeling of ordinal data.

on the preselected criteria (Model 2) or those on the latent factors (Model 3) were constrained to zero. Both the latent factors (χ^2 = 22.537, df = 3, $p < .001$) and the preselected risk criteria (χ^2 = 19.381, df = 3, $p < .001$) were independently associated with the general substance use factor (i.e., constrained versions of Model 1 were rejected). However, all three models had practically equal fit to data (Table 3; see also supplementary analysis of residuals following Maydeu-Olivares, 2017), whereas a model using only sex as a predictor had a significantly worse fit (e.g., RMSEA = 0.082 with CI [0.079, 0.084]).

The criterion-level and the factor-level predictors of general substance use appeared near-multicollinear in the sense that the full-hierarchy model (Model 1) had rather different pattern of regression coefficient compared with the higher- and lower level predictive models (Models 2 and 3, respectively). To sum, although all levels of personality hierarchy were associated with general risk for substance use, the effects of factor versus criteria were largely exchangeable (i.e., traded predictive variance), with G being the dominant predictor (cf. Model 2).

Etiologic Continuity in Inheritance Patterns

Both the aforementioned phenotypic analyses and a supplementary biometric analysis of factor scores (Figure S3 in the online supplemental materials) suggested that mainly the general PD factor played a role in associations between the PDs and the general substance use risk. According to the phenotypic factor rotation (see Table 1), the criteria that loaded primarily only on the general factor were ASPD Criteria 2, 5, and 8 and BPD Criteria 4, 5, and 9. We used a count of these criteria (full or subthreshold) to explore the multivariate genetic and environmental structure of the general PD factor and the different substances (i.e., the orthogonal residual PD factors were excluded for the sake of clarity and computational feasibility).

We started from the most general model, which modeled independent genetic (A) and shared- (C) and nonshared (E) environmental factors for both the overlapping and the specific variance in the variables, as well as sex-specific factor loadings and ordinal thresholds. Although this "independent pathway model" slightly differed

Table 3
Exploratory Structural Equation Regression Models Predicting Substance Use Risk With a Hierarchy of General, Specific (S), and Criterion-Level (Crit.) Personality Pathology

	Model 1			Model 2			Model 3		
Predictor	β	*SE* (β)	*p*	β	*SE* (β)	*p*	β	*SE* (β)	*p*
Male sex	0.421	0.083	.000	0.420	0.083	.000	0.419	0.083	.000
General PD factor	−0.136	0.214	.526	0.764	0.085	.000	—	—	—
S1 (BPD vs. ASPD)	0.334	0.135	.013	−0.148	0.176	.402	—	—	—
S2 (Aggression)	−0.051	0.078	.509	−0.292	0.079	.000	—	—	—
ASPD Crit. 1	0.365	0.090	.000	—	—	—	0.230	0.042	.000
ASPD Crit. 8	0.109	0.054	.044	—	—	—	0.111	0.041	.006
BPD Crit. 4	0.177	0.061	.004	—	—	—	0.243	0.046	.000
	Value	90% CI	*p* (RMSEA < .05)	Value	90% CI	*p* (RMSEA < .05)	Value	90% CI	*p* (RMSEA < .05)
Fit index									
RMSEA	0.038	[0.036, 0.041]	1.000	0.039	[0.036, 0.041]	1.000	0.039	[0.036, 0.041]	1.000
CFI	0.927	—	—	0.925	—	—	0.925	—	—
TLI	0.899	—	—	0.899	—	—	0.899	—	—
AASRC	0.037	—	—	0.039	—	—	0.041	—	—

Note. Because the specific-aggression factor (S2) was inverted, the negative coefficient implies that high aggression is associated with substance use. PD = personality disorder; BPD = borderline personality disorder criterion/trait; ASPD = antisocial personality disorder criterion/trait; CI = confidence interval; RMSEA = root of mean squared error of approximation; CFI = comparative fit index; TLI = Tucker–Lewis index; AASRC = average absolute sample residual correlation. AASRC is just a mean absolute difference between model-predicted and observed correlations, which provides intuition on effect size for model misfit.

from a "common pathway model" ($\chi^2 = 23.71$, df = 23, $p = .022$; cf. Figure 1c), both Akaike's and Bayesian information criteria (AIC and BIC) suggested that a common pathway model was more parsimonious interpretation of the data (i.e., a common pathway model was favored by $\Delta AIC = -0.29$ and $\Delta BIC = -83.53$). A previous simulation study suggested that one should rely on BIC in this model comparison (Markon & Krueger, 2004). The shared environmental influences (C parts) in the model were nonsignificant and were therefore not further interpreted or modeled ($\chi^2 = 3.77$, df = 10, $p = .957$). The model could be further simplified by constraining factor loadings (but not thresholds) across the sexes ($\chi^2 = 6.37$, df = 14, $p = .956$). Figure 2 summarizes the biometric relationships between the substances and the general liability to ASPD and BPD.

From Figure 2, we see that a common-pathway general factor explained roughly 42% (i.e., $100\% \times 0.668^2$), 67%, 41%, and 18% of variance in the (proxy of) general PD factor, illicit drug use, AUD, and smoking, respectively. Of these influences, 89% (i.e., $100\% \times 0.943^2$) were of additive-genetic origin. However, also specific genetic variance existed in the variables ($\chi^2 = 91.25$, df = 4, $p < .001$; Figure 2), in addition to the etiologically continuous latent factor of shared risks.

A Sensitivity Analysis of Content Overlap

In interpreting findings like herein, one is often concerned that the specific criteria may contain a degree of content overlap with substance use outcomes. Our previous research and Figure S3 in the online supplemental materials indicate that simple content overlap is an unlikely explanation of our findings (Gillespie, Aggen, Gentry, et al., 2018; Long et al., 2017; Rosenström et al., 2018).

Discussion

The current study demonstrates that a single shared underlying dimension of risk for both ASPD and BPD criteria largely explains the associations between the PD criteria and substance use. Three simple PD criteria were efficient clinical proxies for this latent dimension: social-norm violations (ASPD Criterion 1), conduct disorder (ASPD Criterion 8), and self-damaging impulsivity (BPD Criterion 4). The latent dimension of general risk for ASPD, BPD, and substance use reflected primarily heritable population variance (89% with 95% CI [77%, 99%]), but also environmental influences were detected (11% with CI [1%, 22%]). It showed evidence

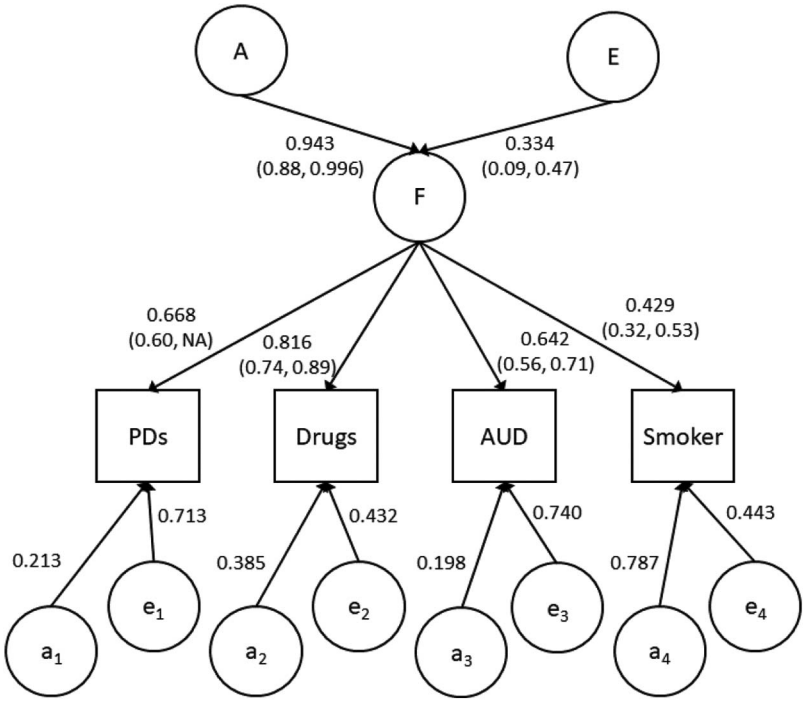

Figure 2. A path diagram of the final multivariate twin model. A biometric common pathway model of general antisocial–borderline personality (PDs), illicit drug use (Drugs), alcohol use disorder (AUD), and regular smoking (Smoker). The model partitions the covariance structure of the variables into a "common-pathway" trait (F), its genetic (A) and environmental (E) influences, and to variable-specific "residual" genetic (a_i) and environmental (e_i) influences ($i = 1 \ldots, 4$). Variances are standardized to unity (i.e., square of a path coefficient indicates proportion of explained variance). Parentheses give 95% likelihood-profile confidence intervals for the path coefficients. Value "NA" was substituted for the upper interval limit without a convergent estimator, and significance of genetic residuals was only collectively tested for similar reasons. The variable "PDs" stands for a liability-threshold model for a count of subthreshold or full endorsements of ASPD Criteria 2, 5, and 8 and BPD Criteria 4, 5, and 9, as they had strong loadings exclusively on the general PD factor in Table 1. ASPD = antisocial personality disorder; BPD = borderline personality disorder.

of etiologic continuity, or lack of genetic and/or environmental confounding. Our findings suggest that ASPD and BPD traits largely fall on the same shared dimension of risk factors with substance use disorders and a wide range of adverse outcomes (Franco et al., 2019).

A previous study on the joint structure of ASPD and BPD criteria suggested that an overarching general dimension of risk for all the criteria plus other psychopathology exists (Chun et al., 2017). Similarly, our investigation underscored the importance of such an overarching dimension of risk. Unlike previous studies, we did not assume a factor loading pattern a priori, but instead used an exploratory approach (Morin et al., 2016) to address concerns about operationalization of PD criteria (Zandersen et al., 2019). The exploration revealed a general factor of liability for all the criteria of both ASPD and BPD ("G factor"), a specific (residual) factor capturing tendency to manifest affective instability instead of norm violations and lack of remorse ("factor S1"), and a specific factor capturing anger proneness ("factor S2"; sign inverted). We do not enter further speculation on the specific factors here, as they were less relevant to substance use than the general factor. However, they may suggest interesting targets for future research.

Our current findings using ESEM analysis framework found that ASPD and BPD criteria were not specific in predicting substance use and thus seemingly depart from our previous report that found three specific criteria as best predictors of AUD using a modern regression analysis framework (Rosenström et al., 2018). However, current methods focused on etiology whereas the previous ones focused on prediction. Even though our present findings suggest a strong etiologic role for factors affecting both ASPD and BPD, they do not necessarily suggest using these diagnostic constructs in clinical predictions of substance-use risk. ASPD and BPD diagnoses poorly predict AUD, for example, because they are so rare in the population in comparison with AUD (Rosenström et al., 2018). Similarly, many of their specific criteria have relatively little variance (see Table 1). Simply formulated and well-predicting criteria can have a high clinical utility, whereas different models are needed to understand their etiologic role (Briley et al., 2019). Indeed, our present *predictive* analysis was in line with the previous one in that three specific PD criteria were sufficient for predicting AUD (Rosenström et al., 2018).

Regarding understanding etiology, we found that the latent dimension of risk for ASPD, BPD, and substance use reflect a "common pathway" rather than independent genetic and environmental influences. Such etiologic continuity appears consistent with previous findings on ASPD, BPD, drug abuse, AUD, externalizing spectrum, and adverse outcomes (Franco et al., 2019; Kendler et al., 2016; Reichborn-Kjennerud et al., 2013; Rosenström et al., 2017, 2019), although it may not generalize to all personality domains (Franić et al., 2013; Kendler et al., 2019).

Although our present analysis does not directly inform what could give rise to an etiologically continuous latent factor associated with ASPD and BPD criteria and substance use, it may be worthwhile to speculate upon certain patterns found in literature. The PD criteria tapping to this factor appeared to lie on a causal pathway from stressful childhood environment to AUD in an earlier article (Rosenström, Czajkowski, et al., 2019). They are also related to "disinhibition," which has been suggested as a unifying construct in understanding how personality disposition undergird psychopathology (Mullins-Sweatt et al., 2019, p. 55): "the construct of disinhibition (*versus* constraint) is a broad personality trait that refers to individual differences in the ability to self-regulate or control one's behavior, p. 13-14." Such ability would presumably develop worse in worse environmental conditions, explaining why environmental enrichment reduce risk of PDs (Raine, Mellingen, Liu, Venables, & Mednick, 2003), and why personality-targeted interventions may lead to long-term reductions in substance-use behaviors (Conrod, Castellanos-Ryan, & Mackie, 2011). Thus, the general PD and substance use risk factor we found could represent or overlap with the construct of disinhibition. In contrast, reverse causation from substance use to PD pathology seems less likely. ASPD and BPD tend to temporally precede non-PD psychopathologies, including substance use (Defoe, Khurana, Betancourt, Hurt, & Romer, 2019; Gunderson et al., 2004; Young, Sweeting, & West, 2008), often exhausting PD-related genetic risk factors (Gillespie, Aggen, Gentry, et al., 2018; Reichborn-Kjennerud et al., 2010; Rosenström et al., 2018). A recent review of BPD and substance use concluded that substance use may exacerbate PD symptomatology, but "because common genetic, personality, and early influences predate overt substance use, it seems unlikely that PDs are simply secondary to substance use" (Trull et al., 2018, p. 10). Normative personality traits also primarily precede, for example, episodes of depression, with only very small reverse effects at the most (Klein, Kotov, & Bufferd, 2011; Rosenström et al., 2014, 2015), although their interaction with alcohol and substance use appears more complex (Hakulinen & Jokela, 2019; Kendler, Ohlsson, Sundquist, & Sundquist, 2014).

Recent studies that included many psychiatric disorders and indicators of personality have reported a very broad underlying dimension of risk that encompasses both personality pathology and other psychopathology (Kotov et al., 2017; Krueger et al., 2007; Oltmanns et al., 2018; Rosenström, Gjerde, et al., 2019). This study concurs in showing that the key personality pathologies in substance use risk can be interpreted as reflecting a broad continuum of risk. Findings on etiologic overlap among the broad dimensional constructs appear solid, but accurate estimates of the full extent of overlap may turn out elusive for practical reasons. Accurate analysis of increasingly complex models may require increasingly large data (although exceptions exist [Rosenström, Czajkowski, et al., 2019; Schmitz, Cherny, & Fulker, 1998]). At the same time, accurate estimation of structural equations on ordinal variables requires repeated numeric integration of multivariate normal distribution. Therefore, complex models with many variables may become prohibitively slow to compute or they may require different methods than problems involving less variables (Gassmann, Deák, & Szántai, 2002; Gibbons & Hedeker, 1992; Kendler et al., 2019; Zahery et al., 2017).

The present findings should be interpreted in the light of important limitations. First, the sample is subject to moderate selection toward good health (Tambs et al., 2009). Ideally the findings should be replicated in a more disabled population; however, care must be taken if the population has been selected based on aggregate measures of ASPD, BPD, or substance use, because that may distort factor structures (Muthén, 1989). Second, a more comprehensive analysis with more substances might reveal further effects. Third, it should be kept in mind that modeling of latent liabilities does not directly translate to ability to predict new observations (but see our previous article for some cross-validated predictions [Rosenström et al., 2018]). Fourth, we had self-report and interview data on substance use, but no objective biomarkers that could eliminate risk of dishonest reporting. Nevertheless, our findings suggest that treatments and theories target-

ing a wider set of behavioral problems than just substance use could be more efficient long-term solutions than those targeting substance-use behaviors only (Bateman, Gunderson, & Mulder, 2015; Conrod et al., 2011; Newton-Howes & Foulds, 2018). Transdiagnostic models of ASPD and BPD could be useful for addiction research.

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