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Distinguishing General and Specific Personality Disorder Features and Implications for Substance Dependence Comorbidity

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Abstract

Clinical and population-based samples show high comorbidity between Substance Use Disorders (SUDs) and Axis II Personality Disorders (PDs). However, Axis II disorders are frequently comorbid with each other, and existing research has generally failed to distinguish the extent to which SUD/PD comorbidity is general or specific with respect to both specific types of PDs and specific types of SUDs. We sought to determine whether ostensibly specific comorbid substance dependence-Axis II diagnoses (e.g., alcohol use dependence and borderline personality disorder)

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are reflective of more pervasive or general personality pathology or whether the comorbidity is specific to individual PDs. Face-to-face interview data from Wave 1 and Wave 2 of the *National Epidemiologic Survey on Alcohol and Related Conditions* were analyzed. Participants included 34,653 adults living in households in the United States. We used hierarchical factor models to statistically partition general and specific personality disorder dimensions while simultaneously testing for specific PD-substance dependence relations. Results indicated that substance dependence-Axis II comorbidity is characterized by general (pervasive) pathology and by Cluster B PD pathology over and above the relationship to the general PD factor. Further, these relations between PD factors and substance dependence diagnoses appeared to largely account for the comorbidity among substance dependence diagnoses in the younger but not older participants. Our findings suggest that a failure to consider the general PD factor, which we interpret as reflecting interpersonal dysfunction, can lead to potential mischaracterizations of the nature of certain PD and SUD comorbidities.

Keywords

substance dependence; personality disorders; hierarchical structural equation modeling; comorbidity

Both clinically ascertained and population-based samples show high comorbidity among Axis I disorders and Axis II Personality Disorders (PDs; Dahl, 1986; Koenigsburg, Kaplan, Gilmore, & Cooper, 1985; Kosten, Rounsaville, & Kleber, 1982; Sher, Trull, Bartholow, & Vieth, 1999; Sher & Trull, 2002; Shea et al., 2004). In particular, the prevalence of several PDs is high in individuals with substance use disorders (SUDs; Sher & Trull, 2002). However, the extent to which specific PD-SUD associations are attributable to general pathology common to all PDs, rather than to specific PD symptoms or symptom clusters (e.g., criteria sets), has not been adequately tested (see Sher et al., 1999). Failure to consider general personality disorder pathology could, thus, either inflate the observed relation attributable to specific PDs or obscure the extent to which unique features of specific PDs are related to specific SUDs. The purpose of the present research was to use hierarchical factor models to partition personality pathology into common (i.e., a general personality pathology factor) and unique sources in order to estimate general and specific forms of Axis II-Substance Dependence comorbidity.¹

PD Comorbidity With SUDs

The elevated rate of PDs among individuals with SUDs, particularly alcohol use disorders (AUDs), is well established (see Sher & Trull, 2002; Trull, Sher, Minks-Brown, Durbin, & Burr, 2000 for reviews). From a theoretical perspective, understanding PD-SUD comorbidity can inform nosology with respect to the distinctiveness of Axis I versus Axis II pathology (Widiger & Shea, 1991), the number of basic dimensions of personality

¹We are well aware of the controversy in the use of the term “comorbidity” (e.g., see Lilienfeld, Waldman, & Israel, 1994). We use the term comorbidity primarily because it is familiar to psychopathology researchers and as of yet has not been supplanted (see critiques of Lilienfeld et al.’s position by Spitzer, 1994 and Rutter, 1994; also see Krueger & Markon, 2006). At the same time, we acknowledge that the term “covariation” may in fact be technically more correct because we do not assume that PD or substance dependence diagnoses represent distinct diagnostic entities (or have specific etiologies).

pathology, and their connections to temperament and personality dimensions in the larger literature (e.g., Krueger, 2005; Krueger & Markon, 2006; Krueger, 1999; Krueger, Caspi, Moffitt, Silva, & McGee, 1996; Trull & Sher, 1994; Trull, Waudby, & Sher, 2004). From a clinical perspective, identifying specific PD-SUD relations could guide clinicians to focus on specific PD presentation when determining potential co-occurring SUD diagnosis or risk (Trull, Waudby, & Sher, 2004) or time to remission and prognosis (Zanarini, Frankenburg, Reich, & Silk, 2004).

Specific PDs may show elevated rates of SUDs (overall) compared with others, and some PDs may show higher rates of comorbidity with abuse and/or dependence of specific substances. For example, the high rate of AUDs among individuals with Antisocial and Borderline personality disorders is robust (Sher et al., 1999). This co-occurrence appears related to individual differences in underlying personality dimensions of impulsivity and affective dysregulation (Sher & Trull, 2002; Trull et al., 2000; Siever & Davis, 1991), which serve as shared risk factors for both groups of disorders.

However, some inconsistencies in research findings have challenged conclusions regarding specific PD-SUD relations. For example, a number of other specific PDs, not predominantly rooted in traits of impulsivity and emotional reactivity, also show significant relations with AUDs. Although some studies suggest that only Cluster B PDs (e.g., Histrionic, Borderline) are related to SUDs (Oldham et al., 1995; Skodol, Oldham, & Gallaher, 1999; Johnson et al., 1999), others report that other personality disorders also co-occur with AUDs, such as Dependent PD (Grant, Stinson, et al., 2004; Zimmerman & Coryell, 1989), Paranoid PD (Zimmerman & Coryell, 1989), and Avoidant PD (Sher & Trull, 2002).

With respect to dependence on other, nonalcohol drugs, individuals with Paranoid, Schizotypal, Histrionic, Antisocial, Avoidant, and Borderline PDs show significantly higher rates *compared with individuals with no PD*, while only Antisocial PD retained a significantly higher association with drug use disorders *compared with other PDs* (Zimmerman & Coryell, 1989). However, another study found that only Borderline and Histrionic PDs were related to the probability of having a drug use disorder among a sample referred for PD treatment (Skodol, Oldham, & Gallaher, 1999). More recent research reported that Histrionic PD was the only PD assessed in adolescence that independently predicted mean levels of noncannabis drug use disorder symptoms in 12–25 year olds (Cohen, Chen, Crawford, Brook, & Gordon, 2007). Finally, analyses from the National Comorbidity Survey Replication study (NCS-R; Lenzenweger, Lane, Loranger, & Kessler, 2007) indicated that Cluster B personality disorders are most highly associated with past-year substance use disorder, especially alcohol and drug use disorder.

Axis II Comorbidity

By definition, personality disorders share certain features associated with maladaptive patterns of cognition, emotion, and behavior (American Psychiatric Association, 2000), and these patterns are commonly manifested in interpersonal relationships. A major issue for the classification of personality disorders is the high degree of overlap among these conditions (Widiger & Trull, 2007). Studies consistently report high comorbidity among PD diagnoses

(Bell & Jackson, 1992; Cohen et al., 2007; Dahl, 1986; Lenzenweger et al., 2007; Morey, 1988; McGlashan, Grilo, & Skodol, 2000; Oldham et al., 1992; Westen & Shedler, 1999; Zimmerman & Coryell, 1989), and research on the psychometric and diagnostic properties of specific *Diagnostic and Statistical Manual of Mental Disorders* (DSM) criteria also shows considerable overlap among PD symptoms (Widiger & Trull, 1998, 2007). As further evidence of comorbidity among personality disorders, patients who meet criteria for one personality disorder typically also meet criteria for multiple PD diagnoses (e.g., Zimmerman, Chelminski, & Young, 2008). One study even reported that patients with one PD met criteria for *at least four additional PD diagnoses* (Westen & Shedler, 1999). Furthermore, PDs which are highly comorbid with each other also show high rates of comorbidity with SUDs (Grant, Stinson, Dawson, Chou, & Ruan, 2005; Sher et al., 1999; Zimmerman & Coryell, 1989). Together, these findings raise the possibility that SUD-Axis II comorbidity may be highly nonspecific and reflect shared PD symptomatology.

Extensive comorbidity among PDs poses a major problem for characterizing PD-SUD comorbidity. One complication is the difficulty determining when an observed co-occurrence between a single PD and SUDs is attributable to characteristics common to most Axis II pathology, as opposed to features specific to a particular PD. This may be partly responsible for the high rates of Axis I comorbidity across all PDs, and for high rates of co-occurrences with SUDs across a variety of PDs (i.e., Clusters A and C; Cohen et al., 2007). Findings which indicate that controlling for multiple PDs results in nonsignificant associations between PDs and SUDs highlight the need to use analytic approaches that disaggregate common and unique sources of PD pathology.

Distinguishing Common and Unique Sources of Variation: A Factor Analytic Approach

Despite this acknowledged overlap between PDs, investigators have not differentiated comorbidity between PDs and SUDs that is (1) attributable to shared common features of personality disorders in general; and (2) attributable to specific or unique features of individual PDs. One method for identifying those features of similar constructs that are unique, while simultaneously measuring those features that are shared, is through hierarchical factor models, also known as the Schmid-Leiman transformation or the bi-factor model (Loehlin, 2004; Schmid & Leiman, 1957; Holzinger & Swineford, 1937). Within this approach, each indicator loads directly onto both a general latent variable as well as a specific latent variable. One latent variable is modeled to account for the shared features among all indicators, leaving only the unique, residual variance that is shared among the indicators to be modeled by specific constructs. This technique has been used in situations where both shared and unique features of constructs are of interest, such as age-associated changes in both general and specific cognitive abilities (Schmiedek & Li, 2004).

In this study, we examined specific PD-substance dependence comorbidity in a population-based sample while controlling for general PD symptomatology. This was accomplished by applying hierarchical structural equation modeling to data from a population-based data set assessing Axis I and II disorders, the *National Epidemiologic Survey on Alcohol and Related Conditions* (NESARC). We predicted that although a general personality disorder factor

would be significantly related to a range of substance dependence diagnoses in the population, specific personality disorder pathology would show differential patterns of relationship to substance dependence—in particular, Cluster B PDs to substance dependence diagnoses.

Method

Data used for the present analysis were selected from Waves 1 and 2 of the National Institute on Alcohol Abuse and Alcoholism (NIAAA) National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). The NESARC is a nationally representative, face-to-face survey that evaluated mental health in the civilian, non-institutionalized population of the United States including citizens residing in Hawaii and Alaska (Grant, Kaplan, Shepard, & Moore, 2003). Participants were sampled according to 2000/2001 census data using stratification on important demographic and population features at the county level, and the data were subsequently weighted accordingly. Weighted data were adjusted to be representative of the United States population on the basis of age, sex, race, ethnicity and region of the country. To ensure adequate inclusion of underrepresented groups, the NESARC oversampled Black and Hispanic individuals, as well as young adults aged 18–24.

Data were collected by 1800 trained interviewers using laptop interview software (see Grant, Kaplan, et al., 2003). The first wave of NESARC was conducted in 2001–2002. Among the 43,093 respondents who participated in the Wave 1 interview, 39,959 persons were eligible for a NESARC Wave 2 interview, and among these, 34,653 Wave 2 interviews were completed in 2004–2005 (Grant & Kaplan, 2005). Data from participants interviewed both at Waves 1 and 2 were analyzed in the present study. Of these, 20,089 (58%) were female.

Personality Disorder Diagnoses

Lifetime personality disorder (PD) symptoms and diagnoses were determined using the NIAAA *Alcohol Use Disorder and Associated Disabilities Interview Schedule- DSM-IV Version* (AUDADIS-IV). Interview questions were keyed to *DSM-IV* PD criteria and asked respondents about long-term patterns of cognition, emotional experience, and behavior that were context-free and not limited to periods of depression, mania, anxiety, heavy drinking, medication or drug influence, or withdrawal (Grant, Hasin et al., 2004). Wave 1 of the NESARC included lifetime measurement of Antisocial, Avoidant, Dependent, Histrionic, Obsessive–compulsive, Paranoid, and Schizoid personality disorders. Borderline, Narcissistic, and Schizotypal PD were assessed at Wave 2, and Antisocial PD was assessed a second time (incorporating Wave 1 diagnostic information; note, however, that we used the Wave 1 antisocial diagnostic information in our analyses). Diagnostic criteria for each PD were measured by asking participants whether each *DSM-IV* PD criterion, as assessed by at least one interview question, was (a) descriptive of the participant (0 = no, 1 = yes), and (b) a cause of problems at work/school or in personal relationships (0 = no, 1 = yes). In the present study, personality disorder diagnoses were assigned to those individuals meeting the requisite number of criteria associated with significant distress, impairment, or dysfunction for the disorder (e.g., four or more of the seven diagnostic criteria for Paranoid

PD). These diagnostic rules produced lower prevalence rates of PDs, as well as higher comorbidity with substance dependence, than the original AUDADIS diagnostic rules for PDs that only required at least one of the requisite number of personality disorder symptoms to have caused social or occupational dysfunction (see Trull, Jahng, Tomko, Wood, & Sher, 2010). Our more conservative PD diagnostic decision rules did produce prevalence estimates roughly similar to those obtained from other nationally representative studies conducted in Great Britain (Coid, Yang, Tyrer, Roberts, & Ullrich, 2006) and in the United States (NCS-R; Lenzenweger et al., 2007).

Previous reports indicate that 10-week test–retest reliability estimates for the seven Wave 1 PD diagnoses were fair to good (Grant, Dawson, et al., 2003). Kappa coefficients for diagnoses ranged from .40 to .67, and intraclass correlations (ICCs) for PD symptom counts ranged from .55 to .79. Concerning the three Wave 2 PDs (Borderline, Narcissistic, Schizotypal), six-week test-retest reliabilities for diagnoses ranged from .67–.71 (kappas) and for symptom counts ranged from .71–.75 (ICCs) (Ruan et al., 2008).

Substance Dependence Diagnoses

Substance dependence diagnoses examined in the present study included *lifetime* measures of alcohol dependence, nicotine dependence, and other drug dependence at Wave 2 (which incorporated Wave 1 diagnostic information). A diagnosis of dependence for each substance required participants to endorse at least three of the seven *DSM–IV* criteria occurring in any 12-month period). Reliability for alcohol and drug diagnoses using the AUDADIS has been examined among both a general population (see Grant, Harford, et al., 2007) and a clinical sample (Hasin, Carpenter, McCloud, Smith, & Grant, 1997), and this interview has received considerable support as a reliable structured interview for substance use disorders (Grant, Dawson, et al., 2003). Specifically, published test–retest reliability coefficients for the AUDADIS lifetime substance dependence diagnoses examined in this study were .60, .66, and .70 for tobacco dependence, drug dependence, and alcohol dependence, respectively (Grant, Dawson et al., 2003; Grant, Harford et al., 1995). As reported by NESARC investigators (Grant, Dawson, et al., 2003; Grant & Kaplan, 2005), missing data for demographic variables were handled by imputation based on estimates derived from nonmissing values for each participant.

We focused on dependence, rather than abuse, in the present study because there is increasing criticism of the construct of abuse as a distinct diagnosis and because dependence tends to be more chronic and strongly associated with other Axis I and Axis II disorders than is abuse (e.g., Vergés, Steinley, Trull, & Sher, 2010). Moreover, in contrast to the abuse construct, the construct of dependence reflects a syndrome of co-occurring physiological and psychological symptoms directly arising from excessive substance use, as compared with abuse, which reflects social/environmental consequences which have been shown to predominate in certain subgroups (e.g., men, Caucasian ethnicity) and is not a “syndrome” of its requirement of only one item/criterion (Harford, Grant, Yi, & Chen, 2005; Saha, Chou, & Grant, 2006).

Lifetime substance dependence, rather than current substance dependence, was examined: (1) to minimize the problem of age gradients with specific disorders, (2) to minimize

confounding “time of measurement” method effects associated with the NESARC’s design which assessed different PDs at different waves (see below), (3) to avoid confounding of occurrence and persistence of diagnosis by overlooking those who have recovered from disorder, (4) to be consistent with lifetime assessment of PD, and (5) because we are examining their associations from a dimensional perspective. From this perspective, underlying dimensions contributing to both substance dependence and Axis II disorders show stability over time, even though at times they may not present in the severity of a diagnosis. Therefore, rather than requiring co-occurrence at a specific point in time, our analyses address underlying dimensions contributing to co-occurrences within the same individuals that could occur at the same or different points in time.

The NESARC’s design with respect to the assessment of co-morbidity using *past-year* diagnoses was deemed to be potentially problematic because seven PDs were assessed at baseline and three PDs were assessed at follow-up; differential patterns of comorbidity could be attributed to a method factor (i.e., measure-occasion specific effects) rather than a particular PD construct. Lifetime assessments were used and adjustments for time of measurement effects were made in order to minimize this potential problem (i.e., modeling correlated errors for PDs assessed at Wave 2).

Results²

Preliminary Analysis

Table 1 shows estimates of lifetime prevalence rates of personality disorders and substance dependence. Prevalence rates of PDs ranged from 0.2% (Dependent PD) to 3.7% (Antisocial PD). Rates associated with alcohol, nicotine, and other drug dependence were 15.2%, 23.1%, and 3.4%, respectively. Table 2 presents estimated tetrachoric correlations among substance dependence and PD diagnoses. As expected, strong correlations were found among substance dependence diagnoses (.51, .53, and .65). PD diagnoses also showed high intercorrelations (.26–.84, $M = .56$, $SD = .16$). Within-cluster correlations were only slightly higher (.26–.84, $M = .59$, $SD = .15$) than between-cluster correlations (.29–.81, $M = .54$, $SD = .16$). PD diagnoses assessed at only Wave 2 (Schizotypal, Borderline, and Narcissistic) were also highly correlated to each other (.81, .76, and .76).

Hierarchical Factor Structure of PDs

High correlations indicative of comorbidity among PD diagnoses may be accounted for by underlying factors associated with personality dysfunction. Using the tetrachoric correlation matrix presented in Table 2, we fit a hierarchical factor model for PD diagnoses. Following the *DSM-IV* PD cluster organization (i.e., Cluster A: odd-eccentric, Cluster B: dramatic-emotional, and Cluster C: anxious-fearful), one may assume three lower-order factors corresponding to the three clusters, as well as a higher-order factor that accounts for correlations among the lower-order factors.³ Alternatively, we used a confirmatory factor analogue of the Schmid-Leiman representation of the hierarchical factor model (Schmid &

²All models in this section were estimated by weighted least squares with mean- and variance adjusted (WLSMV). The degrees of freedom for WLSMV were estimated according to a formula given in the Mplus Technical Appendixes at www.statmodel.com (Asparouhov & Muthen, 2006).

Leiman, 1957). In the present case, a general PD factor accounts for omnibus associations across all 10 PDs, and three lower order factors account for residual correlations among PDs within each cluster (see Figure 1). Because three PDs (i.e., Schizotypal, Borderline, and Narcissistic PD) were assessed at Wave 2, correlations between residuals of these PDs were estimated to explain method variance associated with the Wave 2 assessment. Results of the four-factor hierarchical model of PDs are presented in Table 3.⁴ Although the χ^2 statistic for the model was statistically significant ($\chi^2_{(14)}=34.34, p < .01$), as is often true in large samples, the model still provided an excellent fit to the data (CFI = .996, TLI = .996, RMSEA = .006). Factor loadings from the general PD factor to individual PDs were all significant (all $ps < .001$), as were factor loadings from the residual Cluster B PD factor (all $ps < .05$). However, factor loadings from the residual Cluster A and C PD factors were not significant (all $ps > .50$).⁵

Taken together, these findings suggest that once the influence of a general PD factor on the individual PD diagnoses is controlled, residual factors do not explain a statistically significant amount of covariation except for the residual Cluster B PD factor. Because the factor loadings for the clusters A and C were not significant, we fit a reduced, two-factor model consisting only of a general PD factor and the residual Cluster B PD factor (see Table 3). Although the χ^2 difference test was significant between the four-factor model and the two-factor model ($\Delta\chi^2_{(5)}=13.39, p < .05$),⁶ the two-factor model showed as good a model fit ($\chi^2_{(17)}=42.63, p < .001$, CFI = .995, TLI = .996, RMSEA = .007) as the four-factor model. Consistent with the initial model, factor loadings from the two retained factors were significant (all $ps < .05$) in the reduced model. Based on substantive considerations noted above regarding the presence of a general PD dimension and the importance of Cluster B disorders in SUDs, as well as the model fit of the reduced model and pattern of statistical significance in estimated loadings for the full model (as well as the law of parsimony), we

³Confirmatory factor analyses testing the fit of *DSM-IV* cluster structure to these data (and including correlated errors for those PDs measured at Wave 2) revealed that while the model fit the data well ($\chi^2_{(15)}=39.30, p < .01$; CFI = .995, TLI = .995, RMSEA = .007), latent cluster factors were highly correlated with each other (Cluster A-Cluster B, $r = .90$; Cluster A-Cluster C, $r = .98$; Cluster B-Cluster C, $r = .89$). This strongly suggests that a more general latent factor, indicated by all three clusters, can account for most of the covariation among the PDs.

⁴For single-group analyses, delta parameterization was used to model continuous latent response variables of observed categorical outcomes; that is, the variance of the latent response variables of categorical PD (and substance dependence) variables was scaled to 1. As a result, delta parameterization produced identical estimates of factor loadings and path coefficients for unstandardized and standardized solutions.

⁵As one reviewer noted, the standard errors for estimates associated with both the Cluster A and Cluster C residual factors are large compared with those for the General PD factor and Cluster B residual factor (see Table 3). We considered the possibility that these might indicate identification problems (specifically, empirical underidentification) with the four factor model. However, we do not think this was the case for several reasons: (1) our models did converge, and the factor loadings associated with the General and Cluster B factors were consistent across all models; (2) descriptions of statistical estimation from measure and function theory suggest that standard errors of (near-)zero loadings could well be large, and larger standard errors result from a decrease in estimable precision associated with the nonsignificant multiple factor loadings; and (3) our Monte Carlo simulations of a structural model consisting of a single factor (with eight variables defining this single factor) as well as an additional superfluous factor (defined by four of the variables) using 1000 replications and specifying a sample size of 5000 observations for each replication revealed that the average standard error for the general factor was .0068, whereas the average standard error associated with the (nonsignificant) factor loadings of the nonexistent factor was .30. These results are consistent with the position that superfluous factors, in general, will have large standard errors associated with the loadings.

⁶For model comparisons, the DIFFTEST option in Mplus was used to obtain a correct chi-square difference test because the difference in chi-square values for the two nested models using the WLSMV chi-square values is not distributed as chi-square.

concluded that patterns of comorbidity in PDs (expressed as correlations) are best explained by a general PD factor and residual Cluster B PD factor.

Path Models Between PD Factors and Substance Dependence

In addition to the high correlations among substance dependence diagnoses and among PD diagnoses, notable correlations between individual substance dependence diagnoses and PDs were observed (see Table 2). Specifically, individual PDs correlated with alcohol dependence (r ranging from .15 to .50, $M = .31$, $SD = .10$), nicotine dependence (r ranging from .18 to .45; $M = .29$, $SD = .08$), as well as other drug dependence (ranging from .31 to .62; $M = .44$, $SD = .09$). Although these correlations indicate significant comorbidity between substance dependence and individual PDs, a more parsimonious comorbidity model of PD with substance use disorders is possible if associations are estimated between substance dependence diagnoses and the reduced PD model containing the general and residual Cluster B PD factors as shown in Figure 2. In addition to the model's parsimony, estimated associations between PD factors and individual substance use diagnoses are not attenuated by measurement error associated with individual PD diagnoses.⁷

The fit of the comorbidity of substance dependence with the general and residual Cluster B PD factors was excellent ($\chi^2_{(24)} = 68.17$, $p < .001$, CFI = .994, TLI = .995, RMSEA = .007). As for the model presented in Figure 2, factor loadings associated with the two PD factors were significant (all $ps < .05$; for ease of presentation, not presented in Figure 2). The general PD factor and the residual Cluster B PD factor were significantly associated with each substance dependence diagnosis (all $ps < .001$). Model comparison between the model with and without equality constraints of path coefficients of the two factors to each substance dependence revealed that the general PD factor had lower effects on substance dependence diagnoses than did the residual Cluster B PD factor ($\Delta\chi^2_{(3)} = 54.75$, $p < .001$). Of equal interest, the residual covariances among substance dependence diagnoses were not significant (all $ps > .10$) after controlling the effects of the two PD factors.⁸ This finding suggests that the observed comorbidity among substance dependence disorders potentially can be explained by their common comorbidity with the two PD factors. This finding is particularly impressive, given that the observed zero-order correlations among different forms of substance dependence (shown in Table 2) were substantial (all $rs > .50$ with all $ps < .001$).

⁷Before we fit the model shown in Figure 2, we estimated individual paths from the residuals of individual PDs to substance dependence diagnoses after controlling the effect of the general PD factor on the substance dependence diagnoses. Because all the paths from the residual PDs cannot be simultaneously estimated with the factor loadings and paths associated with the general PD factor (i.e., an underidentified model), we first fit a model without residual PD paths and then estimated those paths while the factor loadings and paths from the general PD factor were fixed as estimated from the first model. All paths from the general PD factor were significant, but paths from residual PDs were not significant except for Antisocial and Borderline PDs to all substance dependence diagnoses (all paths positive), Dependence PD to alcohol dependence (negative), Avoidant PD to nicotine dependence (negative), and Obsessive-compulsive PD to nicotine and other drug dependence (both negative). These results suggest that much of co-occurrence between substance dependence diagnoses and individual PDs is attributable to the general PD factor, but Antisocial and Borderline PDs (both Cluster B PDs) uniquely predict substance dependence over and beyond the general PD factor.

⁸When the paths from the residual Cluster B factor to substance dependence diagnoses were omitted from the model, the residual covariances among substance dependence diagnoses remained significant (partial rs for alcohol dependence with nicotine dependence, alcohol dependence with other drug dependence, and nicotine dependence with other drug dependence were .38, .49, and .37, respectively, with $ps < .001$).

We also investigated whether the estimated path coefficients between the two PD factors and substance dependence diagnoses varied as a function of sex and age. Age was dichotomized such that Participants 29 years old or younger at wave 1 were defined as a *younger group* ($n = 6,719$, 19.4%) and those who were 30 years old or older were defined as an *older group* ($n = 27,934$, 80.6%).⁹ Tables 4 and 5 present the results of path models by these sex and age groups, respectively. All estimated paths remained significant for males and females as well as for younger versus older participants (all $ps < .05$). Path coefficients were not significantly different between males and females ($\Delta\chi^2_{(7)} = 6.81$, $p = .45$) but were significantly different between the younger and older group ($\Delta\chi^2_{(6)} = 20.30$, $p < .01$). The effects of the PD factors on each substance dependence diagnosis were stronger for participants who were 30 years old or older at Wave 1 than for their younger counterparts. This was particularly true for the effects of the residual Cluster B PD factor. This suggests that PD and/or substance dependence diagnoses in the younger group may be developmentally limited in nature or that substance dependence may be more strongly influenced by the general PD factors for older participants.¹⁰

Discussion

Our results revealed a strong general PD factor that spans each of the 10 *DSM-IV* PD diagnoses and a strong general association between this general PD factor and substance dependence disorders. Concerning the former, it is noteworthy that each *DSM-IV* PD loaded strongly and significantly on the General PD factor representing common shared variance across all PD diagnoses. The findings equally highlight the importance of controlling for Axis II comorbidity when examining specific PD-substance dependence associations. These findings provide an important context for interpreting previous studies of SUD-PD comorbidity which typically report positive associations across *all* PDs and SUDs. Likewise, in almost every case in the present study, the tetrachoric correlation between each PD diagnosis and each form of substance dependence was significant, positive, and of moderate to large magnitude (see Table 2). For example, the correlations between Antisocial PD and the three substance dependence diagnoses ranged from .45 to .62. Exceptions to this general pattern were the small to moderate associations between Dependent PD and alcohol dependence as well as between Obsessive-compulsive PD and nicotine dependence. Although these latter findings contrast with those from other studies (Sher & Trull, 2002; Oldham et al., 1995), the general pattern of significant bivariate correlations between PD and SUD diagnoses is consistent with previous studies and the standard approach to estimating PD/SUD relations. Furthermore, we extracted a common, general factor from the

⁹We chose to stratify the sample into an “older” and “younger” adult group recognizing that the peak hazard (and peak prevalence) for substance dependence is in late adolescence and emerging adulthood. Further, there is relatively little new onset of SUDs in the fourth decade of life and beyond. Multiple group path models were estimated by WLSMV with theta parameterization because delta parameterization produced improper solutions. Residual variances of latent response variables of observed categorical PD and substance dependence variables were set to 1 for females and younger group and were estimated for males and older group. Unstandardized and standardized solutions for path coefficients are not identical in this case, whereas those for residual covariances are identical.

¹⁰To examine whether we could be reasonably confident that our results were not primarily attributable to a common Wave 2 time of measurement effect on substance dependence disorders, we reran our major analyses using Wave 1 lifetime substance dependence diagnoses. We obtained almost exactly the same results in terms of patterns of significant covariation and fit of the models. This suggests that our findings are not primarily a function of Wave 2 dependence diagnoses.

DSM-IV personality disorders, and this factor was significantly related to all forms of substance dependence. Therefore, it seems reasonable to conclude that a common factor underlying personality disorders accounts for much of the comorbidity between Axis II personality disorders and substance dependence.

This general finding that personality pathology appears to statistically “explain” comorbidity among different forms of substance dependence is consistent with the finding that different forms of substance dependence have similar personality correlates, especially traits associated with behavioral undercontrol and negative affectivity (e.g., Grekin, Sher, & Wood, 2006; Littlefield & Sher, 2010; Sher et al., 1999; Sher, Bartholow, & Wood, 2000). To the extent that personality disorders represent extreme manifestations of personality variation (e.g., Widiger & Trull, 2007), the general form of our findings is not surprising. However, what is surprising is the magnitude of this effect, as personality pathology appeared to account for the overwhelming amount of observed covariation among alcohol, drug, and tobacco dependence. (We note that these findings are also consistent with the conceptualizations of the externalizing spectrum which includes both normal variation in personality traits as well as substance use disorders; Krueger, Markon, Patrick, Benning, & Kramer, 2007).

Our study also sought to identify unique relations between various forms of personality pathology (e.g., the *DSM-IV* PD clusters) and substance dependence disorders. A unique feature of our study was the use of a hierarchical factor approach to disaggregate general and specific sources of Axis II pathology and estimate their relations to substance dependence diagnoses. In addition to the general factor underlying PD diagnoses, the hierarchical factor analyses revealed a residual Cluster B PD factor. After controlling for the general PD factor, this Cluster B PD factor was positively and significantly associated with all substance dependence diagnoses among both men and women, and among both younger and older participants. This suggests that a factor common to the Cluster B disorders but independent of other PDs is associated with substance dependence. It is also noteworthy that this residual Cluster B factor was more highly related to the substance dependence diagnoses than was the General PD factor.

We posit that the unique residual factor of Cluster B disorders that is significantly related to various forms of substance dependence concerns the personality trait of impulsivity. In contrast to general negative affectivity which appears to underlie many of the PDs, impulsivity seems more specific to Cluster B PDs and especially to Antisocial and Borderline PD. Consistent with this interpretation, impulsivity or disinhibition has been implicated in many forms of substance use disorder, and this trait or dimension of psychopathology characterizes many forms of externalizing disorders including substance use disorders, Antisocial PD, and Borderline PD (Krueger, 1999; Bornoalova, Lejuez, Daughters, Rosenthal, & Lynch, 2005; Bornoalova, Fishman, Strong, Kruglanski, & Lejuez, 2008; Hicks, Markon, Patrick, Kreuger, & Newman, 2004).

With respect to understanding impulsivity and how it could relate to PD-SUD comorbidity, two issues must be considered. The first is that impulsivity is not a single trait or construct and can include a number of related but distinct facets such as lack of planning, lack of

persistence, inability to inhibit a prepotent response, the tendency to act rashly when distressed, the tendency to act rashly when experiencing positive emotions, preference for immediate versus delayed rewards (delay discounting), or diminished resistance to distraction and novelty seeking (Bickel et al., 2007; Cyders & Smith, 2008; Dick et al., 2010; Dougherty, Mathias, Marsh, & Jagar, 2005; Eriksen & Eriksen, 1974; Logan, 1994; Smith, Fischer, Cyders, Annus, & Spillane, 2007). Thus, it seems imperative to move beyond the general observation that impulsivity explains the comorbidity between Cluster B PDs and SUDs in order to conduct research aimed at characterizing specific impulsivity facets that might be common to both sets of disorders.

The second issue is that personality traits associated with impulsivity (e.g., conscientiousness) are not fixed and tend to change over the course of development (Roberts, Walton, & Viechtbauer, 2006). Indeed, there are dramatic mean changes in personality traits over the entire life span and, so, we would expect to see both the prevalence of PDs and SUDs change together over time with decreases in SUDs and PDs as individuals age. The data on the prevalence of SUDs are unambiguous in this regard, although the steepness of the age gradient varies across substances (Agrawal, Lynskey, Madden, Bucholz, & Heath, 2008; Stinson et al., 2005). The data on PDs suggest a similar phenomenon with the clinical literature describing how Antisocial PD and psychopathy “burn out” in mid-adulthood (Harpur & Hare, 1994; Regier et al., 1988; Robins, 1966) and with empirical data indicating that the peak prevalence for borderline, antisocial, narcissistic, and histrionic traits is in early adulthood (Ullrich & Coid, 2009). Recent research has shown that “maturing out” of alcohol problems was associated with reductions in impulsivity (assessed with a measure that largely reflected lack of deliberation) in the third decade of life. Findings like these suggest that the nexus of personality, PDs, and SUDs is a dynamic one that needs to take into account developmental processes (Littlefield, Sher, & Wood, 2009).

Similar to findings with alcohol dependence, specific associations between both nicotine and drug dependence and the residual Cluster B PD factor were revealed. Although on the surface the link between nicotine dependence and impulsivity may not appear to be straightforward, a number of studies have reported associations between this personality trait and smoking initiation and maintenance (Bickel, Odum, & Madden, 1999; Doran, Spring, McChargue, Pergadia, & Richmond, 2004; Perkins et al., 2008).

We wish to point out that our approach to analysis of PD/SUD comorbidity leads to different conclusions than traditional analytic approaches which fail to decompose common and specific aspects of personality pathology. For example, previous analyses of NESARC data reported that nicotine dependence is associated with Cluster C PDs (Grant, Hasin, Chou, Stinson, & Dawson, 2004). For drug dependence, once again, the residual Cluster B factor remained positively associated with this diagnosis after controlling for general PD symptomatology. Our finding that there is a positive and moderately strong relation between the residual Cluster B PD factor and dependence on drugs other than alcohol and tobacco is consistent with previous studies as well as previous analyses of NESARC data showing high comorbidity between Cluster B PDs and DUDs (Trull et al., 2004; Skodol, Oldham, & Gallaher, 1999; Zimmerman & Coryell, 1989; Cohen et al., 2007; Grant, Stinson, et al.,

2005). However, our hierarchical modeling approach does not suggest a unique relation between drug use disorders and Cluster C disorders (e.g., Dependent PD) as has been reported using the NESARC (Grant, Stinson, et al., 2005).

We emphasize that merely showing a statistical association between PDs or even traits related to impulsivity does not necessarily identify a specific etiological process. For example, although traits related to impulsivity could be viewed as contributing to failed quit attempts (i.e., inability to abstain) or using a substance in larger amounts or longer than intended (i.e., impaired control), they could also be associated with social processes (e.g., deviant peer group influences) and psychopharmacological processes (e.g., enhanced drug effects) (Sher, 1991; Sher & Grekin, 2007).

Is Comorbidity Among SUDs Attributable to SUD Comorbidity With PDs?

As noted above, one of the most striking findings to emerge from our analyses is that the residual (i.e., partial) correlations among SUDs were nonsignificant when PD variance was partialled out. The only exception to this general finding was that in the younger (<age 30 at baseline) cohort, correlations were greatly attenuated but still significant. This suggests that PDs may be largely responsible for SUD comorbidity, especially in mid- to later adulthood. Although we believe our findings suggest that much of SUD comorbidity could be attributable to shared common personality pathology, there are a number of types of data suggesting that some drug interactions, such as alcohol cross-tolerance with nicotine (Funk, Marinelli, & Lê, 2006) or unique psychoactive metabolites of concurrently used substances (e.g., coca-ethylene from combined alcohol and cocaine use) could account for some degree of comorbidity (McCance, Price, Kosten, & Jatlow, 1995). Additionally, chronic substance dependence could result in affective dysregulation, thus contributing to personality pathology (Sher & Grekin, 2007), and the current study is silent with respect to numerous alternative mechanisms that could be important in understanding PD/SUD pathology.

Toward a Hierarchical Model of Personality Disorder

A number of influential researchers have questioned the validity of DSM's approach to personality disorder symptomatology on the basis of the excessively high comorbidity among these entities and the fact that the majority of individuals with a personality disorder are found to have multiple personality disorders when assessed using structured instruments (McGlashan et al., 2005; Zimmerman et al., 2008; Zimmerman & Coryell, 1989; Westen & Shedler, 1999; Widiger, Livesley, & Clark, 2009). Clearly, the existing approach to PD nosology fails to "carve nature at her joints" and cries out for alternative approaches to reconceptualizing personality disorders. Many have heeded the call and proposed dimensional alternatives, such as frameworks based on the Five Factor Model (Widiger, Costa, & McCrae, 2002; Widiger & Trull, 2007), or using quantitative systems that represent not only symptom presence or absence but also indicate symptom severity (Oldham & Skodol, 2000; Brown & Barlow, 2005; Tyrer, 2005). Our findings suggest yet another consideration, involving quantification of the severity of general personality pathology distinct from more specific residual symptomatology, wherein estimation of Axis I/Axis II symptomatology occurs at two levels. Indeed, this is likely an oversimplification because Axis I pathology itself appears to be factorially complex (Brown & Barlow, 2009;

Krueger & Markon, 2006; Krueger, 1999), and further investigation of more complex models is warranted. Nevertheless, our findings are clear in that the standard approach to studying PDs in isolation from each other is strongly misspecified in a statistical sense and likely to lead to errors of attribution. Although a similar argument can be made for Axis I pathology (Brown & Barlow, 2009; Krueger & Markon, 2006), the situation would appear to be much more severe when personality pathology is considered.

The Nature of General Personality Disorder Pathology

DSM-IV-TR defines personality disorder as inflexible and mal-adaptive personality traits that are exhibited in a wide range of personal and interpersonal contexts (American Psychiatric Association, 2000). One prominent model of personality disorder is that of Livesley (1998) who views personality pathology as an adaptive failure to achieve life tasks, traceable directly to personality traits (see also Livesley & Jang, 2000). This theory proposes deficits in three major areas: (1) the self system, (2) the interpersonal system, and (3) societal relationships. Therefore, what personality disorders have in common is this general adaptive failure, the nature of which depends on the maladaptive traits in question.

The exact nature of the strong, general personality disorder factor we extracted cannot be determined with certainty, but several possibilities exist. First, the first factor that often appears in factor analyses of personality, personality disorder, or personality pathology scale scores is that of negative affectivity, neuroticism, or emotional dysregulation (Livesley & Jang, 2000). There is little argument that such a broad factor can characterize many of the symptoms of personality disorder. In our study, however, this may not be the case for several reasons. Specifically, because negative affectivity is shared by most if not all forms of psychopathology, the residual variance in personality disorder scores (after partialing out negative affectivity) would be *less* likely to relate to substance dependence disorders.

A second possibility is that this general PD factor represents a lack of insight or a factor reflecting the ego-syntonic nature of personality disorder (American Psychiatric Association, 2000). Although we agree that many personality disorders seem to be characterized by a remarkable lack of insight, this explanation for the general factor we extracted seems less plausible because PD diagnoses that load highly on this general factor do not *require* a lack of insight, and some of the residual Cluster B-substance dependence relations we observed might not be present if this factor was first partialled out of the PD scores.

A third possibility is that the general personality disorder factor reflects interpersonal dysfunction, and examination of the highest loading PDs on the general PD factor (i.e., Paranoid, Schizoid, Avoidant, and Dependent PD) is consistent with this interpretation. These disorders' symptoms have in common interpersonal distance or interpersonal problems that are created by maladaptive beliefs about oneself or others. This general feature of personality disorder corresponds with Livesley's "adaptive failure" in interpersonal relations. It is widely acknowledged that Axis I disorders, like those we examined in this study, have interpersonal sequelae as well. Substance dependence is accompanied by impaired interpersonal functioning, and this impairment is likely

attributable to the impact that the symptoms of these disorders has on the individual and on those around him or her.

In summary, although we cannot specify exactly the nature of this general personality disorder factor, one that clearly drives most of the positive association we see between PDs and substance dependence, our results seem most consistent with characterizing this factor as *interpersonal dysfunction*. By controlling for this factor, we were then able to observe some interesting patterns of association between specific Cluster B PD residual variance and substance dependence disorders. Our results indicate that a failure to consider this general PD factor can lead to potential misunderstandings of the nature of certain PD and substance dependence comorbidities. These misunderstandings can, in turn, misinform both theories of etiology as well as treatment approaches that are likely to be successful. For example, Cluster A and Cluster C PDs do not appear to be associated specifically to substance dependence disorders, once the general personality disorder dysfunction (or interpersonal dysfunction, in our view) is taken into account.

The finding that Cluster B PDs were the only disorders that were positively associated with SUDs after general PD psychopathology was controlled is consistent with evidence that these disorders are those that share significant externalizing (Krueger et al., 2002; Krueger, 2005; Krueger & Markon, 2006) or impulsivity/behavioral disinhibition (Trull & Sher, 1994) in contrast to Cluster A and Cluster C disorders. However, we wish to emphasize that our findings should not be interpreted as suggesting that Cluster A and Cluster C disorders are not clinically relevant to SUDs but that their relevance is attributable to common personality pathology and not pathology unique to those specific conditions.

Limitations

Although the present study has a number of strengths already mentioned, there are a few important limitations. First, not all of the *DSM-IV* personality disorders were included in the first wave of NESARC. As noted in the Method section, this necessitated modeling common method variance to account for the variation in assessment of these disorders. Second, because the NESARC relied upon lay interviewers, the interview that was used (the AUDADIS) is heavily reliant upon self-report of *symptoms* (rather than interviewer judgment and observed *signs* of pathology) and did not include extensive follow-up questions to determine the meaning of symptom endorsement. This raises the possibility that the AUDADIS may be highly reliable but not necessarily as valid as interviews conducted by experienced clinical psychologists or psychiatrists. On the other hand, it is worth noting that the agreement among Axis II interviews, even when administered by experienced clinicians, is modest at best (e.g., Skodol, Oldham, Rosnick, Kellman, & Hyler, 1991; Westen & Shedler, 1999). More research is needed to examine the correspondence of the AUDADIS personality disorders interview section with existing Axis II interviews (e.g., SCID-II, SIDP-IV). Third, the NESARC study assessed lifetime diagnoses of personality disorder diagnoses, compared to some of the other comorbidity studies, such as NCS-R. In addition, we examined lifetime diagnoses of substance dependence. One benefit of analyzing lifetime diagnoses, however, is that many of the disorders we investigated have strong age gradients, and we believed that assessing the relations between trait-like

operationalizations of PDs and current (past 12 month) Axis I diagnoses might present interpretative difficulties. Despite these limitations, we feel that the present study makes important contributions by demonstrating the use of a statistical method for accounting for PD comorbidity and identifying important specific PD-Axis I associations as avenues for future research.

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Figure 1.
Hierarchical factor model of personality disorders.

Figure 2.
Standardized path coefficients for PD factors with substance dependence diagnoses
(numbers in parentheses denote standard errors).

Table 1

Estimated Lifetime Prevalence Rates of Substance Dependence and Personality Disorders

	Wave	Prevalence rate (%)
Substance dependence		
Alcohol	1 & 2	15.2
Nicotine	1 & 2	23.1
Other drug	1 & 2	3.4
Personality disorder		
<i>Cluster A</i>		
Paranoid	1	1.9
Schizoid	1	0.5
Schizotypal	2	0.6
<i>Cluster B</i>		
Antisocial	1 & 2	3.7
Borderline	2	2.7
Histrionic	1	0.3
Narcissistic	2	1.0
<i>Cluster C</i>		
Avoidant	1	1.1
Dependent	1	0.2
Obsessive-compulsive	1	1.9

Table 2

Estimated Tetrachoric Correlations of Substance Dependence and Personality Disorders

	Substance dependence			Cluster A			Personality disorder				Cluster C	
	Alcohol	Nicotine	Other drug	Paranoid	Schizoid	Schizotypal	Antisocial	Borderline	Histrionic	Narcissistic	Avoidant	Dependent
Substance dependence												
Alcohol	—											
Nicotine	0.51	—										
Other drug	0.65	0.53	—									
Personality disorder												
<i>Cluster A</i>												
Paranoid	0.35	0.28	0.45	—								
Schizoid	0.24	0.27	0.41	0.74	—							
Schizotypal	0.30	0.25	0.43	0.57	0.59	—						
<i>Cluster B</i>												
Antisocial	0.50	0.45	0.62	0.45	0.44	0.36	—					
Borderline	0.42	0.36	0.51	0.52	0.48	0.81	0.40	—				
Histrionic	0.35	0.28	0.44	0.70	0.65	0.29	0.50	0.46	—			
Narcissistic	0.28	0.22	0.34	0.43	0.39	0.76	0.36	0.76	0.51	—		
<i>Cluster C</i>												
Avoidant	0.28	0.25	0.47	0.76	0.73	0.51	0.39	0.48	0.56	0.26	—	
Dependent	0.15	0.33	0.46	0.80	0.75	0.49	0.47	0.52	0.74	0.41	0.84	—
Obsessive-compulsive	0.24	0.18	0.31	0.70	0.69	0.46	0.35	0.42	0.68	0.46	0.64	0.71

Note. All $ps < .01$.

Table 3

Factor Loadings for Hierarchical Factor Model of Personality Disorders

Variable	Four-factor model ^a			Two-factor model ^b		
	General PD	Cluster A	Cluster B	Cluster C	General PD	Cluster B
Paranoid	0.90 (0.02)**	-0.06 (0.12)			0.89 (0.01)**	
Schizoid	0.86 (0.03)**	0.32 (0.58)			0.86 (0.03)**	
Schizotypal	0.61 (0.04)**	0.22 (0.36)			0.62 (0.04)**	
Antisocial	0.49 (0.03)**		0.45 (0.17)**		0.49 (0.03)**	0.43 (0.16)**
Borderline	0.56 (0.03)**		0.26 (0.11)*		0.56 (0.03)**	0.27 (0.12)*
Histrionic	0.77 (0.04)**		0.29 (0.11)**		0.78 (0.03)**	0.28 (0.10)**
Narcissistic	0.48 (0.04)**		0.29 (0.13)*		0.48 (0.04)**	0.31 (0.13)*
Avoidant	0.83 (0.02)**			0.28 (0.70)	0.85 (0.02)**	
Dependent	0.89 (0.03)**			0.36 (0.89)	0.93 (0.02)**	
Obsessive-compulsive	0.79 (0.02)**			-0.03 (0.10)	0.78 (0.02)**	

^a $\chi^2_{(14)} = 34.34$, $p < .01$, CFI = .996, TLI = .996, RMSEA = .006.

^b $\chi^2_{(17)} = 42.63$, $p < .001$, CFI = .995, TLI = .996, RMSEA = .007. $\Delta\chi^2_{(5)} = 13.39$, $p < .05$.

* $p < .05$.

** $p < .01$.

Table 4
Path Coefficients of PD Factors With Substance Use Dependence and Partial Correlations by Sex

	Female			Male		
	Alcohol	Nicotine	Other drug	Alcohol	Nicotine	Other drug
Path coefficient ^a						
General PD	0.54 (0.05)**	0.42 (0.05)**	0.99 (0.22)**	0.49 (0.06)**	0.40 (0.04)**	0.87 (0.11)**
	<i>0.40 (0.03)**</i>	<i>0.30 (0.03)**</i>	<i>0.51 (0.04)**</i>	<i>0.35 (0.04)**</i>	<i>0.32 (0.04)**</i>	<i>0.52 (0.04)**</i>
Cluster B	0.73 (0.13)**	0.87 (0.18)**	1.36 (0.46)**	0.87 (0.17)**	0.61 (0.10)**	1.02 (0.24)**
	<i>0.54 (0.06)**</i>	<i>0.63 (0.08)**</i>	<i>0.70 (0.09)**</i>	<i>0.62 (0.07)**</i>	<i>0.49 (0.06)**</i>	<i>0.61 (0.08)**</i>
Residual covariance (partial correlation)						
Alcohol	—			—		
Nicotine	0.09 (0.12)	—		0.13 (0.09)	—	
Other drug	0.20 (0.16)	-0.07 (0.26)	—	0.17 (0.15)	0.05 (0.13)	—

Note. $\chi^2_{(36)} = 93.29$, $p < .001$, CFI = .991, TLI = .991, RMSEA = .01.

^aNonitalicized are unstandardized coefficients and italicized are standardized coefficients. Numbers in parentheses denote standard errors.

* $p < .05$.

** $p < .01$.

Table 5

Path Coefficients of PD Factors With Substance Use Dependence and Partial Correlations by Age Group

	Younger (<30 years old)			Older (30 years or older)		
	Alcohol	Nicotine	Other drug	Alcohol	Nicotine	Other drug
Path coefficient ^a						
General PD	0.30 (0.06) **	0.39 (0.06) **	0.84 (0.11) **	0.62 (0.08) **	0.41 (0.05) **	1.14 (0.44) **
	<i>0.23 (0.05) **</i>	<i>0.32 (0.04) **</i>	<i>0.54 (0.05) **</i>	<i>0.38 (0.02) **</i>	<i>0.28 (0.03) **</i>	<i>0.46 (0.03) **</i>
Cluster B	0.79 (0.16) **	0.61 (0.13) **	0.85 (0.21) **	1.16 (0.25) **	0.94 (0.18) **	1.95 (0.95) *
	<i>0.60 (0.08) **</i>	<i>0.49 (0.08) **</i>	<i>0.54 (0.09) **</i>	<i>0.70 (0.07) **</i>	<i>0.66 (0.07) **</i>	<i>0.79 (0.08) **</i>
Residual covariance (partial correlation)						
Alcohol	—			—		
Nicotine	0.18 (0.10)	—		-0.13 (0.21)	—	
Other drug	0.32 (0.12) **	0.20 (0.11)	—	-0.32 (0.56)	-0.47 (0.54)	—

Note. $\chi^2_{(40)} = 72.77$, $p < .01$, CFI = .995, TLI = .995, RMSEA = .007.

^a Nonitalicized are unstandardized coefficients and italicized are standardized coefficients. Numbers in parentheses denote standard errors.

* $p < .05$.

** $p < .01$.