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The association between personality disorders with alcohol use and misuse: A population-based twin study[^]

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Abstract

Background—A clearer understanding of the etiological overlap between DSM-IV personality disorders (PDs) and alcohol use (AU) and alcohol use disorder (AUD) is needed. To our knowledge, no study has modeled the association between all 10 DSM-IV PDs and lifetime AU and AUD. The aim of the present study is to identify which PDs are most strongly associated with

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Contributors

E.C. Long performed all analyses, prepared all drafts, and prepared all tables. K.S. Kendler, N.A. Gillespie, and T. Reichborn-Kjennerud were joint senior authors. All other co-authors provided critical feedback on drafts of the manuscript. All authors have approved the final article.

Conflicts of Interest

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the phenotypic, genetic, and environmental risks of lifetime AU and AUD, and to determine if these associations are stable across time.

Methods—Participants were Norwegian twins assessed at two waves. At Wave 1, 2,801 twins were assessed for all 10 DSM-IV PD criteria, lifetime AU, and DSM-IV AUD criteria. At Wave 2, six of the 10 PDs were again assessed along with AU and AUD among 2,393 twins. Univariate and multiple logistic regressions were run. Significant predictors were further analyzed using bivariate twin Cholesky decompositions.

Results—Borderline and antisocial PD criteria were the strongest predictors of AU and AUD across the two waves. Despite moderate phenotypic and genetic correlations, genetic variation in these PD criteria explained only 4% and 3% of the risks in AU, and 5% to 10% of the risks in AUD criteria, respectively. At Wave 2, these estimates increased to 8% and 23% for AU, and 17% and 33% for AUD.

Conclusions—Among a large Norwegian twin sample, borderline and antisocial PD criteria were the strongest predictors of the phenotypic and genotypic liability to AU and AUD. This effect remained consistent across time.

Keywords

alcohol use; alcohol use disorder; personality disorders; twin research

1. Introduction

Research has consistently shown that DSM-IV (APA, 2013) personality disorders (PDs) are linked with alcohol use (AU) and alcohol use disorder (AUD; Compton et al., 2005; Grant et al., 2008; Grant et al., 2004; Morgenstern et al., 1997; Skodol et al., 1999; Trull et al., 2000). The two PDs most consistently associated with AUD criteria are antisocial (Compton et al., 2005; Grant et al., 2004; Morgenstern et al., 1997) and borderline (Grant et al., 2008; Morgenstern et al., 1997; Skodol et al., 1999; Trull et al., 2000). Another study has reported significant associations with histrionic and dependent PDs (Grant et al., 2004). However, which PD offers the best prediction of AU and AUD, and the nature of the etiologic overlap, remains unclear.

Twin studies are a commonly used method to estimate the relative proportions of how much latent genetic and environmental risk factors contribute to the development of a particular trait. These studies provide compelling evidence that AU and AUD (Kendler et al., 1994; Reich et al., 1998; Verhulst et al., 2015), and PDs (Kendler et al., 2008a; Reichborn-Kjennerud, 2008) are all complex, heritable phenotypes. Cholesky decompositions, also referred to as bivariate twin models, go beyond the basic univariate twin model and allow us to determine the extent to which genetic and environmental influences are shared by two traits or are trait specific (Neale and Cardon, 1992). Regarding the putative sources of comorbidity between AU and AUD with PDs, evidence suggests that genetic risk factors are shared between borderline PD, alcohol, nicotine, and cannabis misuse (Bornova et al., 2013), as well as between antisocial behavior and AU (McAdams et al., 2012). These shared genetic risks account for up to 50% of the total genetic variance in risk in AUD (Fu et al., 2002). One limitation is that these studies have nearly always relied on single PDs

(Bornovalova et al., 2013; Fu et al., 2002; McAdams et al., 2012). Until now, fully integrative and genetically informative data have not been available to elucidate the genetic and environmental pathways linking PDs to AU and AUD.

We are not aware of any published studies that have investigated the association between all 10 DSM-IV PDs, AU, and AUD. To address this gap, we examined the following three aims: (1) identify which of the 10 PDs provide the strongest phenotypic prediction of the liabilities to AU and AUD; (2) estimate the degree to which the associations between PDs and AU and AUD are due to shared genetic or shared environmental risks; and (3) determine if the patterns of associations between PDs and AU and AUD are stable across time.

2. Method

2.1 Sample

Twins were recruited by the Norwegian Institute of Public Health (NIPH) Twin Panel from the National Medical Birth Registry of Norway, which was established in 1967 (Harris et al., 2002; Tambs et al., 2009). By mandate the registry receives notification of all births in Norway. The NIPH Twin Panel initially ascertained twins born from 1967 through 1974 who were at least 18 years of age. They were first contacted for study of health via a mail-out questionnaire (Q1) in 1992. These twins were re-contacted for a longitudinal follow-up using a second health questionnaire (Q2) in 1998. At that time, a younger cohort born 1975 to 1979 was also recruited and administered the same Q2.

Altogether, 8,045 twins (63%) including 3,334 pairs (53%) responded to Q2. All complete pairs from the Q2 study in which both twins were willing to be contacted again for new studies ($N = 3,153$ twin pairs) were invited by mail to participate in the Wave 1 interviews of mental health used in the present analyses. Due to technical problems, an additional 68 pairs were accidentally drawn from twin pairs that had not completed the Q2. The Wave 1 structured and semi-structured diagnostic interviews were carried out between 1999–2004 and assessed DSM-IV lifetime Axis I and Axis II disorders. Wave 1 interviews were mostly conducted face-to-face, with a small amount conducted by telephone (8.3%).

Data for Wave 2 came from a follow-up telephone interview administered between 2010–2011 (Nilsen et al., 2013). Of the 3,221 twin pairs eligible for Wave 1, there were 1,391 complete pairs (43.2%) and 19 single twins (0.6% pairwise), totaling 2,801 twins who participated (43.4%) and comprising 63% females ($M_{age} = 28$ years, range = 19–36). Of the 2,801 twins eligible for Wave 2, there were 2,393 twins (85.43%), comprising 1,063 complete twin pairs and 267 single twins who participated, including 64% females ($M_{age} = 38$ years, range = 30–44). For more detailed information about the sampling process and twin sample, please see Tambs et al. (2009) and Nilsen et al. (2013).

Monozygotic (MZ) males and females as well as dizygotic (DZ) males, females, and opposite sex twins were included in all analyses. The sample sizes for each group by sex are as follows: MZ males = 225; MZ females = 453; DZ males = 120; DZ females = 267; DZ opposite sex = 345.

Different interviewers assessed each twin pair member. Interviewers at Wave 1 were mostly advanced psychology students, or experienced psychiatric nurses, who received standardized training and supervision during data collection. Interviewers at Wave 2 included senior clinical psychology graduate students, psychiatric nurses, and experienced clinical psychologists who were interviewers at Wave 1. Written informed consent was obtained from all participants who received stipends of \$35 and \$70 at Waves 1 and 2, respectively. Ethical approval for both assessments came from the Regional Ethical Committee.

2.2 Measures

2.2.1 Predictors – Personality Disorder Criteria—Lifetime DSM-IV PDs were assessed using a Norwegian version of the Structured Interview for DSM-IV Personality (SIDP-IV; Pfohl et al., 1995). The SIDP-IV is a comprehensive, semi-structured diagnostic interview that includes non-pejorative questions organized into topical sections rather than by individual PD thereby improving the flow of the interview. The number of criteria for each of the DSM-IV PDs used in the analyses were as follows: schizotypal (9 criteria); schizoid (8 criteria); paranoid (7 criteria); histrionic (8 criteria); borderline (9 criteria); obsessive-compulsive (8 criteria); dependent (8 criteria); avoidant (7 criteria); narcissistic (9 criteria); and antisocial (7 criteria). The SIDP-IV considers the behaviors, cognitions, and feelings that are reported to be predominately present over the past five years of a participant's life to be representative of the individual's personality. Importantly, the SIDP-IV interview was conducted after the Composite International Diagnostic Interview (CIDI; Wittchen, 1994; Wittchen and Pfister, 1997), which assesses Axis 1 disorders. This order of assessment allowed us to conclude that the symptoms of PDs were due to the PD, and not a temporary effect of an Axis I disorder. All 10 PDs were assessed face-to-face at Wave 1.

At Wave 2, six of the 10 PDs were assessed by telephone interview: paranoid, schizotypal, borderline, obsessive-compulsive, avoidant, and antisocial. Each criterion was scored on a 4-point scale (absent, sub-threshold, present, or strongly present), dichotomized (0 = absent, 1 = sub-threshold), and summed into a PD trait score. However, because very few participants endorsed most PD criteria, the PD scores had strong positive skewness with a predominance of zero values. Therefore, for analytic purposes, each PD score was recoded onto a 3-point ordinal scale (0 criteria, 1–2 criteria, 3 criteria). This was also done to establish a common frame of reference to facilitate interpreting comparisons between odds ratios. Complete PD data were available from 2,793 twins for Wave 1 and 2,282 for Wave 2.

2.2.2 Outcome variables - Alcohol Use and Alcohol Use Disorder Criteria—

Lifetime AU and AUD based on the number of DSM-IV criteria for alcohol abuse and dependence were assessed using a Norwegian version of the Composite International Diagnostic Interview (Wittchen and Pfister, 1997). The CIDI has good test-retest and inter-rater reliability (Rubio-Stipec et al., 1999; Wittchen, 1994; Wittchen et al., 1998), and the Norwegian version has been used previously (Landheim et al., 2003). Lifetime AU was assessed for the 12-month period where consumption was highest using a 3-point ordinal scale (0 = never tried; 1 = less than 1 time per month, and 1–3 times per month; 2 = 1–2 times per week, 3–4 times per week, and almost every day). This was followed by questions covering the 11 DSM-IV criteria and one craving item. Criteria sum scores were then

recoded onto a 3-point ordinal alcohol use disorder (AUD) scale (0 = 0 criteria, 1 = 1–3 criteria, and 2 = 4 or more criteria) and used for all subsequent analyses. Complete AU and AUD data were available from 2,482 twins for Wave 1. At Wave 2, data were available from 2,238 twins for AU and 2,239 twins for AUD. See Table 1 for the prevalences of AU and AUD at both waves.

2.2.3 Prevalences of AU and AUD and Reliability—The sample size and prevalences of AU and AUD are shown in Table 1. To obtain reliability estimates between the two waves of data, we estimated weighted kappa coefficients and polychoric correlations. The weighted kappas for AU and AUD between Wave 1 and Wave 2 were 0.31 and 0.40, respectively. The polychoric correlations were 0.49 for AU and 0.58 for AUD. Typical values of weighted kappa for psychiatric diagnoses range from .40 to .60 (Fleiss and Cohen, 1973; Spitzer and Endicott, 1968; Spitzer and Endicott, 1969), and although the weighted kappa for AU was low, the polychoric correlations suggest that agreement between the two waves is adequate.

2.3 Statistical Analyses

2.3.1 Logistic Regressions—Given the number of predictors, we implemented an empirical approach to identify a subset of PD criteria sum scores for inclusion in the bivariate twin analyses to explore the genetic and environmental associations between PDs with AU and AUD. First, univariate ordinal logistic regressions using the `polr()` function in R_{3.1.1} (R Development Core Team, 2013) were fitted for each PD as a predictor at Wave 1 and Wave 2 in order to examine the effects of PD criteria scores independently. Second, four separate multiple regressions were run using the `polr()` function with a stepwise algorithm: (i) the regression of Wave 1 AU onto all 10 Wave 1 PDs; (ii) the regression of Wave 1 AUD onto all 10 Wave 1 PDs; (iii) the regression of Wave 2 AU onto all 6 Wave 2 PDs; and (iv) the regression of Wave 2 AUD onto all 6 Wave 2 PDs. All regressions included sex and age as covariates. PDs that significantly predicted AU and AUD in the multiple regressions were then brought forward into the bivariate twin analyses.

2.3.2 Twin Methods—Data were analyzed using the Full Information Maximum Likelihood (FIML) raw ordinal data method in the R_{3.1.1} OpenMx_{2.0} package (Neale et al., 2015; R Development Core Team, 2013). This approach makes use of all available data from both complete and incomplete twin pairs, thereby increasing the precision of threshold estimates and improving estimation of the correlations between predictors and outcomes. Our approach assumes a multivariate normative liability threshold model in order to estimate thresholds, which are conceptualized as “cut points” along an unobserved continuous distribution of liability on which individuals can be ordered based on the observed frequencies of the ordinal categories.

Standard biometrical genetic methods (Neale and Cardon, 1992 (Jinks and Fulker, 1970)) were used to exploit the expected genetic and environmental correlations of monozygotic (MZ) and of dizygotic (DZ) twin pairs to estimate the size and significance of the genetic and environmental risk pathways between each selected predictor and the ordinalized AU and AUD criteria variables. The biometrical genetic model assumes that the covariance between MZ and DZ twin pairs can be decomposed into additive (A) genetic, shared

environmental (C), and non-shared or unique (E) environmental variance components. Because MZ twin pairs are genetically identical while DZ twin pairs share, on average, half of their genes, the expected twin pair correlations for the genetic (A) effects are fixed at 1.0 and 0.5, respectively. An important assumption in the model is that C is equal in MZ and DZ twin pairs since the model fixes this correlation to 1 for twin 1 and twin 2 in both MZ and DZ twin pairs. E is by definition uncorrelated and also includes random measurement error.

Our approach assumes that regardless of twin order and zygosity, subjects have the same threshold distribution for the AU and AUD outcomes. We were able to equate the thresholds across twin order ($p = 0.36$) and zygosity ($p = 0.33$) without any significant deterioration in model fit for Wave 1 AU. Threshold distributions for Wave 1 AUD could also be constrained equal across twin order and zygosity ($p = 0.77$ for twin order, $p = 0.78$ for zygosity).

Bivariate Cholesky decompositions use the additional information in the cross-correlations between twins for different traits and permit estimating the extent to which genetic and environmental influences are shared by the two traits or are trait specific (Neale and Cardon, 1992). The bivariate Cholesky decomposition specifies that the first and second observed variables have paths coming from the first latent component whereas a second orthogonal latent component has a path to only the second variable. In this decomposition, the first latent component estimates the biometric portion of the covariation that is shared between the two observed variables with the second latent component identifying the portion unique to the second variable. This same factor structure is specified for each of the etiological sources A, C, and E.

The antisocial and borderline criteria included items that made reference to substance use, or substance use related problems. Therefore, in order to determine if any degree of genetic or environmental associations with AU and AUD arise from overlapping content, the analyses were repeated after dropping the items '*Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating)*' and '*Failure to conform to social norms with respect to lawful behavior as indicated by repeatedly performing acts that are grounds for arrest*' from the borderline and antisocial aggregate criteria sum scores, respectively.

All models were run with age and sex as covariates. To determine the best fitting model, the fully saturated 'ACE' model served as a baseline reference to compare models with the shared environmental (i.e., the additive genetic model; A+E model) and genetic (i.e., the shared environmental model; C+E model) parameters dropped by fixing these component pathways to zero. Model comparisons were evaluated using the Akaike Information Criterion (AIC; Akaike, 1987). A stronger emphasis for model selection is placed on this parsimony index because in Maximum Likelihood -2 times the log likelihood (-2LL) values will decrease with the addition of more parameters, which can lead to 'over-fitting.' Indices of parsimony penalize models with increasing numbers of parameters, thereby providing a balance between model complexity and model or data misfit.

3. Results

3.2 Univariate and multiple logistic regressions

3.2.1 Wave 1 Alcohol Use—In the univariate regressions, five of the PDs significantly predicted AU at Wave 1 (see Table 2). In the stepwise multiple regression, only paranoid, borderline, and antisocial PDs remained significant and showed a positive association (See Table 3). Obsessive compulsive and dependent PDs also emerged as statistically significant, such that higher sum scores were associated with reduced AU.

3.2.2 Wave 1 Alcohol Use Disorder—In the univariate regression models predicting Wave 1 AUD criteria, eight out of the 10 PDs were significant positive predictors. In the multiple regression, borderline and antisocial PDs were significant predictors of AUD symptoms, with both showing positive associations. Schizoid and schizotypal PDs also emerged as significant, showing negative associations with AUD criteria.

3.2.3 Wave 2 Alcohol Use—In the univariate regressions predicting Wave 2 AU, four of the six PDs that were assessed at Wave 2 predicted increased AU. In the multiple regression that included the six PDs, only borderline and antisocial remained significant, positive predictors of AU.

3.2.4 Wave 2 Alcohol Use Disorder—In the univariate regression models predicting Wave 2 AUD criteria, all six PDs predicted increased AUD criteria. However, in the multiple regression, borderline and antisocial were again the only PDs that remained significant and positive predictors of AUD symptoms.

3.3 Bivariate Twin Analyses (Cholesky decompositions)

All significant predictors of AU and AUD based on the multiple regressions for Waves 1 and 2 were then brought forward into the bivariate twin analyses: paranoid, obsessive compulsive, dependent, schizoid, schizotypal, borderline, and antisocial PDs. Results showed very little phenotypic (r_P), genetic (r_A), or environmental (r_E) correlations between Wave 1 AU with paranoid, obsessive-compulsive, and dependent PDs, as well as between Wave 1 AUD with schizoid and schizotypal PDs. Thus, full results of models with all correlations less than 0.2 are shown in Supplementary Table 1¹.

3.3.1 Predictors of Wave 1 Alcohol Use—For all of the Wave 1 AU bivariate models, the additive genetic (A+E) model in which the shared environmental components were removed provided the “best” parsimonious fit. As shown in Table 4, there were moderate correlations between AU with borderline and antisocial PDs. The proportions of total variance in AU explained by the genetic and environmental risks in the PDs were obtained by squaring the corresponding path coefficients. Despite the moderate genetic correlations between AU and borderline or antisocial PDs, the additive genetic factors in each of the PDs explained relatively little total variance in AU. Additive genetic factors in borderline PD explained 4% of the total variance in AU, a small but statistically significant amount.

¹Supplementary material can be found by accessing the online version of this paper at <http://dx.doi.org> and by entering doi:...

Additive genetic factors in antisocial PD explained 3% of the total variance in AU, a statistically non-significant amount. Likewise, unique environmental risk factors in each of the PDs explained very little of the total variance in AU (2% and 6%, respectively). Although the environmental risks in antisocial PD explained a modest amount of the variance, it was a statistically significant amount.

Bivariate modeling results for the trimmed PD sum scores are shown at the bottom of Table 4. For borderline PD, note the reduction in the estimated genetic correlation from 0.32 to 0.21, as well as a corresponding drop from 4% to 1% of the total variance in AU attributable to genetic risk factors. For antisocial PD, the changes in the estimates for the trimmed vs. original PD were in the opposite direction, as the genetic correlation with AU increased from 0.33 to 0.39, which is consistent with the corresponding increase from 3% to 4% in terms of the total AU variance explained by antisocial PD genetic risk factors. All proportions of variance for the trimmed variables were statistically non-significant.

3.3.2 Predictors of Wave 1 Alcohol Use Disorder—For all of the Wave 1 AUD models, the additive genetic (A+E) model again showed the best fit. As shown in Table 5, the phenotypic correlations between AUD symptoms and borderline or antisocial PDs were higher (0.33 and 0.43, respectively). The genetic correlations were also higher (0.41 and 0.60), as were the unique environmental correlations (0.29 and 0.35). However, as with AU, the proportions of genetic and unique environmental variance that each PD explained in AUD were modest, but statistically significant (5–10%).

After removing the potentially confounding substance use criteria from the antisocial and borderline PDs, the phenotypic correlations were lower. For the trimmed borderline PD, the phenotypic correlation declined from 0.33 to 0.25 with the corresponding genetic correlation declining from 0.41 to 0.33. Therefore, the proportion of total variance in AUD symptoms attributable to the genetic risks in this PD also dropped from 5% to 3%, a non-significant amount. However, for the trimmed antisocial PD, the genetic correlation showed less of a decline (from 0.60 to 0.57). The proportion of total variance explained by the genetic risks correspondingly only dropped from 10% to 9% and remained statistically significant.

3.3.3 Predictors of Wave 2 Alcohol Use—Similar to the models from Wave 1 AU and AUD, the additive genetic (A+E) model also provided the best fit for all Wave 2 AU models (see Table 6). The phenotypic correlations between borderline and antisocial PDs with AU were similar to those for Wave 1 AU. However, the genetic correlations showed a substantial increase from Wave 1 (0.44 and 0.77 from 0.32 and 0.33, respectively), while the unique environmental correlations decreased dramatically (0.06 and 0.04 from 0.17 and 0.29, respectively). Despite the higher genetic correlations, the genetic factors in each of the PDs similarly explained a relatively small, but statistically significant, amount of the total variance in AU (8% and 23%, respectively), although this is still an increase from Wave 1. The unique environmental risk factors in each of the PDs explained none total variance in Wave 2 AU.

For the trimmed borderline PD, the estimated genetic correlation dropped from 0.44 to 0.25. The total variance in AU attributable to genetic risk factors correspondingly dropped from

8% to 3% and is no longer statistically significant. This reduction is similar to the decrease shown in Wave 1. For the trimmed antisocial PD, the changes in the estimates from the original PD were in the opposite direction, similar to Wave 1 AU. The genetic correlation increased from 0.77 to 1.00 for the trimmed PD. The total AU variance explained by the trimmed antisocial PD genetic risk factors increased from 23% to 38% and remained significant.

3.3.4 Predictors of Wave 2 Alcohol Use Disorder—In contrast to all of the other models thus far, the additive genetic (A+E) model was not the best fitting model for all of the predictors of Wave 2 AUD symptoms (see Table 6). For both the borderline PD and the trimmed borderline PD, the ACE model was the best fitting model, indicating that the shared environmental (C) components could not be dropped to zero. Therefore, Table 7 shows the phenotypic, additive genetic, and unique environmental correlations between the PDs and Wave 2 AUD criteria, as well as the shared environmental correlations for the borderline and trimmed borderline PDs. The phenotypic and unique environmental correlations between borderline PD and AUD symptoms were moderate, with a reduction in the correlation for the trimmed phenotype. The genetic correlation dropped from 0.75 to -0.01 for the trimmed PD, while the shared environmental correlation showed an increase from 0.33 to 0.50. Borderline PD explained a small and statistically non-significant amount of the genetic risk in AUD symptoms (17%), while the trimmed borderline PD explained none of the risk. The proportions of shared environmental and unique environmental variance that borderline PD and the trimmed borderline PD explained in AUD were negligible and statistically non-significant (1–4%).

The pattern of results for the antisocial and trimmed antisocial PDs was similar to those for Wave 1 AU and Wave 2 AU. The additive genetic (A+E) model provided the best fit for both models. The phenotypic correlations were similar to those for Wave 1 AUD (0.37 and 0.31, respectively). As with Wave 2 AU, the genetic correlations increased from 0.85 to 1.00 for the trimmed PD, while the unique environmental correlations decreased from 0.11 to 0.05. The genetic factors in the antisocial and trimmed antisocial PD correspondingly explained a more moderate and statistically significant portion of the total variance in AUD (33% and 44%, respectively). The unique environmental risk factors explained virtually none of the total variance in AUD.

4. Discussion

This is the first study to jointly examine all 10 PDs with AU and AUD and analyze the nature of their associations within a genetically informative twin design. Although there were a number of PDs that significantly predicted AU and AUD, borderline and antisocial PDs showed the strongest phenotypic associations with AU and AUD. Twin analyses also revealed that individual differences in borderline and antisocial PD criteria were the strongest phenotypic and genotypic correlates of AU and AUD at Waves 1 and 2. However, neither these genetic nor unique environmental risk factors in these PDs explained much of the total liability to use or misuse alcohol at Wave 1. The increase in the amount of variance explained by genetic risk factors between Waves 1 and 2 suggests that the genetic associations between borderline and antisocial PDs with AU and AUD become stronger with

age. These results are consistent with previous research showing that genetic influences on alcohol use and on the development of AUD become more important over time (Edwards et al., 2015; Kendler et al., 2011; Kendler et al., 2008b; van Beek et al., 2012).

Our estimates of the total genetic variance in AU and AUD attributable to antisocial PD criteria were lower than those reported by Fu et al. (2002). However, our confidence intervals span their estimated 50% of total genetic variance explained by antisocial PD in Wave 2 AUD. The moderate genetic correlation we found between borderline PD and Wave 1 AU is commensurate with a recent report finding the genetic correlation with alcohol abuse-dependence to be .33 (Few et al., 2014). This correlation was slightly higher for Wave 1 AUD and Wave 2 AU (.41-.44), and highest for Wave 2 AUD (0.75). In terms of sources of covariation, Few et al. (2014) found that the association between borderline and alcohol abuse-dependence was attributable to genetic risk factors only in the absence of neuroticism. Likewise, Distel et al. (2012) found the association between heavy AU and borderline PD to be attributable largely to unique environmental risks. Distel et al.'s results are somewhat inconsistent with our findings since most of the individual differences in borderline PD, AU, and AUD across both Waves were explained by unique environmental risks, which were unshared.

We also found a number of novel findings. For instance, our multiple regression analyses showed that increased obsessive compulsive and dependent PD criteria were associated with lower risk of Wave 1 AU, while increased paranoid PD criteria predicted increased risk of Wave 1 AU. In addition, schizoid PD predicted a decreased risk of Wave 1 AUD. Although our effect sizes were modest, we are unaware of any previous similar findings. Hasin et al.'s (2011) analysis of National Epidemiologic Survey on Alcohol and Related Conditions data found no associations between these PDs and persistent alcohol abuse-dependence. One potential explanation for our results is that alcohol consumption may differ across samples and country of origin. Another explanation is that the analyses conducted by Hasin et al. (2011) had lower power than ours. Although they had a larger sample size, they used only dichotomous diagnoses, which resulted in fewer cases and larger confidence intervals. Consequently, in the absence of a suitable replication sample, it should be emphasized that obsessive compulsive, dependent, paranoid, and schizoid PD criteria each explained very little of the total phenotypic and genetic variance in both AU and AUD. Accordingly, these other PDs remain less informative.

In terms of inconsistent findings, our Wave 1 findings are in sharp contrast to the pattern of results that we have recently observed with cannabis use and misuse (Gillespie et al., manuscript submitted for publication). Based on a similar design using PDs to predict cannabis use and misuse with personality as covariates, it was found that genetic risks in borderline and antisocial PDs shared 29–30% with the genetic risks in cannabis use, and 31–45% of the genetic risks in cannabis use disorder. Despite the fact that alcohol and cannabis use and misuse are frequently comorbid (Agosti et al., 2002; Duncan et al., 2015), one explanation for this discrepancy, apart from cannabis use being more deviant, could be related to AU and AUD being partially genetically distinct from cannabis use and misuse (Kendler et al., 2007). However, we note that our Wave 2 findings are more consistent with the cannabis use and cannabis use disorder estimates.

Our findings are also inconsistent with those of Hasin et al. (2011), who found that in addition to antisocial and borderline, schizotypal PD also predicted three-year persistence of cannabis, alcohol, and nicotine use disorders. In our results, schizotypal PD had a significant negative association with Wave 1 AUD, rather than a positive association.

In a broader context, our results are consistent with role of PDs in the spectrum of externalizing disorders, which is highly heritable (Krueger et al., 2002) and characterized by conduct and substance use disorders, including AUD, (Markon et al., 2005), antisocial PD, and borderline PD (Eaton et al., 2011). Elsewhere, we have shown that correlations across time between these two PDs can be attributed to common, longitudinally stable genetic risk factors (Reichborn-Kjennerud et al., 2015). Overall, our findings suggest that among the DSM-IV PDs, borderline and antisocial PD criteria are the key phenotypic and genotypic correlates of AU and AUD, and that these patterns of association are stable across time.

4.1 Limitations

Our results should be interpreted in the context of five potential limitations. First, all regression models assume independent observations. Failure to account for non-independence or clustered samples, such as twin data, typically does not impact parameter estimates, but may lead to slightly narrower estimates of confidence interval ranges. However, non-independence is in general much less problematic when group or cluster sizes are uniform and small, which is the case for twin pairs.

Second, although our twin analyses identified significant shared genetic pathways between PD criteria to alcohol use and misuse, the set of possible models examined was not exhaustive. In particular, we did not test competing causal hypotheses, which were beyond the scope of this report. Bornovalova et al. (2013) have recently shown that associations between borderline PD and the frequency of past 12-month tobacco, alcohol, and cannabis use could be best explained by a correlated liabilities model, as opposed to any causal mechanism based model. Additionally, it is possible that a high genetic correlation with a high environmental correlation cancelled out any phenotypic association for some of the AU/AUD – PD pairings. A multivariate genetic analysis would be required to determine this.

Third, a natural limitation of twin models is that they cannot identify genetic processes underlying the observed covariation between the PDs and AU and AUD. There is, however, substantial evidence showing that AU and AUD (Kendler et al., 1994; Reich et al., 1998; Verhulst et al., 2015) and PDs (Kendler et al., 2008a; Reichborn-Kjennerud, 2008) are highly polygenic. In other words, the genetic variances and covariance are unlikely to be attributable to a single or pair of discrete genetic structures that influence the development of PDs, AU, or AUD, but rather to many genes of very small effect contributing to these phenotypes. Therefore, we can speculate that the observed genetic covariation between AU and AUD and the PDs are also highly polygenic.

Fourth, while the sample is broadly representative, some attrition occurred from when the NIPH began recruiting twins through to Wave 1. This may have introduced some bias if attrition was non-random with respect to the dependent variables (Rothman, 1986). We have shown that only demographic, and not mental health, or AU indicators predicted

participation at Wave 1 (Tambs et al., 2009). Participation in Wave 2 was predicted by high education ($p < 0.001$ adjusted for sex and age), female sex ($p = 0.003$), and monozygosity ($p = 0.001$). Non-participants in Wave 2 had on average 0.82 more sub-threshold PD criteria than participants ($p < 0.001$). Of the 10 PDs assessed at Wave 1, criteria were significantly higher in non-participants in Wave 2 only for antisocial PD (0.09 criteria difference, $p < 0.001$) and narcissistic PD (0.09 criteria difference, $p = 0.002$). Borderline PD did not predict participation (0.05 criteria difference, $p = 0.06$). Neither the total number of axis I disorders nor any specific disorder were significantly higher in the non-participation group (Reichborn-Kjennerud et al., 2015).

Finally, the sample was underpowered to detect sex differences (Neale and Cardon, 1992). However, previous research has suggested that the magnitude of genetic influences among males and females were equally high, and that these sources of liability were partially overlapping between the two sexes (Prescott et al., 1999).

4.2 Conclusion

Using a large Norwegian twin sample, we have shown that borderline and antisocial PDs were the strongest correlates of the phenotypic and genotypic liability to AU and AUD. These patterns of associations remained consistent across time. Our findings suggest that effective prediction of alcohol use and misuse can rely more heavily on criteria for these two PDs in preference to other PD diagnoses. By contributing to our understanding of the etiologic overlap between PDs and AU/AUD, our findings may ultimately help to improve the treatment of individuals with these disorders.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- The etiological overlap between Personality Disorders (PDs) and Alcohol Use/Alcohol Use Disorder (AU/AUD) is unclear.
- Borderline and antisocial PDs were the strongest predictors of AU/AUD.
- This effect remained consistent across time.

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Table 1

Sample size and prevalence of alcohol use (AU) and DSM-IV alcohol use disorder (AUD) criteria at Wave 1 and Wave 2

AU	Number of Twins	Never tried (0)	Less than 1x/month; 1–3 times/month (1)	1–2x/week; 3–4x/week; Almost every day (2)
Wave 1	2,482	58.2% (1,445)	39.8% (987)	0.02% (50)
Wave 2	2,238	77.1% (1,726)	20.7% (464)	0.02% (48)

AUD		0 criteria (0)	1–3 criteria (1)	More than 4 criteria (2)
Wave 1	2,482	74.2% (1,842)	18.8% (467)	0.07% (173)
Wave 2	2,239	84.1% (1,884)	10.6% (238)	0.05% (117)

Table 2

Univariate logistic regression results of personality disorders predicting WAVE 1 AND 2 ALCOHOL USE and the symptoms of WAVE 1 AND WAVE 2 ALCOHOL USE DISORDER.

	ALCOHOL USE				ALCOHOL USE DISORDER			
	Wave 1		Wave 2		Wave 1		Wave 2	
	OR	(95%CI)s	OR	(95%CI)s	OR	(95%CI)s	OR	(95%CI)s
Schizoid PD	1.03	(0.85–1.24)			1.04	(0.83–1.29)		
Paranoid PD	1.33	(1.15–1.52)	1.45	(1.19–1.75)	1.50	(1.28–1.75)	1.70	(1.38–2.09)
Schizotypal PD	1.16	(0.96–1.39)	1.24	(0.98–1.57)	1.22	(0.99–1.51)	1.58	(1.21–2.04)
Histrionic PD	1.28	(1.12–1.46)			1.39	(1.20–1.61)		
Borderline PD	1.59	(1.39–1.81)	1.62	(1.38–1.90)	2.12	(1.83–2.46)	2.04	(1.71–2.44)
Obsessive Compulsive PD	0.99	(0.87–1.12)	1.23	(1.06–1.42)	1.17	(1.01–1.35)	1.31	(1.10–1.55)
Dependent PD	1.03	(0.89–1.20)			1.32	(1.12–1.55)		
Avoidant PD	1.04	(0.91–1.18)	1.03	(0.86–1.22)	1.27	(1.09–1.47)	1.24	(1.02–1.50)
Narcissistic PD	1.29	(1.12–1.48)			1.53	(1.31–1.78)		
Antisocial PD	2.10	(1.70–2.60)	2.11	(1.62–2.74)	3.08	(2.49–3.80)	2.67	(2.03–3.51)

Note. In Wave 2, only six personality disorders were assessed.

Table 3

Multiple logistic regression results using forward selection with personality disorders predicting *WAVE 1 AND WAVE 2 ALCOHOL USE* and the symptoms of *WAVE 1 AND WAVE 2 ALCOHOL USE DISORDER*.

	ALCOHOL USE				Symptoms of ALCOHOL USE DISORDER			
	Wave 1		Wave 2		Wave 1		Wave 2	
	OR	(95%CIs)	OR	(95%CIs)	OR	(95%CIs)	OR	(95%CIs)
Sex	2.08	(1.70–2.56)	2.46	(1.96–3.08)	2.65	(2.11–3.35)	3.35	(2.59–4.35)
Age at interview	0.94	(0.92–0.96)	0.95	(0.93–0.98)	0.93	(0.90–0.95)	0.94	(0.91–0.97)
Schizoid PD					0.77	(0.60–0.98)		
Paranoid PD	1.18	(1.01–1.39)						
Schizotypal PD					0.75	(0.58–0.97)		
Histrionic PD								
Borderline PD	1.50	(1.28–1.75)	1.45	(1.20–1.75)	1.75	(1.47–2.09)	1.79	(1.48–2.16)
Obsessive Compulsive PD	0.83	(0.72–0.95)						
Dependent PD	0.82	(0.70–0.96)						
Avoidant PD								
Narcissistic PD								
Antisocial PD	1.74	(1.39–2.19)	1.69	(1.28–2.23)	2.29	(1.82–2.89)	2.02	(1.51–2.69)

Note. In Wave 2, only six personality disorders were assessed.

Table 4

Bivariate Cholesky A, C, and E decomposition comparisons and summaries for each personality disorder with *WAVE 1 ALCOHOL USE*

Bivariate model fit comparisons					Correlations (95% CI)		Proportion of total variance in ALCOHOL USE, shared (with each predictor) versus unshared (95% CI)				
Predictor	Model	-2LL	df	AIC	rP	rA	rE	Ashared	Aunshared	Eshared	Eunshared
Borderline PD (total)	ACE	9065.33	5266	-1466.67							
	AE	9066.74	5269	-1471.27	0.22 (0.16 – 0.28)	0.32 (0.26 – 0.52)	0.17 (0.06 – 0.19)	4% (1 – 9%)	30% (20 – 40%)	2% (0 – 5%)	65% (55 – 75%)
	CE	9073.87	5269	-1464.13							
	E	9173.62	5272	-1370.38							
Antisocial PD (total)	ACE	6589.46	5266	-3942.54							
	AE	6589.66	5269	-3948.34	0.30 (0.23 – 0.36)	0.33 (0.07 – 0.58)	0.29 (0.15 – 0.42)	3% (0 – 10%)	27% (16 – 37%)	6% (2 – 12%)	64% (53 – 75%)
	CE	6602.51	5269	-3935.50							
	E	6648.52	5272	-3895.48							
Borderline PD (trimmed)	ACE	8746.26	5266	-1785.74							
	AE	8746.29	5269	-1791.71	0.14 (0.09 – 0.20)	0.21 (0.00 – 0.42)	0.11 (-0.01 – 0.22)	1% (0 – 6%)	32% (22 – 42%)	1% (0 – 3%)	65% (55 – 75%)
	CE	8756.47	5269	-1781.53							
	E	8843.32	5272	-1700.68							
Antisocial PD (trimmed)	ACE	6166.89	5266	-4365.11							
	AE	6167.68	5269	-4370.32	0.26 (0.19 – 0.33)	0.39 (0.10 – 0.69)	0.21 (0.06 – 0.35)	4% (0 – 14%)	26% (16 – 37%)	3% (0 – 8%)	67% (56 – 78%)
	CE	6178.42	5269	-4359.58							
	E	6219.48	5272	-4324.52							

CI = confidence interval; -2LL = -2 X Log Likelihood; AIC = Akaike Information Criteria; ACE = additive genetic + shared environment + unique environmental risks; Trimmed = Borderline PD excluded 'Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating)'; Antisocial PD excluded 'Failure to conform to social norms with respect to lawful behavior as indicated by repeatedly performing acts that are grounds for arrest.'

Bivariate Cholesky A, C, and E decomposition comparisons and summaries for each personality disorder with *WAVE 1 DSM-IV ALCOHOL USE DISORDER* ordinal symptom criteria composite

Table 5

Bivariate model fit comparisons					Correlations (95% CI)			Proportion of total variance in symptoms of ALCOHOL USE DISORDER shared (with each predictor) versus unshared (95% CI)			
Predictor	Model	-2LL	df	AIC	rP	rA	rE	Ashared	Aunshared	Eshared	Eunshared
Borderline PD (total)	ACE	8806.52	5266	-1725.48							
	AE	8809.00	5269	-1729.00	0.33 (0.28 – 0.39)	0.41 (0.20 – 0.62)	0.29 (0.18 – 0.40)	5% (1 – 12%)	26% (14 – 37%)	6% (2 – 12%)	63% (52 – 75%)
	CE	8817.40	5269	-1720.61							
	E	8900.76	5272	-1643.241							
Antisocial PD (total)	ACE	6296.44	5266	-4235.56							
	AE	6296.47	5269	-4241.53	0.43 (0.37 – 0.49)	0.60 (0.34 – 0.88)	0.35 (0.22 – 0.47)	10% (3–21%)	18% (5–30%)	9% (3–17%)	63% (51 – 76%)
	CE	6311.77	5269	-4226.23							
	E	6343.73	5272	-4200.268							
Borderline PD (trimmed)	ACE	8515.76	5266	-2016.24							
	AE	8515.87	5269	-2022.13	0.25 (0.19 – 0.31)	0.33 (0.10 – 0.56)	0.21 (0.08 – 0.33)	3% (0 – 10%)	29% (16 – 40%)	3% (0 – 7%)	65% (53 – 77%)
	CE	8528.44	5269	-2009.57							
	E	8598.42	5272	-1945.58							
Antisocial PD (trimmed)	ACE	5903.17	5266	-4628.84							
	AE	5903.17	5269	-4634.84	0.38 (0.31 – 0.44)	0.57 (0.27 – 0.90)	0.29 (0.14 – 0.42)	9% (2 – 21%)	18% (4 – 31%)	6% (1 – 14%)	67% (55 – 80%)
	CE	5914.90	5269	-4623.10							
	E	5942.26	5272	-4601.74							

CI = confidence interval; -2LL = -2 X Log Likelihood; AIC = Akaike Information Criteria; ACE = additive genetic + shared environment + unique environmental risks; Trimmed = Borderline PD excluded 'Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating)'; Antisocial PD excluded 'Failure to conform to social norms with respect to lawful behavior as indicated by repeatedly performing acts that are grounds for arrest.'

Table 6

Bivariate Cholesky A, C, and E decomposition comparisons and summaries for each personality disorder with *WAVE 2 ALCOHOL USE*

Bivariate model fit comparisons					Correlations (95% CI)			Proportion of total variance in ALCOHOL USE shared (with each predictor) versus unique (95% CI)			
Predictor	Model	-2LL	df	AIC	rP	rA	rE	Ashared	Aunshared	Eshared	Eunshared
Borderline PD (total)	ACE	6470.03	4511	-2551.97							
	AE	6472.89	4514	-2555.11	0.20 (0.13 – 0.26)	0.44 (0.20 – 0.70)	0.06 (–0.09 – 0.21)	8% (2–20%)	34% (18–47%)	0% (0–3%)	58% (46–72%)
	CE	6475.83	4514	-2552.18							
	E	6475.83	4514	-2552.18							
Antisocial PD (total)	ACE	4133.74	4510	-4886.26							
	AE	4133.79	4513	-4892.22	0.28 (0.20 – 0.37)	0.77 (0.39 – 0.99)	0.04 (–0.15 – 0.24)	23% (6–49%)	16% (0–37%)	0% (0–4%)	61% (49–75%)
	CE	4139.39	4513	-4886.61							
	E	4139.39	4513	-4886.61							
Borderline PD (trimmed)	ACE	6277.30	4511	-2744.70							
	AE	6278.74	4514	-2749.26	0.10 (0.03 – 0.17)	0.25 (–0.01 – 0.52)	0.02 (–0.14 – 0.17)	3% (0–11%)	40% (25–53%)	0% (0–2%)	58% (45–71%)
	CE	6282.48	4514	-2745.52							
	E	6282.48	4514	-2745.52							
Antisocial PD* (trimmed)	ACE	3453.59	4512	-5570.41							
	AE	3453.64	4515	-5576.36	0.23 (0.13 – 0.32)	1.00** (1.00 – 1.00)	0.00 (–0.19 – 0.20)	38% (7–51%)	0% (0–35%)	0% (0–2%)	62% (49–76%)
	CE	3456.92	4515	-5573.08							
	E	3487.41	4518	-5548.59							

CI = confidence interval; -2LL = -2 X Log Likelihood; AIC = Akaike Information Criteria; ACE = additive genetic + shared environment + unique environmental risks; Trimmed = Borderline PD excluded 'Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating)'; Antisocial PD excluded 'Failure to conform to social norms with respect to lawful behavior as indicated by repeatedly performing acts that are grounds for arrest.'

*The Antisocial PD and alcohol use variables were recoded into binary variables for this model only due to empty cells.

**Model estimation of the upper and/or lower 95% CIs failed.

Table 7

Bivariate Cholesky A, C, and E decomposition comparisons and summaries for each personality disorder with *WAVE 2 DSM-IV ALCOHOL USE DISORDER* ordinal symptom criteria composite

Bivariate model fit comparisons					Correlations (95% CI)			Proportion of total variance in symptoms of ALCOHOL USE DISORDER shared (with each predictor) versus unshared (95% CI)					
Predictor	Model	-2LL	df	AIC	rP	rA	rC	rE	Ashared	Cshared	Cunshared	Eshared	Eunshared
Borderline PD (total)	ACE	6127.23	4512	-2896.77									
	AE	6130.44	4515	-2899.56									
	CE	6129.11	4515	-2900.89	0.27 (0.20–0.34)	0.75 *** (–0.99–0.99)	0.33 * * (–1.00–0.99)	0.18 (0.01–0.36)	17% (0–59%)	2% (0–38%)	13% (0–40%)	2% (0–7%)	53% (38–69%)
	E	6197.97	4518	-2838.03									
Antisocial PD (total)	ACE	3777.41	4511	-5244.59									
	AE	3777.96	4514	-5250.04									
	CE	3781.50	4514	-5246.50	0.37 (0.28–0.45)	0.85 (0.49–0.99)	-	0.11 (–0.09–0.31)	33% (10–57%)	-	-	1% (0–6%)	55% (41–70%)
	E	3818.39	4517	-5215.61									
Borderline PD (trimmed)	ACE	5948.43	4512	-3075.57									
	AE	5950.88	4515	-3079.12									
	CE	5950.38	4515	-3079.62	0.15 (0.07–0.22)	–0.01 * * (–1.00–1.00)	0.50 * * (–0.99–0.99)	0.10 (–0.09–0.29)	0% (0–60%)	4% (0–44%)	13% (0–44%)	1% (0–4%)	53% (38–70%)
	E	6017.44	4518	-3018.56									
Antisocial PD * (trimmed)	ACE	2979.70	4513	-6046.30									
	AE	2980.02	4516	-6051.98									
	CE	2981.67	4516	-6050.33	0.31 (0.21–0.41)	1.00 * * (0.99–1.00)	-	0.05 (–0.15–0.25)	44% (13–58%)	-	-	0% (0–4%)	56% (42–72%)
	E	3014.49	4519	-6023.51									

CI = confidence interval; -2LL = -2 X Log Likelihood; AIC = Akaike Information Criteria; ACE = additive genetic + shared environment + unique environmental risks; Trimmed = Borderline PD excluded 'Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating)' ; Antisocial PD excluded 'Failure to conform to social norms with respect to lawful behavior as indicated by repeatedly performing acts that are grounds for arrest.'

* The Antisocial PD and alcohol use disorder variables were recoded into binary variables for this model only due to empty cells.

** Model estimation of the upper and/or lower 95% CIs failed.