Genetic and environmental influences on dimensional representations of DSM-IV cluster C personality disorders: a population-based multivariate twin study

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ABSTRACT

Background. The DSM-IV cluster C Axis II disorders include avoidant (AVPD), dependent (DEPD) and obsessive-compulsive (OCPD) personality disorders. We aimed to estimate the genetic and environmental influences on dimensional representations of these disorders and examine the validity of the cluster C construct by determining to what extent common familial factors influence the individual PDs.

Method. PDs were assessed using the Structured Interview for DSM-IV Personality (SIDP-IV) in a sample of 1386 young adult twin pairs from the Norwegian Institute of Public Health Twin Panel (NIPHTP). A single-factor independent pathway multivariate model was applied to the number of endorsed criteria for the three cluster C disorders, using the statistical modeling program Mx.

Results. The best-fitting model included genetic and unique environmental factors only, and equated parameters for males and females. Heritability ranged from 27% to 35%. The proportion of genetic variance explained by a common factor was 83, 48 and 15% respectively for AVPD, DEPD and OCPD. Common genetic and environmental factors accounted for 54% and 64% respectively of the variance in AVPD and DEPD but only 11% of the variance in OCPD.

Conclusion. Cluster C PDs are moderately heritable. No evidence was found for shared environmental or sex effects. Common genetic and individual environmental factors account for a substantial proportion of the variance in AVPD and DEPD. However, OCPD appears to be largely etiologically distinct from the other two PDs. The results do not support the validity of the DSM-IV cluster C construct in its present form.

INTRODUCTION

The DSM-IV (APA, 1994) includes 10 personality disorders (PDs), coded on Axis II and grouped into three clusters A, B and C, often called the ‘Odd’, ‘Dramatic’ and ‘Anxious’. Cluster C consists of the avoidant (AVPD), dependent (DEPD) and obsessive-compulsive (OCPD) personality disorders.

Although numerous studies have examined the genetic epidemiology of DSM Axis I anxiety disorders (for a review, see Hettema et al. 2001), few have applied these methods to Axis II anxious PDs. Only one family study of cluster C
PDs has been published (Reich, 1989). The results indicated significant familiality for the DSM-III anxious cluster PDs as a whole, and for AVPD and DEPD (OCPD was not investigated separately). In a twin study of DSM-III-R PDs based on patient populations, Torgersen et al. (2000) found that the best-fitting models of cluster C PDs all included genetic factors. No population-based twin study of DSM PDs has been published.

The DSM-IV manual emphasizes that the classification of PDs into these three clusters is based on ‘descriptive similarities’ and has ‘serious limitations and has not been consistently validated’ (APA, 1994). Examination of the phenotypic structure, that is the pattern of covariance or co-occurrence between the disorders, has been used to test the justification of the cluster constructs. Although the results are equivocal, several studies have suggested that OCPD is only weakly related to the three traditional clusters (Kass et al. 1985; Hyler & Lyons, 1988; Nestadt et al. 1994; O’Connor & Lyons, 1988; Sanislow et al. 2002). Following Robins and Guze (1970), a more powerful way to validate the cluster C construct would be to determine the degree to which AVPD, DEPD and OCPD share familial/genetic risk factors. Multivariate twin studies have been used to evaluate the genetic structure of PD traits (Livesley et al. 1998) and normal personality (Krueger, 2000; McCrae et al. 2001), and this approach is among the research strategies expected to play a useful role in generating an empirical data base for the next edition of the DSM Axis II classification (Livesley, 2005; Widiger et al. 2005).

In this study we conducted multivariate twin analyses of a population-based sample of young adult twins to examine the genetic and environmental influences on dimensional representations of DSM-IV cluster C PDs, in order to examine the validity of the cluster C construct. We attempted to address two main questions: (1) What is the relative influence of genetic and environmental factors on AVPD, DEPD and OCPD in males and females? (2) To what extent are cluster C PDs influenced by common genetic, shared environmental and individual-specific environmental factors and to what extent are these factors specific to each individual PD?

**METHOD**

**Sample**

Data for these analyses come from the Norwegian Institute of Public Health Twin Panel (NIPHTP). The twins are identified through information contained in the Norwegian Medical Birth Registry, established 1 January 1967, which receives mandatory notification of all births. The current panel includes information on 15,370 like- and unlike-sexed twins born from 1967 to 1979. Two questionnaire studies have been conducted in this sample: in 1992 (twins born 1967–1974) and in 1998 (twins born 1967–1979). Altogether, 12,700 twins received the second questionnaire, and 8,045 responded after one reminder (response rate 63%). The sample included 3,334 pairs and 1,377 single responders. The NIPHTP is described in detail elsewhere (Harris et al. 2002).

Data for the present report derive from an interview study of Axis I and Axis II PDs, which began in 1999. Participants were recruited among 3,153 complete pairs who, in the second questionnaire, agreed to participate in the interview study, and 68 pairs who were drawn directly from NIPHTP. Of these 3,221 eligible pairs, 0.8% were unwilling or unable to participate, and in 16.2% of pairs only one twin agreed to the interview. After two contacts requesting participation, 38.2% did not respond. A total of 2,794 twins (44% of those eligible) were interviewed for the assessment of PDs. Attrition was not associated with measures of psychopathology (see Discussion). In 22 pairs where both twins initially agreed to be interviewed, one of the twins was later unable or unwilling to participate in the interview. The mean age of participants was 28.2 years (range 19–36 years).

Zygosity was initially determined by questionnaire items previously shown to categorize correctly 97.5% of pairs (Harris et al. 2002). In all but 385 like-sexed pairs, where one or both of the twins was either unwilling or unable to donate a blood sample, zygosity was also determined by molecular methods based on the genotyping of 24 microsatellite markers. Seventeen of these pairs with DNA information (2.5%) were found to be misclassified by the questionnaire data and were corrected. From the corrected data we estimated that, in our
entire sample, the zygosity misclassification rate was 0.7%, which is unlikely to substantially bias results (Neale, 2003). Our final sample consisted of 1022 males and 1772 females: 221 monozygotic male (MZM) pairs, 116 dizygotic male (DZM) pairs, 448 monozygotic female (MZF) pairs, 261 dizygotic female (DZF) pairs, 340 dizygotic opposite sex (DZO) pairs and 22 single responders.

Measurements

A Norwegian version of the Structured Interview for DSM-IV Personality (SIDP-IV; Pfohl et al. 1995) was used to assess PDs. This instrument is a comprehensive semi-structured diagnostic interview for the assessment of all DSM-IV Axis II diagnoses. The SIDP was initially developed in 1983, and has been used in a number of studies in many countries including Norway (Torgersen et al. 2001; Helgeland et al. 2005). The instrument includes non-pejorative questions organized into topical sections rather than by disorders. This allows for a more natural flow of the interview and increases the likelihood that useful information from related questions will be taken into account when rating related criteria within that section. The specific DSM-IV criterion associated with each set of questions is rated using the following scoring guidelines: 0 = not present or limited to rare isolated examples, 1 = subthreshold (some evidence of the trait, but not sufficiently pervasive to consider the criterion present), 2 = present (criterion is clearly present for most of the past 5 years, i.e. present at least 50% of the time), 3 = strongly present. The SIDP-IV interview is conducted after the Axis I interview, which helps the interviewer to distinguish more easily longstanding behavior reported by the subject from temporary states due to an episodic psychiatric disorder. The SIDP-IV uses the ‘5-year rule’, meaning that the behavior, cognitions and feelings predominating for most of the past 5 years are considered to be representative of the individual’s long-term personality functioning.

Interviewers were mostly psychology students in their final part of training and experienced psychiatric nurses. They were trained by professionals (one psychiatrist and two psychologists) with extensive previous experience with the instrument, and closely followed up individually during the whole data collection period. The interviews were carried out between June 1999 and May 2004, and were largely conducted face-to-face. For practical reasons, 231 interviews (8.3%) were obtained by telephone. Each twin in a pair was interviewed by different interviewers.

Inter-rater reliability was assessed based on two raters scoring 70 audiotaped interviews. The number of subjects with specific PDs was too low to calculate \( \kappa \) coefficients. Intra-class and polychoric correlations for the scaled PDs, using the number of endorsed criteria at the subthreshold level (see below), were all high: AVPD, +0.96, +0.97; DEPD, +0.96, +0.99; OCPD, +0.92, +0.87. Reliability measured by Cronbach’s \( \alpha \) based on polychoric correlations showed good internal consistencies: AVPD, 0.96; DEPD, 0.94; OCPD, 0.90.

Approval was received from the Norwegian Data Inspectorate and the Regional Ethical Committee, and written informed consent was obtained from all participants after complete description of the study.

Statistical methods

In this population-based sample of twins, the prevalence rate for categorical diagnoses of the cluster C PDs were too low for useful analyses. We therefore used a dimensional approach (Widiger & Samuel, 2005), constructing ordinal variables based on the number of criteria endorsed. To optimize statistical power and produce maximally stable results, we used the number of subthreshold criteria endorsed (\( \geq 1 \)) instead of criteria above the threshold (\( \geq 2 \)), assuming that the liability for each trait is continuous and normally distributed, that is that the classification (0–3) represents different degrees of severity. This assumption was evaluated using multiple threshold tests for each of the criteria. All the tests were performed separately for each zygosity group, and none was significant (all \( p \) values >0.05). Few subjects endorsed a high proportion of all or most of the criteria for an individual PD. To avoid empty cells, we collapsed the upper categories for the summed score. The maximum number of categories was created for each disorder. However, low prevalence in general for the DEPD items and low endorsement for the AVPD items in the DZM group limited the number of categories for these
two PDs to four (0–3). The ordinal variables for OCPD included five categories (0–4). The same procedure as described above was used to test the assumption that the number of positive criteria for each individual PD represented different degrees of severity for the PD. None of the multiple threshold tests was significant (all \( p \) values > 0.05).

In the classical twin model, individual differences in liability are assumed to arise from three latent factors: additive genetic factors (A), in which genetic effects combine additively; common or shared environment factors (C), which include all environmental exposures that are shared by the twins and contribute to their similarity; and individual-specific or unique environment factors (E), which include all environmental factors not shared by the twins plus measurement error. Because MZ twins share all their genes and DZ twins share on average 50% of their segregating genes, A contributes twice as much to the resemblance in MZ compared to DZ twins for a particular trait or disorder. By definition, MZ and DZ twins share all their C factors and none of their E factors. Non-additive genetic factors such as dominance or epistasis may be parameterized as an alternative to C, with which they are confounded. This model would fit more poorly in this study because the DZ correlations are not less than half of those of the MZ.

Model fitting was performed using the software package Mx (Neale et al. 1999), the most commonly used program for twin analyses. To test the degree to which the covariation between the three cluster C PDs resulted from common factors, we applied a single-factor independent pathway model containing three common latent variables (\( A_C \), \( C_C \) and \( E_C \)) in addition to three disorder-specific variables (\( A_S \), \( C_S \) and \( E_S \)) for each PD. The degree to which the cluster C PDs share genetic and environmental risk factors will be reflected in the loadings on the common versus disorder-specific factors. We chose a single common factor model for two reasons. First, because it instantiates the DSM construct of cluster C, that is the degree to which all three cluster C PDs share common genetic and environmental risk factors versus disorder-specific factors. Second, for statistical reasons, with only three disorders, models with two common factors are not identified.

A full model, including all latent variables and with different parameters specified for males and females, was tested against nested sub-models with reduced numbers of parameters. The fit of the alternative models can be compared using the difference in twice the log likelihood (2 ln L), which, under certain regularity conditions, is asymptotically distributed as \( \chi^2 \) with degrees of freedom (df) equal to the difference in the number of parameters (\( \Delta \chi^2 \) test). According to the principle of parsimony, models with fewer parameters are preferable if they do not result in a significant deterioration of fit. A useful index of parsimony is the Akaike Information Criterion (AIC), which is calculated as \( \Delta \chi^2 - 2\Delta df \) (Akaike, 1987). A lower AIC value indicates superior fit.

A basic assumption in traditional twin analyses is that MZ and DZ twins are equally correlated in their exposure to trait-relevant environments. We tested the validity of this ‘equal environment assumption’ by applying polychotomous logistic regression controlling for the correlational structure of our data using independent estimating equations as operationalized in the SAS procedure GENMOD (SAS Institute, 2005). Two variables that reflected, respectively, similarity of childhood (number of years that the twins were in the same class at school and the years the twins lived in the same residence) and adult environments (frequency of in-person and telephone contact during the past year and the distance between their current residences) were constructed. In same-sex pairs, we tested whether the PD score in twin 1 interacted with our measure of environmental similarity in predicting the relevant PD score in twin 2 (dependent variable). We controlled for main effects of zygosity, sex, age and level of environmental similarity as well as shared environment effects and genetic effects. None of the six analyses testing the impact of environmental similarity on twin resemblance for AVPD, DEPD and OCPD approached significance (all \( p \) values > 0.10).

RESULTS

Prevalence and co-occurrence

Prevalence rates for categorical DSM-IV cluster C PD diagnoses were 2.1% (\( n = 59 \)) for AVPD (males 1.4%, females 2.5%), 0.3% (\( n = 7 \)) for
DEPD (males 0.2%, females 0.3%) and 2.5% \((n=69)\) for OCPD (males 2.5%, females 2.4%).

The mean (S.D.) numbers of criteria \((\geq 1)\) met for the cluster C PDs were: AVPD, 0.95 (1.40); DEPD, 0.76 (1.17); OCPD, 1.93 (1.62). The proportion of individuals who endorsed none, 1 or 2 or more criteria were: AVPD, 55.0, 20.6 and 24.4%; DEPD, 57.7, 23.3, and 9.0%; OCPD, 22.7, 23.4 and 53.9% respectively. Females endorsed a significantly higher number of criteria for DEPD \((\chi^2=20, 25, df=8, p=0.009)\) and OCPD \((\chi^2=22, 61, df=8, p=0.004)\) but not for AVPD \((\chi^2=8, 79, df=7, p=0.27)\).

The phenotypic (within-individual) correlations based on dimensional representations of the PDs (Table 1), indicate substantial co-occurrence of AVPD and DEPD and significantly lower correlation between OCPD and the other two PDs for both males and females.

### Table 1. Phenotypic correlations based on dimensional representations of DSM-IV Cluster C personality disorders (PDs) in males and females

<table>
<thead>
<tr>
<th>Avoidant PD</th>
<th>Dependent PD</th>
<th>Obsessive-compulsive PD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avoidant PD</td>
<td>*</td>
<td>0.50 (0.44–0.56)</td>
</tr>
<tr>
<td>Dependent</td>
<td>0.59 (0.55–0.63) *</td>
<td>0.22 (0.15–0.29)</td>
</tr>
<tr>
<td>Obsessive</td>
<td>0.25 (0.20–0.31) *</td>
<td>0.26 (0.21–0.31) *</td>
</tr>
</tbody>
</table>

* Results for men are depicted above, and for women below, the diagonal formed by the asterisks.

DEPD (males 0.2%, females 0.3%) and 2.5% \((n=69)\) for OCPD (males 2.5%, females 2.4%).

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### Model fitting

The results of model fitting are shown in Table 2. Based on results from the univariate analyses, we used an ACE model with only quantitative sex-effects as the multivariate model against which nested submodels were compared (model I). Specifying equal parameters for males and females resulted in a non-significant deterioration in fit and an increase in AIC (model II), and the subsequent models were therefore fitted without sex-specific effects. An AE model (without common or specific C) fitted the data well (model III), whereas a CE model without any genetic effects (model IV) was firmly rejected by the \(\chi^2_{15}\) test \((40.78, p<0.001)\), indicating a significant contribution by additive genetic effects on cluster C PDs. Models without common or specific Cs (model V and VI) were both compatible with the data, whereas a model with no specific A (model VII) was rejected by the \(\chi^2_{12}\) test \((29.14, p=0.004)\), indicating that the genetic effects on cluster C PDs are partly specific to each disorder.

The parameter estimates for the best-fitting model (AE, model III) are shown in Fig. 1. Genetic effects accounted for 35% of the variance in AVPD, 31% of the variance in DEPD and 27% of the variance in OCPD. The common genetic factor accounted for 83% of the genetic influence on AVPD, 48% in DEPD and 15% in OCPD. Figure 2 summarizes the proportion of variance accounted for by common and PD-specific genetic and environmental factors. Common A and E factors accounted for 54% of the variance in AVPD and 64% of the variance in DEPD but only 11% of the variance in OCPD, indicating that OCPD is mostly etiologically distinct from the two other cluster C PDs.

### DISCUSSION

To our knowledge, this is the first population-based study of the genetic and environmental influences on DSM-IV cluster C PDs and their inter-relationship.

### Genetic and environmental risk factors in males and females

Familial aggregation of a trait or a disorder can be caused by genetic and/or shared environmental factors. Our results indicate that for AVPD, DEPD and OCPD, familiality is best explained by genetic factors alone, with moderate genetic influence on all three PDs. Given the moderate size of our sample and thus our limited power (Neale et al. 1994; Sullivan & Eaves, 2002) we cannot rule out shared environmental effects. However, in the only twin study of DSM PDs previously published, none of the best-fitting models included shared environment (Torgersen et al. 2000). Heritability estimates for DSM-III-R AVPD, DEPD and OCPD in that study, including mostly patients with severe psychiatric disorders, were 0.28, 0.57 and 0.77 respectively. Confidence intervals were not presented, but the small sample size suggests that they would have been wide. Our results can also usefully be compared to estimates from other...
conceptualizations of PDs. Livesley et al. (1993) and Jang et al. (1996) studied dimensions and facets of PD traits in samples of volunteer twin pairs from the general population. In the largest sample (Jang et al. 1996), no evidence was found for shared environmental effects. Heritability estimates ranged from 0.25 to 0.53 for traits related to cluster C PDs. Numerous studies have shown that DSM PDs can be represented by other models (Trull, 2005), for example the Five-Factor Model (FFM) of normal personality (Widiger & Costa, 2002). The domains and facets related to cluster C PDs in the most popular operationalization of the FFM, the NEO-Personality Inventory Revised (NEO-PI-R; Costa & McCrae, 1992), have been shown to be heritable in the range 29–46%, with no evidence of shared environmental effects (Jang et al. 1998). Our heritability estimates thus appear to be in the low end of those previously reported for personality traits resembling cluster C PDs. Our PD measures include a smaller number of items than most personality trait assessments, and may therefore include a greater proportion of measurement error (Neale et al. 2005), which may explain this result.

No evidence was found for any sex differences in genetic and environmental influences on

### Table 2. Multivariate model-fitting results

<table>
<thead>
<tr>
<th>Model</th>
<th>Sex effects</th>
<th>AC</th>
<th>CC</th>
<th>EC</th>
<th>AS</th>
<th>CS</th>
<th>ES</th>
<th>χ²</th>
<th>df</th>
<th>p</th>
<th>AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>16.67</td>
<td>9</td>
<td>0.05</td>
<td>−1.33</td>
</tr>
<tr>
<td>II</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>17.41</td>
<td>15</td>
<td>0.30</td>
<td>−12.59</td>
</tr>
<tr>
<td>III</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>40.78</td>
<td>15</td>
<td>&lt;0.001</td>
<td>10.87</td>
</tr>
<tr>
<td>IV</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>17.88</td>
<td>12</td>
<td>0.12</td>
<td>−6.12</td>
</tr>
<tr>
<td>V</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>16.81</td>
<td>12</td>
<td>0.16</td>
<td>−7.19</td>
</tr>
<tr>
<td>VI</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>29.14</td>
<td>12</td>
<td>0.004</td>
<td>5.14</td>
</tr>
<tr>
<td>VII</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
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</tr>
</tbody>
</table>

* Best-fitting model.

AC and AS, additive genetic effects; CC and CS, shared environmental effects; EC and ES, unique environmental effects; df, degrees of freedom; AIC, Akaike’s Information Criterion; +, factor estimated in model; −, factor set to zero or constrained in the model.

![Fig. 1](#)

Fig. 1. Best-fitting model with parameter estimates and 95% confidence intervals. AC and EC, common additive genetic and individual environmental factors; AS and ES, disorder-specific genetic and individual environmental factors.

![Fig. 2](#)

Fig. 2. Proportion of variance accounted for by common and specific genetic and environmental factors. AC (■) and EC (□), common additive genetic and individual environmental factors; AS (○) and ES (●), disorder-specific genetic and individual environmental factors; PD, personality disorder. Proportion of variance accounted for by common genetic and environmental factors is below the dotted line.
cluster C PDs. Sex differences were not explored in any of the above-mentioned studies. Evidence has, however, been found for quantitative sex differences in the heritability of Neuroticism (Lake et al. 2000), a trait closely related to AVPD and DEPD (Dyce & O’Connor, 1998). For Axis I anxiety disorders, sex differences in genetic effects have not been reported (Hettema et al. 2005). With our sample size and level of measurement we do not have the statistical power to conclude with confidence that sex effects were not present.

**Common genetic and environmental risk factors**

A common genetic factor accounted for most of the genetic variance in AVPD and about half of the genetic variance in DEPD, consistent with findings from the only family study of cluster C PDs that showed a close familial relationship between AVPD and DEPD (Reich, 1989).

Genetic influences on OCPD were mostly specific to this PD. This is broadly consistent with results from studies using alternative conceptualizations of PDs. The multivariate genetic analyses of lower order PD traits by Livesley et al. (1998) yielded four genetic factors that were remarkably similar to the phenotypic factors identified by principal component analysis. The lower-order trait Compulsivity, which resembles OCPD, appeared to be distinct from other traits both genetically and phenotypically. Anxiousness and social avoidance associated with AVPD and submissiveness and insecure attachment associated with DEPD were phenotypically and genetically related to the same higher-order factor, Emotional Dysregulation. In a population-based sample, Dyce & O’Connor (1998) found that DSM AVPD and DEPD correlated strongly positively with NEO-PI-R Neuroticism and weakly negatively with Conscientiousness. By contrast, OCPD correlated strongly positively with Conscientiousness and weakly negatively with Neuroticism (Dyce & O’Connor, 1998).McCrae et al. (2001) have shown that the five-factor structure of the NEO-PI-R was found on both the phenotypic and genetic level, indicating that genetic influences on both Neuroticism and Conscientiousness are highly trait specific.

In addition to indicating that our constructs are not identifying unique genetic liabilities, the results suggest that some of the same individual environmental experiences influence different PD traits, that is environmental effects are not specific. Common unique environmental factors accounted for most of the environmental variance in DEPD and more than one-third of the environmental variance in AVPD. However, similar to the pattern found for genetic effects, unique environmental factors influencing OCPD were mostly specific to this PD. From this type of study it is not possible to determine which genetic or environmental factors may be involved.

Our results do not provide support for the validity of the DSM-IV cluster C construct in its present form. Given that several phenotypic studies indicate that OCPD stands apart from the other DSM clusters (Kass et al. 1985; Hyler & Lyons, 1988; Sanislow et al. 2002) and twin studies of personality traits show that the phenotypic structure closely reflects the underlying genetic structure (Livesley et al. 1998; Krueger, 2000; McCrae et al. 2001; Livesley, 2005), our results can be viewed as supporting the hypothesis that OCPD represents a separate Axis II secondary domain.

**Limitations**

The results from our study should be interpreted in light of several limitations. First, because of the low prevalence, we were unable to analyze the categorical PD diagnoses. To increase power, we instead examined dimensional representations of the DSM-IV diagnoses conceptualized as the number of criteria (≥1) endorsed. As twin analysis are based on the liability threshold model (Falconer, 1965), it should in principle make no difference if the variable studied is categorical or dimensional as long as the dimensional variable reflects the same underlying liability as the categorical diagnosis. We supported this assumption using multiple threshold tests for each individual criterion and for the dimensional representations of the three PDs. We also compared our model fitting results for OCPD in females (where the prevalence of criteria ≥2 was sufficiently high) and found that the parameter estimates were almost identical. Dimensional representations of DSM-IV PDs (Oldham & Skodol, 2000) have been shown to predict functional impairment as well as categorical diagnoses (Skodol et al. 2005).
Second, although we included a large number of twins, substantial attrition was observed in this sample from the birth registry through three waves of contact consisting of two questionnaires and a personal interview. We will report detailed analyses of the predictors of non-response across waves elsewhere (Harris et al. unpublished observations). In brief, cooperation was strongly and consistently predicted by female sex, monozygosity, older age and higher educational status, but not symptoms of mental disorder. In particular, we assessed PD traits at the second questionnaire with 91 self-report items. We used these items to predict the PD scores from the interview. The polychoric correlations between the scores based on the questionnaires and those from the interview were 0.60 for AVPD, 0.49 for DEPD, and 0.35 for OCPD. Controlling for demographic variables, these weighted scores from the second-wave questionnaire did not predict participation in the personal interview (all \( p > 0.20 \)). While we cannot be certain that our sample was representative with respect to cluster C psychopathology, these findings suggest that a substantial bias is unlikely. Third, the twins were interviewed only once. Although we demonstrated high inter-rater reliability and internal consistency, we could not estimate the test–retest reliability over time. Previous studies have shown that the 2-year test–retest reliability of AVPD and OCPD is relatively low (McGlashan et al. 2005). In twin analyses, measurement errors are reflected in \( E \), which implies that a reduction in reliability would result in decreased heritability estimates. Furthermore, analyses of sum scores may yield biased estimates of variance components of the latent trait. Thus, the analyses reported here may be subject to these biases, which are likely to deflate the familial (A and C) and inflate the non-familial (E) components (Neale et al. 2005). Finally, these results were obtained from a sample of young Norwegian adults, and may or may not extrapolate to other age cohorts or ethnic groups. However, prevalence estimates from a recent Norwegian epidemiological study in a community sample were within the same range as those reported from community studies in other western countries (Torgersen et al. 2001). The participants in our sample were twins. A previous study of personality failed to show any systematic differences between twin and non-twin samples (Johnson et al. 2002).

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**DECLARATION OF INTEREST**

None.

**REFERENCES**


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