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Dimensional representations of DSM-IV Cluster A personality disorders in a population-based sample of Norwegian twins: a multivariate study

KENNETH S. KENDLER[1,2]*, NIKOLAI CZAJKOWSKI[3], KRISTIAN TAMBS[3], SVENN TORGERSEN[5,6,7], STEVEN H. AGGEN[1], MICHAEL C. NEALE[1,2] AND TED REICHBORN-KJENNERUD[3,4]

Departments of [1] *Psychiatry and* [2] *Human Genetics, Medical College of Virginia of Virginia Commonwealth University, Richmond, VA, USA*; [3] *Division of Mental Health, Norwegian Institute of Public Health, Norway*; *Institutes of* [4] *Psychiatry and* [5] *Psychology, University of Oslo, Norway*; [6] *Center for Child and Adolescent Mental Health, Eastern and Southern Norway*; [7] *Nic Waal's Institute, Norway*

ABSTRACT

Background. The 'odd' or 'Cluster A' personality disorders (PDs) – paranoid, schizoid and schizotypal PDs – were created in DSM-III with little empirical foundation. We have examined the relationship between the genetic and environmental risk factors for dimensional representations of these three personality disorders.

Method. These personality disorders were assessed using the Structured Interview for DSM-IV Personality (SIDP-IV) in 1386 young adult twin pairs from the Norwegian Institute of Public Health Twin Panel. Using *Mx*, a single-factor independent pathway twin model was fitted to the number of endorsed criteria for the three disorders.

Results. The best-fit model included genetic and unique environmental common factors and genetic and unique environmental effects specific to each personality disorder. Total heritability was modest for these personality disorders and ranged from 21% to 28%. Loadings on the common genetic and unique environmental factors were substantially higher for schizotypal than for paranoid or schizoid PD. The proportion of genetic liability shared with all Cluster A disorders was estimated at 100, 43 and 26% respectively for schizotypal, paranoid and schizoid PDs.

Conclusion. In support of the validity of the Cluster A construct, dimensional representations of schizotypal, paranoid and schizoid PD are all modestly heritable and share a portion of their genetic and environmental risk factors. No evidence was found for shared environmental or sex effects for these PDs. Schizotypal PD most closely reflects the genetic and environmental liability common to all three Cluster A disorders. These results should be interpreted in the context of the limited power of this sample.

INTRODUCTION

When the Axis II personality disorders were created in DSM-III (APA, 1980), they were grouped into three clusters. DSM-III stated: 'The first cluster includes Paranoid, Schizoid and Schizotypal Personality Disorders. Individuals with these disorders often appear "odd" or eccentric' (APA, 1980, p. 307). DSM-IV-TR added: 'The Personality Disorders are grouped into three clusters based on descriptive similarities … It should be noted that this clustering system, although useful in some research and educational situations, has serious limitations and has not been consistently validated' (APA, 2000, pp. 685–686).

* Address for correspondence: Kenneth S. Kendler, M.D., Virginia Commonwealth University, Virginia Institute for Psychiatric and Behavioral Genetics, Box 980126, Richmond, VA 23298-0126, USA.
(Email: Kendler@vcu.edu)

Prior studies have suggested that familial/genetic factors contribute to the etiology of the three syndromes making up Cluster A: paranoid personality disorder (PPD), schizoid personality disorder (SPD) and schizotypal personality disorder (STPD) (Parnas *et al.* 2005). Therefore, one useful method to evaluate the validity of the Cluster A construct is to determine the degree to which these three syndromes result from of a common set of familial/genetic factors (Robins & Guze, 1970).

Although population-based twin studies with personal psychiatric interviews have proven quite fruitful in clarifying a number of questions about the etiology and nosologic relationship between a range of Axis I psychiatric disorders (e.g. Kendler *et al.* 2003*a*, *b*; Slutske *et al.* 2000), this method has yet to be applied to Axis II disorders.

In this study, we applied multivariate twin methods to a sample of 1386 pairs of young adult twins ascertained from the Norwegian Population-Based Twin Register and interviewed, face to face, with the Structured Interview for DSM-IV Personality (SIDP-IV) (Pfohl *et al.* 1995). Because of the rarity of twins meeting full diagnostic criteria for PPD, SPD and STPD, we analyzed these personality disorders as dimensional traits. Our analyses sought to address two major questions. First, what is the magnitude of the roles for genetic, shared environmental and individual-specific environmental factors in the etiology of dimensional representations of PPD, SPD and STPD? Second (in order to test the validity of the Cluster A construct), to what extent are these factors shared in common in all three Cluster A personality disorders *versus* specific to the individual personality disorder?

METHOD

Sample and assessment methods

Subjects for this study were recruited from the Norwegian Institute of Public Health Twin Panel (NIPHTP). Twins were identified through the Norwegian National Medical Birth Registry, established 1 January 1967, which receives mandatory notification of all live births. The current panel began with 15 370 like- and unlike-sex twins born 1967–1979. Two questionnaire studies have been conducted thus far, in 1992 (twins born 1967–1974) and in 1998 (twins born 1967–1979). Altogether, 12 700 twins received the second questionnaire, and 8045 responded after one reminder (response rate 63%). The sample included 3334 pairs and 1377 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 personality disorders begun in 1999. To reduce the number of interviewed single twins, twins were not approached for interview until preliminary consent had been obtained from both members of the pair. Participants were recruited among 3153 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 3221 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. The reasons for non-cooperation are illustrated by a breakdown of the pairs in which only one twin agreed to the interview. In these pairs, the uncooperative twin had the following outcome: no response to our contacts 96·0%, unknown address 2·9%, and refusal 1·1%. A total of 2794 twins (44% of those eligible) were interviewed for the assessment of personality disorders. 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.

A Norwegian version of the SIDP-IV (Pfohl *et al.* 1995) was used to assess personality disorders. A DSM-III-R version had been used previously in a large community survey in Norway (Torgersen *et al.* 2001), and the DSM-IV version was used in a 25–40 years follow-up study of patients from a Norwegian youth guidance clinic (Helgeland *et al.* 2005). This instrument is a comprehensive semi-structured diagnostic interview for the assessment of all DSM-IV Axis II diagnoses. The instrument includes non-pejorative questions organized into topical sections (e.g. social relationships, work style, emotions) rather than by disorders so that the flow of the interview is more natural. The SIDP-IV was conducted after an extensive interview assessing Axis I disorders, which

helped the interviewers to distinguish long-standing behaviors from temporary states due to an episodic Axis I disorder.

The SIDP-IV uses the '5-year rule,' meaning that behaviors, cognitions and feelings that have predominated for most of the past 5 years are considered to be representative of the individual's long-term personality functioning. In this interview, each DSM-IV criterion is scored as 0 (absent), 1 (subthreshold), 2 (present) and 3 (strongly present).

Interviewers (mostly psychology students in the final part of their training and experienced psychiatric nurses) were trained by professionals (one psychiatrist and two psychologists) with extensive previous experience with the instrument. The interviews, largely conducted face-to-face, were carried out between June 1999 and May 2004. For practical reasons, 231 interviews (8·3%) were conducted 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. They obtained high intraclass (and polychoric) correlations for the number of endorsed criteria at the subthreshold level: PPD +0·92 (+0·94), SPD +0·81 (+0·86) and STPD +0·86 (+0·90).

Zygosity diagnosis

Zygosity was initially determined by questionnaire items previously shown to categorize correctly more than 97% of the pairs in another Norwegian twin cohort (Harris *et al.* 2002). Twenty-four microsatellite markers were then genotyped on a subsample of 676 of the like-sex pairs in the sample. Results from these markers were used as dependent variables in a discriminant analysis with the above-mentioned questionnaire items as independent variables. Seventeen of these pairs with DNA information (2·5%) were found to be misclassified by the questionnaire data and were corrected. From these data, we estimate that in our entire sample, zygosity misclassification rates were under 1%, a rate that is unlikely to substantially bias results (Neale, 2003).

Statistical methods

Using the threshold of $\geqslant 2$ for each item, the proportion of individuals meeting full diagnostic DSM-IV criteria for the Cluster A personality disorders (PPD 0·5%, SPD 0·1% and STPD 0·04%) were too low to permit useful analysis. Rather than examining personality disorders as dichotomous traits, we modeled them as dimensional traits operationalized as the number of endorsed criteria. However, exploratory analyses revealed that defining a criterion to be present if scored at a level of 2 or higher produced statistically unstable results. Therefore, we used the number of endorsed criteria, defining endorsement as a score of $\geqslant 1$. We evaluated with a multiple threshold test whether the four response options for scoring individual criterion in the SIDP-IV reflected levels of 'severity' on a single underlying continuum of liability. The three personality disorders together have 23 criteria. Testing these in each of five zygosity groups produced 115 possible tests. Nine of these failed due to sparse data. Of the remaining 106, only two failed at the 5% level, a lower number than would be expected by chance.

Very few individuals endorsed a high proportion of all the criteria for an individual personality disorder. Therefore, to avoid null cells, we collapsed the criteria count into three to five categories. We tested the validity of this approach by examining the fit of the multiple threshold model, which evaluates whether our categories of the number of endorsed criteria can be treated as differences of severity on a single normally distributed continuum of liability. Five tests (one for each zygosity group) were available for each disorder. None of the 15 tests was statistically significant.

We therefore used a standard liability-threshold model to estimate the genetic and environmental contributions to twin resemblance for dimensional representations of these three personality disorders. For ease of expression, we refer, in the remainder of this article, to *personality disorders* in place of the more accurate but cumbersome term, *dimensional representations* of personality disorders. Liability is assumed to be continuous and normally distributed in the population. Individual differences in liability are assumed to arise from three sources: additive genetic ('A'), from genes whose allelic effects combine additively; shared environment ('C'), which includes all sources shared by members of a twin pair, including family environment, social class, schools; and unique environment ('E'),

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which includes all remaining environmental factors not shared within a twin pair plus measurement error. Monozygotic (MZ) twins within a pair resemble one another because they share all of their A and C components, while dizygotic (DZ) pairs share (on average) half of their A and all of their C. Although it is possible to include non-additive genetic effects such as dominance or epistasis, they are not considered here because the statistical power to detect such effects is very low (Neale *et al*. 1994).

In this paper, we fit a single-factor independent pathway model to ordinal criteria counts for all three personality disorders. The independent pathway model permits the pattern of loadings on the individual disorders to differ across the additive genetic, shared environmental and unique environmental common factors (Kendler *et al*. 1987*b*). This model also includes additive genetic, shared environmental and unique environmental factors specific to the individual disorder. We chose a single common factor model for two reasons. First, for statistical reasons, with only three disorders, models with two common factor models are not identified. Second, the single common factor model instantiates the DSM construct of Cluster A. That is, the degree to which all three Cluster A personality disorders share common genetic and environmental risk factors will be reflected in the relative loadings of the common *versus* disorder specific factors. We did not constrain the disorder-specific unique-environmental loadings to zero as that would imply, unrealistically, that these constructs were measured without error.

We also examined quantitative sex effects, exploring whether the magnitude of the loadings on the genetic or environmental risk factors for the three personality disorders differed significantly in men and women. We did not fit models containing qualitative sex effects (i.e. where genetic or environmental risk factors differ in males and females) for three reasons. First, technical difficulties remain in fitting these models in this kind of multivariate context. Second, our modest number of opposite-sex pair twins gave us very limited power to test these effects. Third, we found no evidence for such effects when we examined each personality disorder one at a time.

Model fitting was performed using the *Mx* statistical package (Neale *et al*. 2003). Alternative models are evaluated by comparing the difference in their χ^2 values relative to the difference in their degrees of freedom (df). Our goal was to obtain the maximal balance between explanatory power and parsimony, which we operationalize by Akaike's information criterion (AIC) statistic (Akaike, 1987), calculated as: $\chi^2 - 2$ df.

Traditional twin models assume 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 constructing variables that reflected, respectively, similarity of childhood and adult environments. The former was indexed by two items assessing the years that the twins were in the same class at school and the years the twins lived in the same residence. The similarity of adult environment was indexed by three items that inquired about the frequency of in-person and telephone contact during the past year and the distance between their current residences. We tested the equal environment assumption using a double entry approach and polychotomous logistic regression in same-sex pairs. The dependent variable was the number of endorsed criteria in twin 2. We controlled for main effects of zygosity, sex, age and level of environmental similarity. Controlling for shared environmental and genetic effects, we tested whether the criteria count in twin 1 interacted with our measure of environmental similarity in predicting the criteria count in twin 2. If the equal environment assumptions were incorrect, then controlling for all these background variables, we would predict that the criteria count in twin 1 would be a better predictor of that count in twin 2 given high *versus* low environmental similarity. We controlled for the correlational structure of our data in these analyses using independent estimating equations as operationalized in the SAS procedure GENMOD (SAS Institute, 2005).

RESULTS

Descriptive statistics

Usable information on the Cluster A personality disorders was available on 1022 males and 1722 females with the following zygosity

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Table 1. *Cluster A personality disorder traits: model fitting results*

		Common factors			Trait-specific factors					
Model	Sex effects	A	C	E	A	C	E	χ^2	df	AIC
I	+	+	+	+	+	+	+	—	—	—
II	−	+	+	+	+	+	+	17·0	9	−1·0
III	−	+	−	+	+	−	+	17·4	15	−12·6[a]
IV	−	−	+	+	−	+	+	23·7	15	−6·3
V	−	+	+	−	+	+	+	271·0	12	247·0
VI	−	+	−	+	+	+	+	17·4	12	−6·6
VII	−	+	+	+	+	−	+	17·0	12	−7·0
VIII	+	+	−	+	+	−	+	12·9	9	−5·1

A, additive genetic effects; C, shared environmental effects; E, unique environmental effects; df, degrees of freedom; AIC, Akaike's information criterion (Akaike, 1987); +, factor estimated in model; −, factor set to zero or constrained in the model.

[a] Best-fit model.

distribution: 221 MZ male pairs, 116 DZ male pairs, 448 MZ female pairs, 261 DZ female pairs, 340 DZ opposite-sex pairs and 22 single twins. The mean age of the twins at interview was 28·2 years (range 19–36). The mean (S.D.) number of criteria met for the three Cluster A personality disorders was: PPD 0·79 (1·18), SPD 0·40 (0·81) and STPD 0·37 (0·73). The percentage of individuals who endorsed 0, 1 or 2 or more criteria were: PPD 57·2, 23·1 and 19·7; SPD 73·6, 18·6 and 7·8; and STPD 72·9, 18·7 and 8·3. The polychoric correlations in MZ and DZ twins for these disorders were respectively: PPD +0·24 and +0·11; SPD +0·29 and +0·14; and STPD +0·27 and +0·18.

Equal environment assumption

We conducted six analyses testing the impact of childhood and adult environmental similarity on twin resemblance for PPD, SPD and STPD. None of these analyses approached significance (all p values $>0{\cdot}10$).

Model fitting

We defined our full model (model I in Table 1) to include quantitative sex effects, common A, C and E factors and individual-specific A, C and E factors for all three personality disorders. This model fit with a −2 log likelihood of 13636·7 with 8347 degrees of freedom. In model II, we constrained all the genetic and environmental parameters to equality in the two sexes. This resulted in a modest improvement in fit (AIC = −1·0). In models III and IV, we set to zero, respectively, all the shared environmental and all the genetic parameters. Model III provided a much greater additional improvement in fit (AIC = −12·6) than did model IV (AIC = −6·3). In model V, we attempted to constrain to zero the common E factor but this resulted in a very poor fit (AIC = +247·0). Although we did not find much support for the presence of aggregate shared environmental effects, we attempted to improve model III by adding either individual-specific (model VI) or common C effects (model VII). However, neither of these models improved the AIC obtained by model III. Because the fits of models I and II were relatively close to each other, we re-examined the evidence for sex-limited effects by taking the best-fit model III and reintroducing quantitative sex effects (model VIII). This did not improve the fit of model III.

The parameter estimates for model III are depicted in Fig. 1 and summarized in Table 2. Four results are noteworthy. First, overall heritabilities were modest for all three personality disorders, ranging from 0·21 for PPD to 0·28 for SPD. Second, the loadings on the common genetic factor differed widely in the three Cluster A personality disorders, being much higher for STPD than for PPD or SPD. Third, genetic factors specific for the personality disorders also differed substantially in magnitude between the three syndromes, estimated at 0 for STPD to 0·20 for SPD. Fourth, loadings on the common unique environmental factor were similar to those of the common genetic factor, being strongest for STPD.

The results summarized in Table 2 indicate that STPD has the strongest and SPD the

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Table 2. *Parameter estimates (and 95% confidence intervals) for genetic and environmental sources of liability to Cluster A personality disorder traits estimated from the best-fit model*

Personality disorder traits	Genetic			Individual-specific environment		
	Common	Specific	Total	Common	Specific	Total
Paranoid	0·09 (0·02–0·18)	0·12 (0·03–0·19)	0·21	0·30 (0·20–0·42)	0·49 (0·39–0·60)	0·79
Schizoid	0·08 (0·02–0·20)	0·20 (0·08–0·30)	0·28	0·18 (0·11–0·26)	0·54 (0·45–0·65)	0·72
Schizotypal	0·26 (0·11–0·36)	0·00 (0·00–0·13)	0·26	0·62 (0·48–0·82)	0·12 (0·00–0·24)	0·74

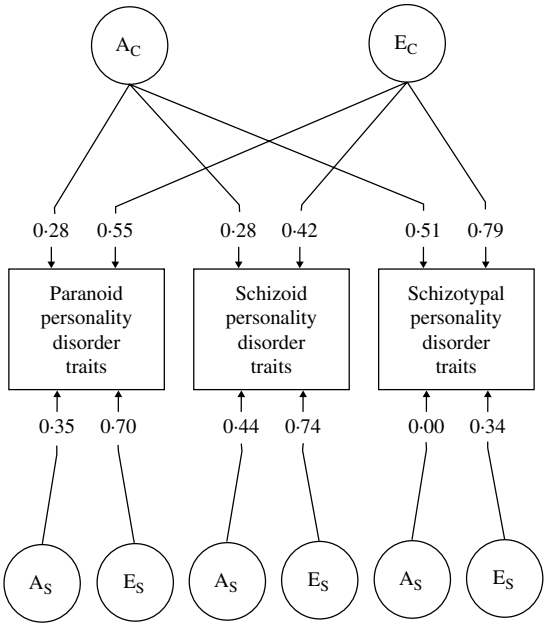

FIG. 1. Parameter estimates from the best-fit model (model III from Table 1). A and E stand for additive genetic and unique environmental factors respectively. The subscripts C and S stand for common and disorder specific respectively.

weakest genetic relationship to the common genetic liability to the Cluster A disorders. This pattern is clearly seen when calculating the proportion of genetic liability to the personality disorder that is shared in common with the other Cluster A disorders. For STPD, PPD and SPD, this figure is estimated at 100, 43 and 29% respectively. A similar pattern of findings is seen for unique environmental effects. The proportion of individual specific environmental liability to the personality disorders that are shared in common with the other Cluster A disorders can be estimated to be 86, 38 and 25% for STPD, PPD and SPD respectively.

One advantage of multivariate twin models is that they permit the decomposition of comorbidity into that portion due to shared genes *versus* shared environmental experiences. The best-fit model predicts the phenotypic correlations between the three Axis II disorders to be: PPD–SPD +0·31; PPD–STPD +0·58; and SPD–STPD +0·48. The genetic contribution to these three correlations is similar. Genetic factors account for 25% of the PPD–SPD correlation, 25% of the PPD–STPD correlation and 30% of the SPD–STPD correlation.

DISCUSSION

We report, for the first time to our knowledge, the inter-relationship of genetic and environmental risk factors for dimensional representations of Cluster A personality disorders in a population-based sample of twins. Five results are worthy of emphasis. First, familial resemblance for these personality disorders could be explained most parsimoniously by genetic effects only. Given the rarity of high scores on these dimensions and our moderate sample size, we cannot rule out with confidence shared environmental effects (Neale *et al.* 1994). However, we found little statistical support for their presence in these analyses. Second, we found no evidence for either qualitative or quantitative sex effects in these data. Again, these conclusions should be viewed in the context of our limited power. However, the available evidence suggests that the role of genes and environment in the etiology of Axis I traits is similar in men and women. Third, the heritability of all three Axis II personality disorders in Cluster A was modest. Genetic factors accounted for only around one-quarter of the total variance in liability to these syndromes. Fourth, the pattern of findings for the individual Cluster

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A syndromes differed substantially. In our results, STPD much more closely reflected the common liability to Cluster A disorders than did PPD or SPD. One plausible reason for this pattern is that the criteria for STPD contain features characteristic of both PPD (suspiciousness, paranoid ideation, ideas of reference) and SPD (social isolation defined by lack of close friends). Fifth, environmental correlations between these three personality disorder traits were large and higher than those seen for the genetic correlations. In decomposing the sources of the correlation in liability across the three Cluster A personality disorder traits, shared environmental factors were considerably more important than shared genetic factors.

Our results can be particularly usefully compared to the prior study of Torgersen *et al.* (2000), who examined 221 personally interviewed twin pairs ascertained through Norwegian psychiatric treatment facilities. Using prevalence rates obtained from an independent Norwegian epidemiologic study, Torgersen *et al.* (2001) also performed univariate twin modeling for all personality disorders. Consistent with our own findings, the AE model provided the best fit for all three of the Cluster A syndromes. Thus, neither study was able to detect substantial shared environmental effects of these three disorders. Torgersen *et al.* (2000) found heritability estimates comparable to those found in this study for PPD (0·28) and SPD (0·29) but found much higher heritability estimates for STPD (0·61). Standard errors for these estimates were not presented, but the small sample size suggests that they would have been substantial.

Our results should also be viewed in context of a number of family and adoption studies that have examined the risk for PPD, SPD and STPD in relatives of schizophrenic and control probands. While a few studies can be found where all three Cluster A personality disorders are at increased risk in relatives of schizophrenic probands (Kendler *et al.* 1993; Parnas *et al.* 1993), more common are studies that find that only STPD (Onstad *et al.* 1991; Torgersen *et al.* 1993; Kety *et al.* 1994; Asarnow *et al.* 2001; Tienari *et al.* 2003) or STPD and PPD (Baron *et al.* 1985) have a significant familial relationship with schizophrenia. These results suggest that STPD is the personality disorder with the closest familial relationship to schizophrenia, followed by PPD and then SPD. This order – STPD, PPD and SPD – is the same as that observed in Table 2 for the proportion of genetic risk due to the common genetic factor. The congruence of these results is consistent with the hypothesis that the common genetic risk factor for Cluster A personality disorders reflects – in the general population – the liability to schizophrenia.

Our results are broadly congruent with a series of twin studies that examine various measures of schizoid-, schizotypal- and paranoid-like traits using self-report questionnaires (e.g. Claridge & Hewitt, 1987; Kendler & Hewitt, 1992; Kendler *et al.* 1987*a*; Linney *et al.* 2003; Jang *et al.* 2005). As in this investigation, these studies have nearly uniformly found significantly heritability for these traits and failed to find shared environmental effects. However, heritabilities are typically higher than those reported here, most frequently in the range 35–60%.

The prevalence of fully syndromal Cluster A personality disorders in this sample was toward the low end of those reported in other community studies and lower than those found in the prior Norwegian epidemiologic sample (Torgersen *et al.* 2001). While our study sampled twins from all over Norway, the prior Norwegian community study of personality disorders took place in the Oslo area. Some evidence suggests that rates for psychiatric disorders may be higher in the urban area of Oslo than in the remaining largely rural areas of Norway (Hammer & Vaglum, 1990; Torgersen *et al.* unpublished results).

Finally, our results provide empirical support for the Cluster A construct. These three personality disorders share a sufficient proportion of their genetic and environmental risk factors that it is reasonable to consider them as parts of a higher-order diagnostic construct. The analyses presented here do not, however, address the question of whether STPD, SPD and PPD form a natural cluster when considered in the context of all the other personality disorders.

Limitations

These results should be viewed in the context of six potentially significant methodologic

limitations. First, because of their rarity, we did not examine the fully syndromal versions of PPD, SPD and STPD. Instead we examined a dimensional representation of these disorders operationalized as the number of endorsed criteria using a low threshold of endorsement. Statistically, we showed that this 'criteria count' was indexing the same liability that underlay the fully syndromal conditions. Furthermore, many in the field have argued that personality disorders are best conceptualized as dimensional rather than dichotomous constructs (Oldham & Skodol, 2000; Skodol *et al.* 2005; Widiger & Samuel, 2005; Widiger & Simonsen, 2005; Cramer *et al.* 2006). However, it is important to note that much of the information in these analyses comes from symptoms reported by individuals who do not meet full diagnostic criteria for PPD, SPD and/or STPD. Second, twins were interviewed only once and so unreliability of measurement was confounded with individual-specific environment. Although we demonstrated high inter-rater reliability, the test–retest reliability is likely to be considerably lower (McGlashan *et al.* 2005). Indeed, heritabilities observed here for the Cluster A personality disorder traits could be lower than those traditionally seen for personality dimensions (Loehlin, 1992) because of lower reliability of our measures. Third, statistical power in these analyses was limited. Although we found no evidence for sex or shared environmental effects on PPD, SPD and STPD, it is quite plausible that such effects existed in our data but were below the threshold of statistical detection (Sullivan & Eaves, 2002). Fourth, these results were obtained on a particular population – young adult Norwegians – and may or may not extrapolate to other cultural and ethnic groups. Fifth, the rarity of cases of schizophrenia-like psychoses in this population-based sample made it impossible for us to test directly the genetic relationship of the Cluster A personality disorders to schizophrenia. Sixth, substantial attrition was observed in this twin sample from the original birth registry through three waves of contact. We 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 by psychiatric symptoms or psychoactive drug use. In particular, we assessed personality disorder traits at the second questionnaire with 90 self-report items (Torgersen *et al.* 2001). We used multiple linear regression analysis to create weightings of these 90 items to maximize our ability to predict the number of criteria endorsed for PPD, SPD and STPD, achieving correlations of +0·42, +0·40 and +0·40 respectively. Controlling for demographic variables, these weighted scores from the second-wave questionnaire did not predict participation in the personal interview (all $p > 0{\cdot}20$). While we cannot be certain that our sample was representative with respect to Cluster A psychopathology, these findings suggest that a substantial bias is unlikely.

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DECLARATION OF INTEREST

None.

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