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ORIGINAL ARTICLE

# A longitudinal twin study of cluster A personality disorders

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**Background.** While cluster A personality disorders (PDs) have been shown to be moderately heritable, we know little about the temporal stability of these genetic risk factors.

**Method.** Paranoid PD (PPD) and schizotypal PD (STPD) were assessed using the Structured Interview for DSM-IV Personality in 2793 young adult twins from the Norwegian Institute of Public Health Twin Panel at wave 1 and 2282 twins on average 10 years later at wave 2. Using the program Mx, we fitted a longitudinal latent factor model using the number of endorsed criteria for PPD and STPD.

**Results.** The stability over time of the criteria counts for PPD and STPD, estimated as polychoric correlations, were +0.34 and +0.40, respectively. The best-fit longitudinal model included only additive genetic and individual-specific environmental factors with parameter estimates constrained to equality across the two waves. The cross-wave genetic and individual-specific environmental correlations for a latent cluster A factor were estimated to equal +1.00 and +0.13, respectively. The cross-time correlations for genetic and environmental effects specific to the individual PDs were estimated at +1.00 and +0.16–0.20, respectively. We found that 68% and 71% of the temporal stability of PPD and STPD derived, respectively, from the effect of genetic factors.

**Conclusion.** Shared genetic risk factors for two of the cluster A PDs are highly stable in adults over a 10-year period while environmental risk factors are relatively transient. Over two-thirds of the long-term stability of the common cluster A PD liability can be attributed to genetic influences.

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**Key words:** Cluster A personality disorders, longitudinal studies, paranoid personality disorder, personality disorders, schizotypal personality disorder.

## Introduction

Cross-sectional studies have shown that genetic factors contribute substantially to risk for cluster A personality disorders (PDs) and associated traits (Claridge & Hewitt, 1987; Kendler *et al.* 1987; Kendler & Hewitt, 1992; Torgersen *et al.* 2000; Linney *et al.* 2003; Jang *et al.* 2005; Parnas *et al.* 2005; Kendler *et al.* 2006). While prior twin studies have suggested, with considerable consistency, that genetic influences on normative personality traits in adulthood are relatively stable over time (McGue *et al.* 1993; Viken *et al.* 1994; Blonigen *et al.* 2008; Bleidorn *et al.* 2009; Kandler *et al.* 2010), we know little about the stability of genetic influences on pathological PDs and traits.

A prior cross-sectional twin study of the three cluster A PDs [schizotypal PD (STPD), paranoid PD (PPD) and schizoid PD] showed that these three PDs, and especially STPD and PPD, share common genetic and environmental risk factors (Kendler *et al.* 2006). In this study, we report on a longitudinal twin study of two of these cluster A PDs – STPD and PPD – assessed at personal interview in a population-based twin sample twice, 10 years apart. We examine these two disorders assessed at two times using a single-factor model that treats STPD and PPD as indicators of a common underlying cluster A vulnerability.

We are particularly interested in determining the stability of the genetic and environmental influences

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on these two PDs, and to what extent this stability is the result of factors that reflect the latent liability shared between the two PDs *versus* factors specific to the individual PDs.

## Method

### *Sample and assessment methods*

Twins were recruited from the Norwegian Twin Registry (NTR) at the Norwegian Institute of Public Health. The twins were originally identified through the Medical Birth Registry of Norway (MBR). The MBR was established 1 January 1967, and receives mandatory notifications of all births. The twins born 1967–1979 ($n = 15\,374$) were invited to participate in questionnaire studies in 1992 and in 1998. Altogether, 12 700 people received the second questionnaire and 8045 twins responded after one reminder (response rate 63%). Zygosity of the twins was determined by the use of questionnaire items for the entire sample and by microsatellite markers for 676 of the same-sex pairs, which in a combined discriminant analysis predicted a zygosity misclassification rate of about 1% of pairs (Harris *et al.* 2006). The NTR is further described in detail elsewhere (Harris *et al.* 2002; Nilsen *et al.* 2012, 2013).

The data used in this study derive from the interview study 'Axis I and Axis II psychiatric disorders in Norwegian Twins' (AI/AII) conducted from 1999 to 2004 (wave 1), and a follow-up interview and questionnaire study (AI/AII FU) conducted in 2010 and 2011 (wave 2). The wave 1 interviews were mainly conducted face to face, although for practical reasons 231 interviews (8.3%) were conducted by telephone. Of the 6442 eligible twins (3153 complete twin pairs that completed the second questionnaire in 1998 plus 68 pairs unintentionally drawn directly from the NTR), 2801 twins (43.5% of the eligible) participated in wave 1 (1390 complete twin pairs and 21 single twins). This low cooperation rate was a result of stringent guidelines then in force that limited all contact with potential participants to two mailed letters.

To maximize participation rate, all the interviews in wave 2 included only a subset of the disorders assessed at wave 1, and were conducted by telephone. Of the 2801 twins that participated in wave 1, 17 had withdrawn their consent to participate in further research, 14 had unknown addresses and 12 had died, leaving 2758 eligible twins that were invited to participate in a follow-up study. Altogether, after two written reminders and telephone contact with non-responders, 2284 twins (987 complete pairs and 310 single twins) were interviewed in wave 2 (82.8% of the eligible). Interviewers at wave 1 were mainly senior clinical psychology graduate students (75%) at the end of their 6-year training course (including at least 6 months of clinical practice) in addition to experienced psychiatric nurses (18%) and medical students (7%). Interviewers at wave 2 comprised senior clinical psychology graduate students (50%), experienced psychiatric nurses (25%), highly experienced clinical psychologists, who also were interviewers at wave 1 (15%), and clinical nurse specialists (10%). At both waves each twin in a pair was interviewed by a different interviewer.

Inter-rater reliability at wave 1 was assessed based on two raters scoring 70 audiotaped interviews. The number of subjects with specific PDs was too low to calculate $\kappa$ coefficients (Cohen, 1960). Intra-class (and polychoric) correlations for the number of endorsed criteria at the subthreshold level (see below) were +0.86 (+0.90) for STPD and +0.92 (+0.94) for PPD. At wave 2, inter-rater reliability was assessed similarly by two interviewers re-scoring 95 audio-recorded interviews. Intra-class (and polychoric) correlations for the number of endorsed criteria at subthreshold level were +0.77 (+0.86) for STPD and +0.80 (+0.91) for PPD. The studies were approved by the Regional Committees for Medical and Health Research Ethics, and informed consent was obtained from all participants after they received a complete description of the study.

A Norwegian version of the Structured Interview for DSM-IV Personality (SIDP-IV; Pfohl *et al.* 1995) was used to assess all 10 DSM-IV PDs at wave 1, and six of these 10 PDs at wave 2: PPD, STPD, antisocial, borderline, avoidant, and obsessive–compulsive. The DSM-III-R and DSM-IV versions of this interview have been used previously in large-scale studies in Norway (Torgersen *et al.* 2001; Helgeland *et al.* 2005). In wave 2, the SIDP-IV interviews were computerized for ease of recording the information. No change of content or flow was introduced. The SIDP-IV is a comprehensive semi-structured diagnostic interview that includes non-pejorative questions organized into topical sections (e.g. social relationships and work style) rather than by individual PDs, thereby improving the interview flow. The SIDP-IV interview was in both waves conducted after an extensive interview assessing DSM-IV Axis I disorders, which helped interviewers to distinguish long-standing behaviors from temporary states resulting from Axis I disorders.

The SIDP-IV uses the '5-year rule', meaning that behaviors, cognitions and feelings that predominated for most of the past 5 years are considered to be representative of an individual's long-term personality. Each DSM-IV criterion is scored on a four-point scale (0=absent, 1=subthreshold, 2=present, or 3=strongly present).

From the interviews in wave 1, 2793 twins had valid data for DSM-IV PDs: 220 monozygotic male (MZM), 117 dizygotic male (DZM), 449 monozygotic female

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(MZF), 259 dizygotic female (DZF) and 340 dizygotic opposite-sex (DZO) twin pairs, in addition to 23 single twins. From the follow-up interview in wave 2, 2282 twins had valid data for DSM-IV PDs: 154 MZM, 76 DZM, 358 MZF, 179 DZF, and 219 DZO twin pairs, in addition to 310 single twins.

### *Modeling*

The liability to STPD and PPD was assessed as the sum (or 'count variable') of the individual DSM-IV criteria endorsed at a subthreshold level. Thus this score ranged from 0 to 7 for PPD and 0 to 9 for STPD. We applied a multiple threshold model to these scores. This was a similar approach taken to analyses of PDs in earlier analyses from this sample (e.g. Kendler *et al*. 2006, 2008; Reichborn-Kjennerud *et al*. 2013). After collapsing infrequent high count values, we utilized maximum count variables with 6 and 7 values for STPD and PPD, respectively. We tested whether our results were consistent with the multiple threshold model – that the count of subthreshold endorsed criteria reflected differing points on a single continuum of liability. We fit a total of 28 models for each sex × zygosity group consisting of all comparisons within and across twins, within and across waves, and within and across PDs. Of the 140 tests, nine had a $p$ value under 10% and only two under 5%, fewer than chance expectations.

The twin model utilized in these analyses, depicted in Fig. 1 (shown for one twin member), has four main features. First, it assumes a latent continuous liability for the cluster A disorder that is indexed both at time 1 and at time 2 by the observed counts of endorsed symptoms of PPD and STPD. Second, this latent liability is defined by the two PDs and their estimated factor loading coefficients that were all fixed to be estimated as a single $\lambda$. In our initial modeling approach, we permitted separate loadings on the common factor for PPD and STPD within time but to be invariant across time. Although we had sufficient degrees of freedom in principle to estimate them separately, in practice the model became quite unstable, which led to constraining all $\lambda$'s to be invariant within and across time. Third, this model decomposes the longitudinal stability of the cluster A PDs into two components: (i) those that reflect a common latent liability to cluster A in the top half of the model and (ii) those that reflect influences that are specific to PPD and STPD in the bottom half of the figure. Fourth, the model assumes that the resemblance of the risk factors over time by means of genetic ($r_g$) and environmental ($r_e$) correlations. Each of these correlations can range from −1.0 to +1.0 and reflects the degree to which variability in the operative genetic and environmental influences are similar at the two times of assessments. At the extreme, a value of $r_g$ or $r_e$ of +1.0 would indicate that the sources of risk remain completely stable in their impact across the assessment times.

Longitudinal common-pathway Cholesky structured twin models were fitted using the full-information maximum likelihood method in Mx (Neale *et al.* 2003). Once the best-fit model was selected, for ease of interpretation, we algebraically converted the Cholesky path decomposition results into genetic and environmental correlations as shown in Fig. 1. Because of the small sample size of DZM and DZO twin pairs assessed at both waves, we were insufficiently powered to test for qualitative or quantitative sex effects (that is, respectively, whether the same genetic factors were influencing cluster A PDs in males and females or whether these effects were of the same magnitude). The model with the optimal balance of explanatory power and parsimony was selected using the Akaike information criterion (Akaike, 1987).

## Results

### *Descriptive findings*

The mean age was 28.2 (range 19–36) years for participants at wave 1 and 37.9 (range 30–44) years for the participants at wave 2. The means of item endorsement counts for PPD at the time 1 interview in males and females were, respectively, 0.74 (S.D. = 1.13) and 0.82 (S.D. = 1.22). At time 2, the parallel results were 0.31 (S.D. = 0.73) and 0.49 (S.D. = 0.99). The means of item endorsement counts for STPD at the time 1 interview in males and females were, respectively, 0.37 (S.D. = 0.75) and 0.43 (S.D. = 0.84). At time 2, the parallel results were 0.22 (S.D. = 0.62) and 0.32 (S.D. = 0.74).

The polychoric correlation for the number of endorsed symptoms across waves was +0.35 [95% confidence interval (CI) 0.32–0.37] for PPD and +0.40 (95% CI 0.36–0.43) for STPD. The parallel figures expressed as Pearson and intraclass correlations were, respectively, +0.27 (95% CI 0.24–0.31) and +0.25 (95% CI 0.20–0.31) for PPD, and +0.30 (95% CI 0.26–0.34) and +0.30 (95% CI 0.23–0.36) for STPD.

### *Model fitting*

Starting from a full model (model I), we set to zero, in models II and III, respectively, all shared environmental and all genetic paths (Table 1). The model fit improved substantially in model II – suggesting no evidence for shared environmental influences on the cluster A PDs. By contrast, the fit of model III deteriorated markedly, indicating the relative importance of additive genetic influences. Working from model II, in models IV and V, we progressively constrained to

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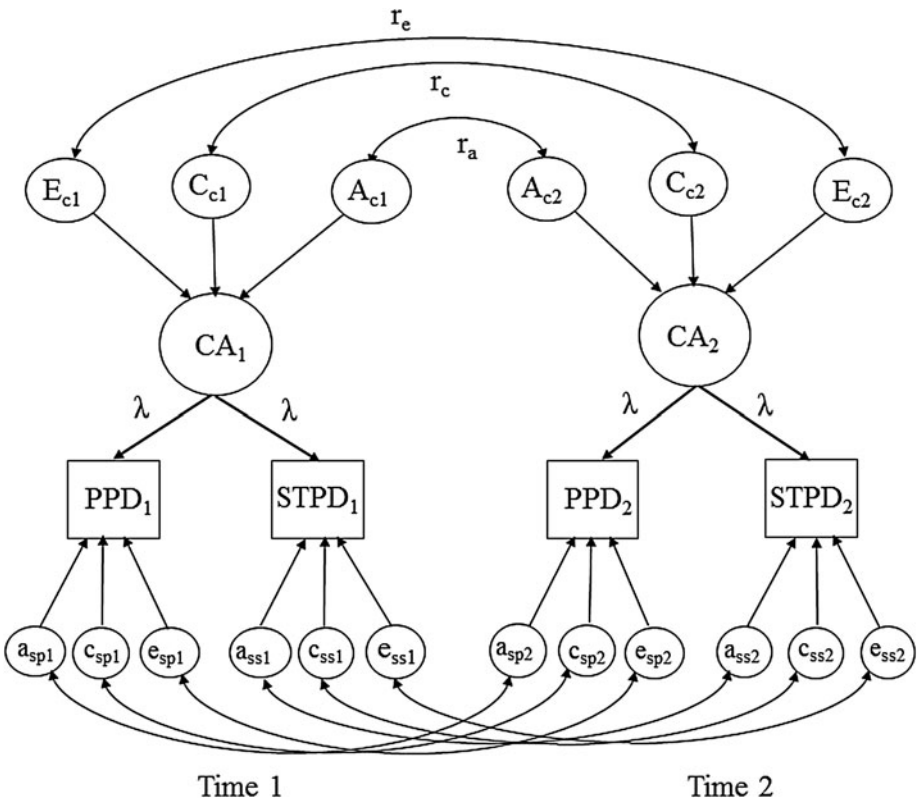


**Fig. 1.** Longitudinal structural equation path model for one member of a twin pair used for the analysis of this sample. $r_e$, Individual unique environmental correlation; $r_c$, shared environment correlation; $r_a$, additive genetic correlation; E, individual unique environment; subscript c, common; subscript 1, time 1; C, shared (or familial) environment; A, additive genetic effects; subscript 2, time 2; CA, cluster A factor; $\lambda$, factor loading; PPD, paranoid personality disorder; STPD, schizotypal personality disorder; subscript sp, specific, paranoid personality disorder; subscript ss, specific, schizotypal personality disorder; subscript c, common across both personality disorders. All variables except CA, PPD and STPD (both 1 and 2) are assumed to have a fixed variance of 1, not shown in the diagram.

unity the genetic correlation ($r_a$) between our time 1 and time 2 assessments for, respectively, the genetic effects on the common cluster A factor and the specific genetic effects for PPD and STPD. In both cases, the model fit improved. Then in model VI, we constrained $A_c$ and $A_s$ (additive genetic effects that were, respectively, common to both personality disorders or specific to an individual disorder) to equality across waves. The fit improved further. Model VI was therefore the best-fit model, for which parameter estimates and 95% CIs are shown in Fig. 2.

There are seven noteworthy features of this model. First, the heritability of latent liability to cluster A PDs is stable over time and equal to 30.3%. Second, the latent genetic factors that influence cluster A PDs are entirely constant over time, with the best-fit model setting the genetic correlation from time 1 to time 2 to equal +1.00. Third, by comparison, the individual unique environmental influences common to both PDs were much less temporally stable, with $r_e$ estimated at +0.13.

Fourth, genetic effects specific to the individual PDs and not shared with the common factor are more pronounced for STPD than for PPD. The total heritability for PPD at time 1 was 23.3%, of which 25% was specific to PPD, and 75% derives from the cluster A common factor. Parallel values for STPD at time 1 were 27.7, 37 and 63%. With very small differences in model estimation, the results were identical at time 2.

Fifth, the genetic effects specific to the individual PDs were also highly stable over the decade between the time 1 and time 2 assessments, both being constrained to unity in the best-fit model. Sixth, the unique environmental influences making an impact solely on the individual-specific components of the PDs were only weakly correlated over time, being estimated at +0.16 for PPD and +0.20 for STPD.

Finally, the model permits us to decompose the sources of the temporal stability of the two PDs into four etiological sources: genetic and environmental effects operating through the cluster A common factor, and genetic and environmental effects influencing the individual-specific PD components. The cross-time correlation for PPD was estimated at +0.34, of which 51, 15, 17 and 17% came, respectively, from each of

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**Table 1.** *Model fitting results for longitudinal model of cluster A personality disorders*[a]

| | A | C | E | Δ $\chi^2$ | Δ df | AIC |
|---|---|---|---|---|---|---|
| I – full[b] | + | + | + | – | – | – |
| II – drop all C effects | + | – | + | +25.0 | 38 | −51.0 |
| III – drop all A effects | – | + | + | +111.8 | 38 | +35.8 |
| IV – constrain $r_a = 1.0$ for common genetic factor | + | – | + | +25.0 | 39 | −53.0 |
| V – constrain $r_a = 1.0$ for specific genetic factors | + | – | + | +25.0 | 41 | −57.0 |
| VI – constrain $A_c$ and $A_s$ to equality across time[c] | + | – | + | +27.5 | 44 | −60.5 |

A, Additive genetic effects; C, shared familial environmental effects; E, individual unique environmental effects; df, degrees of freedom; AIC, Akaike information criterion (Akaike, 1987); $r_a$, additive genetic correlation; $A_c$ and $A_s$, additive genetic effects that were, respectively, common to both personality disorders or specific to an individual disorder.

[a] All comparisons of $\chi^2$, df and AIC are against the full model. The lower the AIC, the better is the overall fit of the model.

[b] For the full model, −2 log likelihood=17070.80, df=10042 and number of parameters estimated = 118.

[c] Best-fit model.

these four sources. Parallel values for STPD were +0.39 and 45, 13, 26 and 16%. Thus common and disorder-specific genetic influences accounted for 68% and 71% of the temporal stability of endorsed criteria for PPD and STPD, respectively.

## Discussion

The primary aim of this research was to investigate the temporal stability of the genetic and environmental influences on cluster A PDs in a general-population adult twin sample interviewed twice a decade apart. Our data and modeling approach permitted a decomposition of the sources of this stability in genetic and environmental factors associated with the latent liability to cluster A PDs as well as the liabilities specific to the individual PDs.

Prior to reviewing our main findings, it is germane to ask whether the stabilities that we observed for PPD and STPD symptoms were in line with expectation. Stability estimates can vary depending on the methods used (Morey & Hopwood, 2013). In addition to absolute stability (e.g. mean level of criteria count over time), differential stability (retest correlations or rank order) is most commonly used. We found three studies reporting long-term differential stability of symptoms of PPD and STPD in adults. Hopwood *et al.* (2013) examined the 10-year rank-order stability of the DSM-IV PD criteria count assessed using a structured interview, calculated as a Pearson correlation, in the Collaborative Longitudinal Study of Personality Disorders and found it to equal +0.39 for PPD and +0.42 for STPD. Nestadt *et al.* (2010) reported a 12- to 18-year follow-up of an epidemiological cohort using a non-structured assessment interview and reported much lower stability – calculated as intraclass correlations – for DSM-III criteria: +0.12 for STPD and −0.06 for PPD. In a longitudinal study, also based on non-structured assessment interviews, in a representative community sample followed from adolescence to adulthood, stability coefficients based on Pearson correlations were 0.42 for PPD and 0.30 for STPD (Johnson *et al.* 2000). Our results were broadly in line with expectation in that they fell between these prior estimates, although clearly higher than those reported by Nestadt *et al.* (2010).

Furthermore, there were substantial declines over 10 years in the level of symptoms of STPD and PPD. The collaborative study found that STPD symptom levels declined over 10 years: 54% in males and 43% in females (Sanislow *et al.* 2009). Johnson *et al.* (2000) reported a decline in symptom levels over 8 years of 46% for PPD and 65% for STPD. These results are broadly similar to what we found, with more modest reductions for STPD symptoms of 41% and 26% in males and females, respectively, with parallel figures for PPD of 58% and 40%.

Turning to our major findings, we would emphasize five of them. First, we found that genetic influences on the cluster A PDs were highly stable over a 10-year period in early to middle adulthood. Indeed, the relevant genetic correlations were set to unity in our best-fit model. These findings are consistent with prior studies of normative personality (McGue *et al.* 1993; Viken *et al.* 1994; Blonigen *et al.* 2008; Bleidorn *et al.* 2009; Kandler *et al.* 2010) and suggest that the temporal stability of genetic influences may be similar across normative and pathological personality traits. However, our results differ from those reported by Ericson *et al.* (2011) who found substantial changes in genetic risk factors for STPD symptoms, as assessed by the Schizotypal Personality Questionnaire (Raine *et al.* 1995) at ages 11 and 16 years. While confirmation by further research is needed, these results – consistent with patterns seen for normative personality (Dworkin *et al.* 1976; Blonigen *et al.* 2008) – suggest that the genetic substrate for pathological personality traits may be variable during childhood and adolescence, and stabilize in early to mid-adulthood.

Second, the correlation between the environmental influences on the cluster A common factor was much lower than that seen for the genetic influences, estimated at only +0.13. In our latent variable model,

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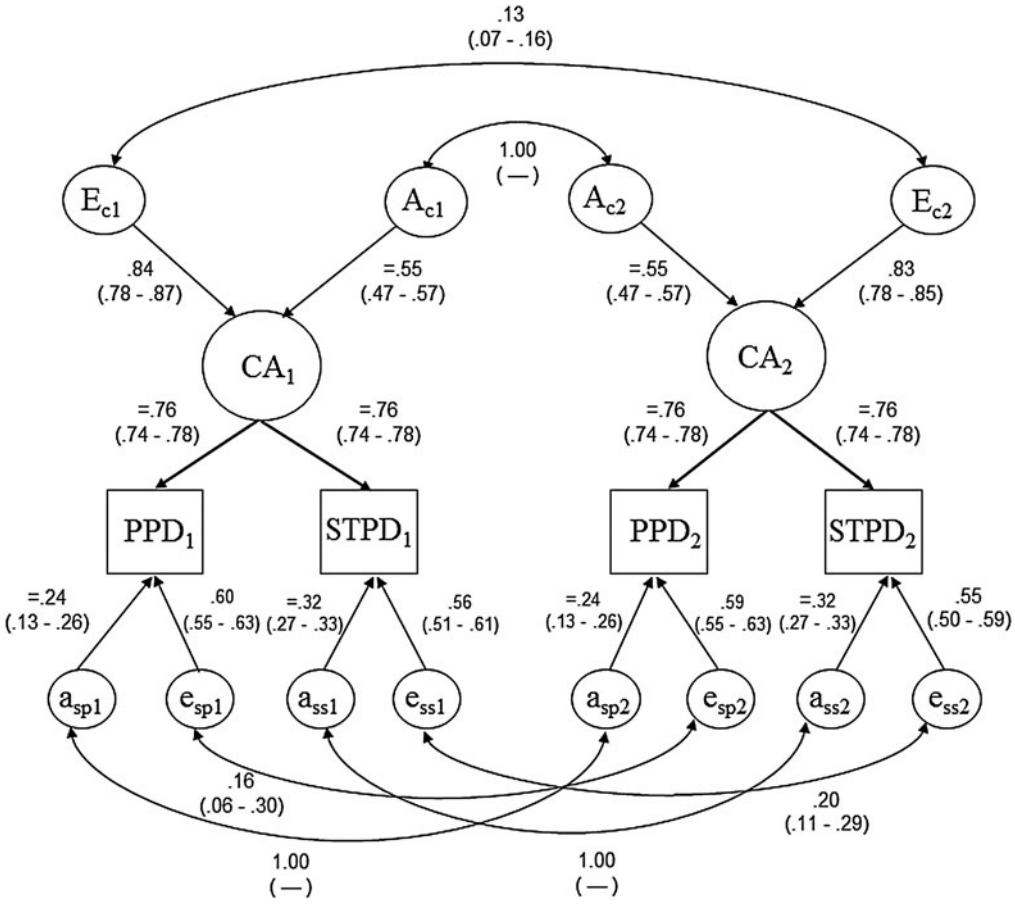


**Fig. 2.** Parameter estimates and 95% confidence intervals in the best-fit model (model V in Table 1) for a longitudinal common-pathway twin model with across time invariance on cluster A paranoid and schizotypal personality disorder. E, Individual-specific environment; subscript c, common; subscript 1, time 1; A, additive genetic effects; subscript 2, time 2; CA, cluster A factor; PPD, paranoid personality disorder; STPD, schizotypal personality disorder; subscript sp, specific, paranoid personality disorder; subscript ss, specific, schizotypal personality disorder. All variables except CA, PPD and STPD (both 1 and 2) are assumed to have a fixed variance of 1, not shown in the diagram.

random errors of measurement should contribute only to the environmental effects specific to each PD. The environmental influences that influence the common factor should reflect either 'true' environmental effects that make an impact, at each time of assessment, on liability to both STPD and PD, or the effects of correlated errors. Our results suggest that the substantial environmental influences on cluster A PDs have low temporal stability and are only quite modestly correlated over a 10-year interval. Unfortunately, our latent variable modeling cannot clarify the specific nature of the environmental risk factors of these PDs which, while important at each time point, were in aggregate of relatively transient effect.

Third, in accord with prior findings from this sample (Kendler *et al.* 2006, 2008), cluster A PDs were only modestly heritable and lower than most prior estimates of schizotypal-like personality features assessed by self-report questionnaires (e.g. Claridge & Hewitt, 1987; Kendler *et al.* 1987; Kendler & Hewitt, 1992; Linney *et al.* 2003; Jang *et al.* 2005). This might be a result of greater errors of measurement associated with interview-based measures.

Fourth, our results are reassuringly similar to our prior analysis of all three cluster A PDs (PPD, STPD and schizoid PD) in our wave 1 data showing that STPD was moderately more heritable than PPD (Kendler *et al.* 2006).

Fifth, from our best-fit model, we could estimate that 68% of the temporal stability of PPD derives from genetic factors, with the parallel figure for STPD being 71%. While environmental experiences that differ between twins have enduring effects on cluster A symptoms, our results suggest that over two-thirds of the long-term stability of these symptoms arise from genetic differences between individuals.

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### *Limitations*

These results should be viewed in the context of four potentially significant methodological limitations. First, because of their rarity, we did not examine the fully syndromal versions of STPD and PPD. Instead we examined a dimensional representation of these disorders operationalized as the number of endorsed criteria using a low threshold of endorsement. Statistically, this 'criteria count' indexes the same liability that underlay the fully syndromal conditions. Furthermore, many in the field have conceptualized PDs as dimensional rather than dichotomous constructs (Oldham & Skodol, 2000; Skodol *et al.* 2005; Widiger & Samuel, 2005; Morey *et al.* 2007). 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 or STPD.

Second, substantial attrition was observed in this twin sample from the original birth registry through to our wave 1 interview. We report detailed analyses of this attrition elsewhere where we show that cooperation was strongly and consistently predicted by female sex, monozygosity, older age, and higher educational status, but not psychiatric symptoms or psychoactive drug use (Tambs *et al.* 2009). Controlling for sex and age, participation at wave 2 was not significantly predicted by level of PPD symptoms at wave 1 ($\chi^2 = 0.00$, $p = 0.98$) but was significantly predicted by level of STPD ($\chi^2 = 5.28$, $p = 0.02$). For every STPD criterion endorsed at wave 1, cooperation at wave 2 declined 17% (95% CI 3–33%). Thus, some attrition bias in our results is possible. However, the full-information maximum likelihood methods used here are robust to missing data when this is either completely random or predicted by other variables in the analysis, which is at least partly the case here.

Third, a major limitation of these analyses was the lack of assessment of schizoid PD at wave 2. Since that interview was by telephone, we had to shorten the assessment and picked two PDs from each of the three clusters. Schizoid PD was the one cluster A PD to be dropped, in part because, in our previous analyses, it had the lowest degree of sharing of common genetic and environmental risk factors with the other cluster A PDs (Kendler *et al.* 2006).

Fourth, our longitudinal sample was poorly powered to detect sex effects on genetic risk factors for cluster A PDs. Nonetheless, we conducted separate exploratory analyses for qualitative and quantitative sex effects. In neither case did we find any evidence for such effects in the data.

## Conclusions

In a population-based twin sample, we assessed at personal interview, 10 years apart, two of the three cluster A PDs. The stability of the criteria count for these PDs was moderate. However, the best-fit longitudinal twin model estimated the cross-wave genetic correlation to be unity. The correlation between the environmental risk factors was much more modest (+0.13). We conclude that the underlying genetic risk factors for cluster A PDs are highly stable over middle adulthood while environmental risk factors are relatively unstable. Over two-thirds of the long-term stability of the common cluster A PD liability can be attributed to genetic influences.

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## Declaration of Interest

None.

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