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ORIGINAL RESEARCH

Genetic and Environmental Structure of DSM-IV Criteria for Antisocial Personality Disorder: A Twin Study

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Abstract Results from previous studies on DSM-IV and DSM-5 Antisocial Personality Disorder (ASPD) have suggested that the construct is etiologically multidimensional. To our knowledge, however, the structure of genetic and environmental influences in ASPD has not been examined using an appropriate range of biometric models and diagnostic interviews. The 7 ASPD criteria (section A) were assessed in a population-based sample of 2794 Norwegian twins by a structured interview for DSM-IV personality disorders. Exploratory analyses were conducted at the phenotypic level. Multivariate biometric models, including both independent and common pathways, were compared. A single phenotypic factor was found, and the best-fitting biometric model was a single-factor common pathway model, with common-factor heritability of 51% (95% CI 40–67%). In other words, both genetic and environmental correlations between the ASPD criteria could be accounted for by a single common latent variable. The findings support the validity of ASPD as a unidimensional diagnostic construct.

Keywords Unidimensionality · Common pathway · Multivariate biometric model · Psychometrics · Diagnostics

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Introduction

Understanding the etiology of antisocial behavior and criminality is important, given their high societal costs. Twin and adoption studies have estimated that genetic influences account for roughly 40% of the variance in antisocial behavior across assessment methods (Rhee and Waldman 2002), but findings from molecular genetic studies have been inconsistent and have failed to replicate (Kendler 2006; Tielbeek et al. 2012; Ficks and Waldman 2014; Salvatore et al. 2015; Pappa et al. 2016). In addition to statistical power issues and potential gene-environment interactions, another possible explanation for mixed findings is that the assessed phenotypes, such as the DSM-IV Antisocial Personality Disorder (ASPD), reflect multiple etiologically distinct factors. This is plausible given that ASPD diagnosis is based on seven different criteria which may or may not reflect a unidimensional liability factor [an eighth criterion, childhood conduct disorder, is required for diagnosis, but often studied separately (Kendler et al. 2012,

2013; American Psychiatric Association 2013; Venables et al. 2014; Derefinko and Widiger 2016)].

Developmental studies of antisocial behavior suggest that it is more heritable when combined with callous-unemotional traits than when these traits are not present; among incarcerated adults, this trait combination is called "psychopathy" (Viding and McCrory 2012). Psychopathy is also frequently modeled as a four-dimensional construct, involving variation along interpersonal, affective, lifestyle, and "antisocial behavior" dimensions (Neumann et al. 2015). What may be confusing to many is that the content of the seven ASPD criteria distribute to all these dimensions instead of just the "antisocial" dimension.

The first ASPD criterion assesses failure to conform to social norms, "as indicated by repeatedly performing acts that are grounds for arrest" (could fit the "antisocial" psychopathy dimension). The second criterion assesses deceitful behaviors (interpersonal dimension). The third criterion assesses impulsivity or failure to plan ahead (lifestyle dimension). The fourth criterion assesses irritability and aggressiveness "indicated by repeated physical fights or assaults" (antisocial dimension). The fifth criterion assesses reckless disregard for safety of self or others (lifestyle dimension). The sixth criterion assesses consistent irresponsibility regarding work behavior or financial obligations (lifestyle dimension), and the seventh criterion assesses lack of remorse, "as indicated by being indifferent to or rationalizing having hurt, mistreated, or stolen from other" (affective dimension). The partial correlations between the psychopathy dimensions and sum of ASPD criteria directly reflect the above content analysis, with the interpersonal dimension (represented by one ASPD criterion) being least correlated with ASPD sum score and the lifestyle dimension (represented by 3 criteria) most correlated with the sum score (e.g., Table 1 in Coid and Ullrich 2010).

While the four psychopathy dimensions are correlated and consistent with "a fundamental link between antisociality and other features of psychopathy" (Neumann et al. 2015), the apparent dispersal of ASPD content across such multiple dimensions rises questions regarding homogeneity of the ASPD construct. Yet, in factor analyses of all the criteria of all or multiple DSM-IV personality disorders, ASPD has been among the disorders that most consistently load onto a single factor (Blais and Norman 1997; Warren and South 2009; Huprich et al. 2010). That is at the "phenotypic" level, referring to study of unrelated individuals. Family studies provide a unique opportunity to further understand the population variation in characteristic patterns of both normal and disordered behavior, or personality (Franić et al. 2013; Livesley 2005).

To our knowledge, only one genetically informative study on the dimensional (factor) structure of ASPD

criteria has been published (Kendler et al. 2012). Kendler et al. (2012) found evidence for two correlated phenotypic factors, dubbed "aggressive-disregard" (ASPD criteria #1, #4, and #5) and "disinhibition" (criteria #2, #3, #6, and #7). Multivariate twin modeling then identified two genetic factors underlying the phenotypic factors (though criterion #7 was not well-represented) and an additional environmental factor, along with criteria-specific genetic and environmental effects. This led the authors to conclude that from a genetic perspective, the DSM-IV criteria for ASPD do not reflect a single dimension of liability, but instead two dimensions of genetic risk reflecting aggressive-disregard and disinhibition influence ASPD. However, they tested only a limited number of biometric models, and assessed the ASPD criteria by self-report questionnaire items that were mapped onto the DSM-IV ASPD criteria.

The previously tested set of biometric models was limited in the sense that it contained only "independent pathway" models and no "common pathway" models (Neale and Maes 2002; Franić et al. 2013; Livesley 2005; Markon and Krueger 2004). This means that genetic and environmental factors were assumed to independently influence the ASPD criteria, even though there is some evidence that "the phenotype of antisocial behavior is much more than a sum of the genetic and environmental parts" (Derefinko and Widiger 2016; Hyde et al. 2016; Viding and McCrory 2012). A common pathway model instead assumes that genes and environment influence an intermediate phenotype (latent factor) that can further influence the criteria, and is a frequently considered alternative for the independent pathway model. In addition to studying common pathway models, it is of interest to extend the biometric results on self-reported ASPD criteria to widely used interview criteria because assessment method moderates the estimated heritability of antisocial behavior (Rhee and Waldman 2002). In this paper, we therefore: (1) replicate the previous multivariate biometric study on the genetic and environmental structure of DSM-IV ASPD criteria using data from structured interviews instead of self-report questionnaire items, and (2) extend the analyses by applying previously unstudied common pathway biometric models in addition to the independent pathway models.

Methods

Sample

Participants in the present study were recruited from the Norwegian Institute of Health Twin Panel, a population-based sample of Norwegian twins (Harris et al. 2002). Psychiatric Axis I and II disorders were assessed at interview in 2801 twins (43.5% of those who were eligible) between

the years 1999 and 2004. Their mean age was 28.2 years and age range 19–36. Zygosity was determined by a combination of questionnaire items and genotyping, resulting in a less than 1% miss-classification rate, which is unlikely to substantially bias results (Neale 2003). The sample has been used in many previous investigations (Kendler et al. 2008; Tambs et al. 2009; Torgersen et al. 2008, 2012; Reichborn-Kjennerud et al. 2013). It included 225 monozygotic (MZ) male-twin pairs with data on ASPD criteria (with 5 pairs lacking the other member), 120 dizygotic (DZ) male-twin pairs (including 3 part pairs), 453 MZ female-twin pairs (4 partial pairs), 267 DZ female-twin pairs (8 partial pairs), and 343 pairs of DZ opposite-sex twins (2 partial pairs); a total of 2794 individuals and 1408 (full or partial) twin pairs. To assess sex effects and to compare with a previous study on same-sex twins (Kendler et al. 2012), we also studied the full pairs of same-sex twins (total $n = 2090$; twin-pair $n = 1045$). Approval was received from The Norwegian Data Inspectorate and the Regional Committee for Medical and Health Research Ethics, and written informed consent was obtained from all participants after a complete description of the study.

Measures

Personality disorders were assessed using a Norwegian version of the Structured Interview for DSM-IV Personality (Pfohl et al. 1995). The method 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). It is a comprehensive semi-structured interview of all DSM-IV Axis II diagnoses, rating the specific DSM-IV criteria according to following guidelines: 0 = not present or limited to rare isolated examples; 1 = subthreshold (some evidence of the trait, but not sufficiently pervasive for the criterion to be considered present); 2 = present (criterion clearly present for most of the time during last 5 years); 3 = strongly present (associated with subjective distress or some impairment in social or occupational functioning or intimate relationships). The criteria were modeled based on an inferred ordered continuous threshold liability model of the endorsed ordinal category frequencies (e.g. polychoric correlations); to lessen the impact of empty cell conditions, the ordinal classes 2 and 3 were collapsed into a single class.

Most of the interviewers were psychology students in their final part of training or experienced psychiatric nurses. They were trained by professionals (1 psychiatrist and 2 psychologists) who had extensive previous experience with the instrument, and they were closely followed up individually during the entire data collection period. Most of the interviews were conducted face to face, but for practical reasons, 231 (8.3%) were obtained by telephone. Each twin in a pair was interviewed by a different interviewer. Inter-rater reliability was assessed based on 2 raters' scoring of 70 audiotaped interviews: intra-class correlation of 0.91 for the number of endorsed ASPD criteria at the subthreshold level has been reported (Torgersen et al. 2008).

Statistical analyses

We first investigated the number of phenotypic factors for the 7 ASPD criteria to map the manifest structure of the criteria, and then proceeded to carry out biometric analysis. Sex differences have been studied a lot for antisocial behavior (Rhee and Waldman 2002), and while we lacked power to adequately test for sex-limited genetic effects (Neale et al. 2006; Torgersen et al. 2008; Reichborn-Kjennerud et al. 2015), we studied structural invariance of ASPD with respect to sex both phenotypically and biometrically, as explained below. This was done using the full pairs of same-sex twins, and when no sex differences were found, the models were estimated for the entire sample.

Phenotypic Analyses First, an omnibus test of sex differences in the phenotypic correlations was conducted using a random permutation test (2000 permutations) on the Frobenius norm of the male–female difference in correlation matrices (i.e., their Euclidian distance) (Good 2005). Permuting the male–female status leaves the twin-dependencies unchanged in the permutation/comparison distribution, and therefore the test is not biased by the non-independence of twins. A polychoric approach for ordinal-item endorsement assumes (in this case) that latent liabilities of individuals to endorse a criterion are normally distributed and an individual endorses the criterion in category "1" when his or her liability exceeds the lower estimated threshold but is below the upper estimated threshold, and endorses it in category "2" when the latent liability exceeds the upper threshold (the lower bound of the first category is −∞ and the upper bound of last the category is +∞). Different criteria can have different thresholds, and men and women can differ in all these thresholds (excluding the "infinity bounds").

Exploratory Factor Analysis (EFA) is a traditional method for investigating the covariance structure among multiple assessment items (e.g. ASPD criteria) to find evidence on shared liability factors (Lawley and Maxwell 1971). The aim is to identify the minimum number of latent factors that can account for the shared covariance among the items. We conducted EFAs using Mplus version 7.31 using the mean- and variance-adjusted weighted-least squares estimator and the complex-sample option (sandwich estimator) to account for the non-independence between twins (Asparouhov 2005; Kendler et al. 2012). Other computations than phenotypic factor analyses were carried out in R software version 3.2.2.

Because the frequently used likelihood-ratio test for factor number can be biased towards extracting too many factors (Hayashi et al. 2007), we used the Parallel Analysis test applied to polychoric correlations to identify the number of factors in our data (Horn 1965; Humphreys and Montanelli Jr 1975). In parallel analysis test, one generates the same number of uncorrelated observations as in the real data to gauge the extent that mere sampling variability inflates observed correlations, or the eigenvalues reflecting them. The usual Scree plot is then compared to the synthetic null-correlation Scree plot to avoid over-extracting factors from sampling variability/noise. Instead of computing effective degrees of freedom, which is a number between the number of twin pairs and the number of twins, we simply show that both the boundary values lead to a same conclusion herein (Jones 2011). In case of disagreement, we also ran a confirmatory factor analysis to verify that the model implied by our EFA supersedes the previously found model, at least in our data. This was done using the "MLR" estimator of Mplus (robust maximum likelihood for non-normal and dependent observations) that allows likelihood-based inference and the information criteria described below.

Differential Item Functioning (DIF) has been previously reported for 3 of the ASPD criteria (Jane et al. 2007). DIF with respect to sex means that one sex endorses a specific criterion differently from the other sex despite adjusting for possible differences in the overall (or 'latent') ASPD between the sexes (Penfield and Camilli 2007). We tested DIF using "lordif" R package, which is an automated procedure for flagging ordinal items with DIF [with options: significance level 0.01, Chi-squared detection criterion, and minimum cell count of 4 (Choi et al. 2011)].

Biometric Analyses Although not a novel idea (Heath et al. 1989; Kendler et al. 1992), researchers have been increasingly interested in the possibility of clarifying diagnostic constructs using samples of twins (Kendler et al. 2008, 2012, 2013; Reichborn-Kjennerud et al. 2013; Franić et al. 2013; Livesley 2005; Jang et al. 2002; Johnson and Krueger 2004). In traditional phenotypic EFA approaches, there is no way to differentiate the contribution of genetic *versus* environmental effects in the covariance between the diagnostic items, and therefore no way to know whether they conflict and thus confound the structural inferences based on EFA (Franić et al. 2013). Because MZ twins share roughly 100% of their segregating genes and DZ twins only on average 50%, their respective criteria correlations can be used to partition the covariance structure of the criteria into distinct genetic and environmental sources of variation (Neale and Maes 2002; Plomin et al. 2012). In the classic twin model, the covariance is partitioned into Additive genetic effects (A), Common or shared environmental exposures that make twins similar (C), and non-shared Environmental effects (E), which comprise all influences

making twins different, including measurement error (Neale and Maes 2002; Plomin et al. 2012). When an EFA-type model is applied to these distinct sources of between-person variation in diagnostic criteria, we will refer to it as "biometric factor analysis".

In a "common pathway model", the diagnostic criteria reflect one or multiple latent factors each possibly influenced by A, C, or E (Neale and Maes 2002; Franić et al. 2013). A or C contributions can be negligible and sometimes are dropped from the model, but E is always included because it theoretically includes ubiquitous measurement errors. Figure 1a illustrates an example of a single-factor common pathway model with four observed criteria. According to the present notation, this is a one-factor model, with only A-E part of the A-C-E partitioning available in twin studies, denoted here by "1-A-E" for the factor part and by "a-e" for the specific-effects part. These models can be extended to include two, three or any number of latent factors each influenced by A, C and E. The biometric factor analysis model can also be specified as an "independent pathway model" wherein each of the criteria is directly influenced by one or multiple genetic and environmental factors. Figure 1b shows a model with only one set of A and E factors. Independent pathway models thus can estimate separate latent factors for each of the modeled A, C, and E covariance components, whereas common pathway models estimate A, C, and E components separately for each of the latent factors. Panels c and d in Fig. 1 exemplify further possible models.

All the previously studied independent pathway models (Kendler et al. 2012) and their corresponding common pathway biometric models were estimated using the "raw data" (full-information maximum likelihood) option of the Open Mx software for structural equation modeling [this makes twin pairs with a missing member usable, implying a total of 2816 informative twins (Boker et al. 2011)]. Confidence intervals (CI) are 95% likelihood-based intervals (Neale and Miller 1997).

The importance of explicitly comparing the common- and independent pathway models is exemplified by recent studies on borderline personality disorder, a diagnosis closely related to ASPD (Torgersen et al. 2008; Reichborn-Kjennerud et al. 2013, 2015). A common-pathway model rather than any of the studied independent-pathway models was found to be the "best" description for the borderline personality criteria (Reichborn-Kjennerud et al. 2013). When comparing models, we used Bayesian Information Criterion (BIC) which has been both applied previously for ASPD and shown to perform well for the models of this type in general (Kass and Raftery 1995; Kendler et al. 2012; Markon and Krueger 2004). BIC is a rough approximation for minus twice the logarithm of Bayes Factor, with a difference of 10 or more being considered as very strong
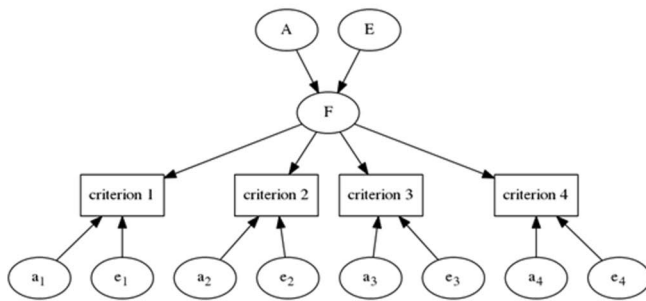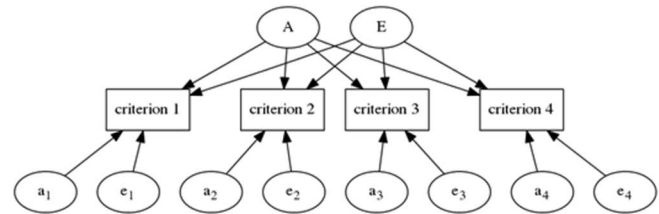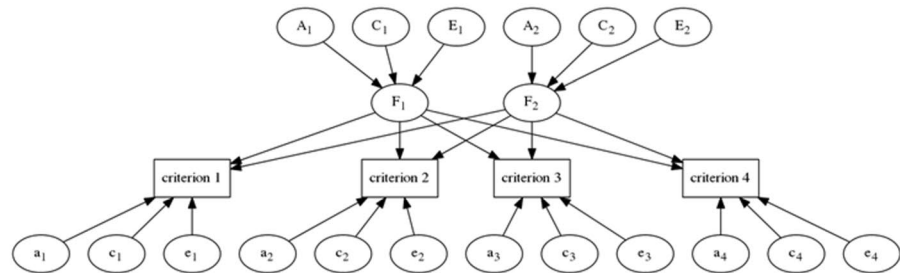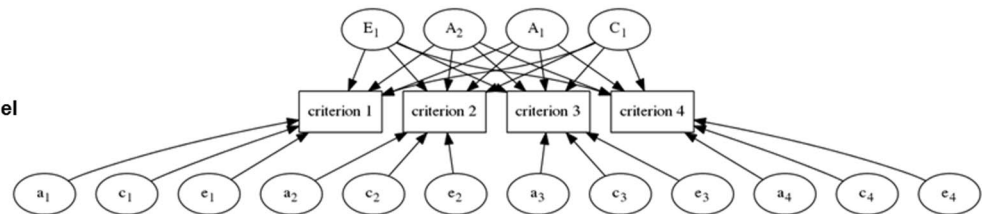
Fig. 1 Examples of alternative common and independent pathway biometric models. **a** A path diagram of a common pathway biometric factor analysis model without shared environmental effects is shown for 4 criteria for illustration. The common factor F is partitioned into heritable variance A and environmental variance E, both of which similarly drive the individual psychiatric criteria. The criteria can have both heritable and environmental specific variances too (*lower-case letters*). Unobserved variables (*ellipses*) have unit variance, but may have distinct loading weights (associated with *arrows*) onto the observed variables (*rectangles*). **b** A path diagram of an analogous independent-pathway biometric factor analysis model (a one-genetic factor, zero-shared-environmental factor, and one-non-shared environmental factor model, i.e. "1A-0C-1E" model, or "1A-1E" model. **c** A path diagram for a biometric model with two common pathways for the A, C, and E variance components. **d** A path diagram for a biometric model with two independent genetic pathways, one non-shared environmental, and one shared environmental pathway

evidence for the model with the lowest BIC, and anything less than 2 barely worth mentioning (Kass and Raftery 1995). As a test of robustness for the selected information criterion, we compared results for those obtained with another well-performing measure, Sample-size Adjusted BIC [SABIC (Sclove 1987; Markon and Krueger 2004; Nylund et al. 2007)].

As in a previous study on ASPD (Kendler et al. 2012), we assessed overall invariance of factor structure over the sexes in a baseline biometric model by constraining its (non-threshold) parameters across the sexes using the same-sex twins only, and if evidence is found for invariance, compared the rest of the models using full data. However, it is possible that the best-fitting biometric model has more statistical power to detect DIF by sex than baseline

models or omnibus tests. A connection between certain factor analysis models and classic DIF exists (Muthén 1989; Muthén et al. 1991; MacIntosh and Hashim 2003), allowing us to evaluate DIF also in the context of the biometric models by comparing models that impose different constraints. We explain this more thoroughly in the supplementary material, and only briefly outline the key points here: (1) if one constrains the factor loadings across the sexes, the modeled criteria are rendered equally sensitive to changes in the latent factor for both the sexes (equal "discriminability" for both sexes in DIF parlance). (2) If one also constrains the residual variances of the factor model, the sensitivity of the criteria for the latent trait has equal mode of inheritance (equal biometric structure) across the sexes. (3) Constraining the latent factor's ACE partitioning

further implies equal mode of inheritance for the latent scores across the sexes. (4) If one further constrains the criteria thresholds except for a constant translation of all the thresholds for the other sex (cf. Supplementary Figure S1), there is *almost* no evidence for DIF, only an overall sex difference in the factor scores (see supplementary material for the exact interpretation and a more explicit explanation of biometric DIFs).

Results

Phenotypic analysis

Table 1 displays the category endorsement rates for each of the ASPD symptoms. Excluding the conduct-disorder criterion, only 11 participants (0.4%) had fully endorsed three or more symptoms as suggested in the diagnostic algorithms, whereas altogether 76 (2.7%) had at least three sub-threshold endorsements, 113 (4.0%) had at least one full endorsement, and 517 (18.5%) had at least one sub-threshold endorsement. Furthermore, 109 (3.9%) fulfilled the conduct disorder, with 426 (15.2%) having a sub-threshold endorsement. No sex differences were found in the phenotypic (polychoric) correlations (ΔFrobenius norm = 1.945, $p = 0.209$), but on average, men endorsed 0.10 ASPD criteria (0.50 if including sub-threshold level) and women only 0.03 criteria (0.18 if including sub-threshold endorsements). Based on contingency table analyses, each individual criterion had a significant sex difference [$\chi^2(1) > 3.858$,

$p < 0.05$ for all], with criterion 6 ("irresponsibility") being borderline significant only [$\chi^2(1) = 3.637$, $p = 0.057$].

The parallel-analysis testing for the number of factors indicated that a single factor was adequate (Fig. 2; for the factor loadings, see Table 1, EFA column). When comparing confirmatory models, the single factor solution (BIC = 6777.7; SABIC = 6711.0) outperformed the previously reported two-factor solution with criteria #1, #4, and #5 loading on the first factor and the rest on the second factor (BIC = 6782.9; SABIC = 6713.0). On the phenotypic level, the automated "lordif" procedure applied to the same-sex twin data flagged just one criterion for DIF, the criterion 6 ("irresponsibility"; all χ^2 indices had $p < 0.001$): given the same total/latent ASPD, women were more likely to endorse the criterion 6 compared to men. Few women had high levels of ASPD, however, and the detected DIF had a negligible effect on the estimated latent ASPD scores (<0.01 s.d. in mean and median difference). In addition, DIF by zygosity can be concern for factor studies (Neale et al. 2005), but also therein the "lordif" procedure flagged just one criterion ("Recklessness"; $p = 0.002$ for uniform DIF, $p = 0.272$ for non-uniform).

Comparison of biometric models

As done previously, we tested in same-sex twins whether constraining all parameters except the thresholds across men and women improved the fit of 1A-1C-1E factor independent pathway and 1-ACE factor common pathway biometric models. In both the cases (supplementary Table S3),

Table 1 Criteria endorsement and 1-factor exploratory factor analysis (EFA) loadings

ASPD criterion	Missing	Not endorsed	Sub-threshold	Endorsed	EFA
Men					
1. Not conforming	2	872	116	34	0.789
2. Deceitfulness	2	916	102	4	0.617
3. Impulsivity, or failure to plan	3	976	33	12	0.725
4. Irritability/repeated fights	2	1006	8	8	0.818
5. Reckless disregard	3	935	70	16	0.608
6. Irresponsibility	2	946	63	13	0.752
7. Lack of remorse	3	983	27	11	0.915
Women					
1. Not conforming	6	1693	58	20	–
2. Deceitfulness	6	1698	69	4	–
3. Impulsivity, or failure to plan	11	1717	40	9	–
4. Irritability/repeated fights	6	1761	3	7	–
5. Reckless disregard	11	1737	22	7	–
6. Irresponsibility	6	1699	58	14	–
7. Lack of remorse	11	1752	11	3	–

The factor loadings are for the full data, including men and women, because no sex differences were detected in the criteria correlation matrix
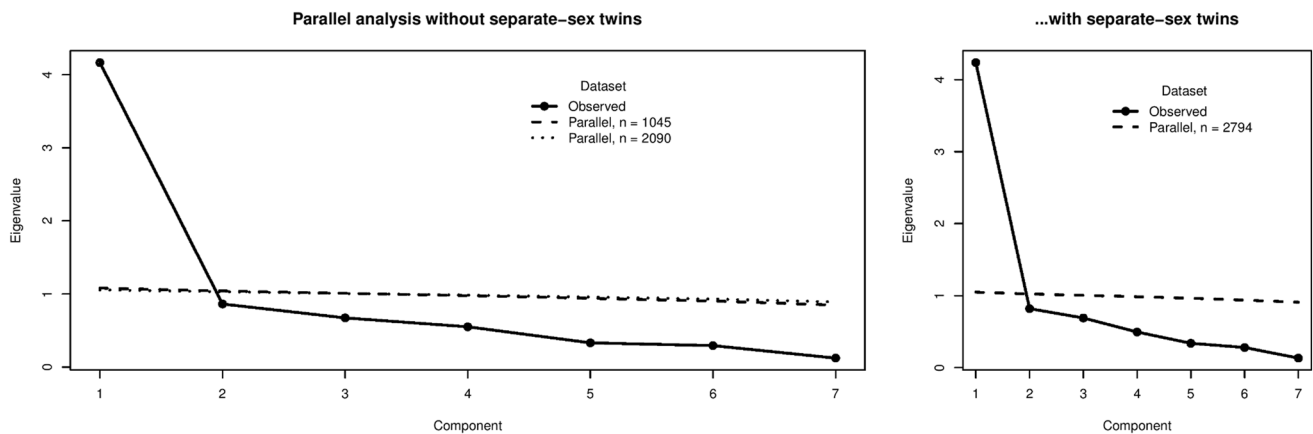
Fig. 2 Scree plot and parallel analysis test for ASPD criteria. *First panel* The *solid line* shows the eigenvalues of the weighted-least squares mean- and variance-adjusted polychoric correlation matrix, whereas the *dashed* (simulated sample size n = 1045) and the *dotted* (simulated sample size n = 2090) *lines* indicate 5th percentile values across 1000 replications in parallel analyses using uncorrelated criteria. Scree-plotted observed eigenvalues above the parallel-analyses

lines represent structure (i.e., factors) over and above sampling variance. The two parallel analyses *lines* simply indicate that both perfect correlation (minimum information) and no correlation (maximum information) between the twins would nevertheless lead to the same conclusion. *Second panel* Same as the *first panel*, but with using the full data, including separate-sex dizygotic twins. The same conclusion holds for both the *panels*

the independent and common pathway models with sex effects absent provided better fits to the data.

In the full data with opposite-sex twins included, the best fitting independent pathway model according to both BIC and SABIC was the Model III that had one genetic factor and one factor for the non-shared environment (Table 2). Among the common pathway models, Model IV had the most parsimonious fit. Importantly, the best common pathway model had a better fit than the best independent pathway model according to BIC, but not according to SABIC. In the same-sex twin data, however, the common pathway model outperformed all independent pathway models also according to SABIC (Table S3). When the conduct-disorder criterion was included, the conclusion was again the same according to BIC, but SABIC picked out yet another model (Table S3). Thus, the common pathway Model IV was the most robust 'best' fit model among those examined. We used this model to evaluate possible biometric DIF in the same-sex twin data.

The common-factor Model IV was estimated with all the parameters set free across the sexes (BIC = −97011.4; SABIC = 4778.3; df = 14,592), by constraining only factor loadings to be equal across the sexes (ΔBIC = −37.8; ΔSABIC = −22.6; Δdf = 6), by constraining both factor loadings and specific/residual variances to be equal across the sexes (ΔBIC = −78.4; ΔSABIC = −67.4; Δdf = 13), by fixing all the parameters across the sexes, except for the ordinal criterion thresholds (ΔBIC = −93.3; ΔSABIC = −75.9; Δdf = 15), and by constraining all parameters to be equal across sex except for a uniform scalar translation in men's criteria

endorsement liabilities relative to women (i.e., no other differences but overall higher endorsements in men). This latter model provided the best fit to the data (ΔBIC = −137.1, ΔSABIC = −78.4). Thus, we did not detect any omnibus sex differences in the biometric measurement models.

The best fit biometric model

Although models with multiple common pathways were tested (Table 2 and Table S3), the best-fit model had only one factor with 51% heritability (CI = 40–67%) and 49% contribution from non-shared environment (CI = 33–65%), with no shared environmental effects. Figure 3 shows a path diagram and parameter estimates for this model. Clearly the genetic and non-shared environmental effects conform to the same factor we observed in the phenotypic analysis (Table 1). However, statistically significant criterion-specific genetic effects emerge in the biometric analysis [χ^2(7 d.f.) = 20.6, p = 0.004 in likelihood-ratio test], showing that the unique variances of EFA contain more than just measurement errors. Based on the estimated thresholds' scalar shift men were on average 0.48 standard deviations (CI = 0.39–0.58) higher on the liability to endorse any of the ASPD criteria compared to women. The best-fit model directly implies the extent of genetic and environmental contributions per criterion, and what proportion of these are attributable to the common co-variation among the ASPD criteria. For readability, Table 3 provides the values.

Table 2 Comparison of biometric models

Model	Common factors	Specific factors	Δdf	ΔBIC	ΔSABIC
Independent pathway models	#A-#C-#E	a-c-e			
II	1A-1C-1E	a-c-e	–	–	–
III	1A-0C-1E	a-e	14	**−88.7**	**−44.2**[a]
IV	0A-1C-1E	c-e	14	−67.2	−22.7
V	2A-0C-1E	a-e	8	−68.8	−43.3
VI	1A-0C-2E	a-e	8	−63.2	−37.8
VII	3A-0C-1E	a-e	3	−37.8	−28.2
Common pathway models	#(A-C-E)	a-c-e			
II	1-ACE	a-c-e	12	−43.5	−1.28
III	1-CE	c-e	20	−88.7	−21
IV	1-AE	a-e	20	**−101.0**[a]	**−33.7**
V	2-ACE	a-c-e	0	−7.9	0.22
VI	2-CE	c-e	10	−61.7	−21.8
VII	2-AE	a-e	10	−79.5	−39.6
VIII	3-AE	a-e	-2	−2.8	3.1

Numbers of factors in a biometric ACE model are given in the form A-C-E with the number of factors in front of the *letters*. Presence versus absence of criterion-specific effects is indicated by corresponding sub-set of "a-c-e" components. We denote e.g. an independent-pathway model with 2 A-factors, 0 C-factors and 1 E-factor by "2A-0C-1E", and the presence versus absence of its specific effects by a subset "a-e" of the full variance decomposition "a-c-e". Number of factors for a common-pathway model is just a single quantity (e.g., "2-AE" for two-factor model for additive and non-shared environment, assuming shared environmental effects are negligible). Model numbering follows a previous study (Kendler et al. 2012)

Baseline values (independent-pathway Model II) were 19 470 degrees of freedom (df), Bayesian Information Criterion (BIC) value of −134 855.7, and sample-size adjusted BIC (SABIC) value of 6 613.2

Δ*df* denotes change in model degrees of freedom compared to the baseline model, and Δ*BIC* denotes change in BIC. Lower BIC values indicate more parsimonious (i.e. better) model. Δ*SABIC* denotes change in SABIC. Lower SABIC values indicate more parsimonious (i.e. better) model

[a]The overall best fit according to the information criterion. Bold font indicates the overall best fit within the two subsets of models considered separately, for independent-pathway models and for common-pathway models

Discussion

To our knowledge this is the first study to fully explore the genetic and environmental factors underlying DSM-IV and −5 ASPD criteria by comparing both independent and common pathway biometric models using structured interview data and a population-based sample of twins. Our main findings indicate that a single, highly heritable common factor could account for the correlations between the ASPD criteria, thereby suggesting that ASPD reflects a single shared dimension of liability, plus criterion-specific liabilities. This supports the validity of ASPD as a diagnostic construct (Franić et al. 2013), and supports the use of ASPD diagnosis in molecular genetics studies more than the previously reported two-dimensional genetic structure.

Because the content of the ASPD criteria is dispersed across several psychopathy factors (Coid and Ullrich 2010), our findings are of relevance for psychopathy research. We found that the genetic and environmental influences on multiple relevant behaviors (the ASPD criteria) are statistically associated rather than independent. This aligns with the observations that full-blown psychopathy usually involves both genetic and environmental exposures (Derefinko and Widiger 2016; Hyde et al. 2016; Viding and McCrory 2012). However, another study has reported statistically independent genetic and environmental factors, as discussed next.

Our findings differ from a previous study using twin self-report data and examining a more restricted set of biometric models (Kendler et al. 2012). The study found evidence for two genetic factors and an independent environmental factor, plus criterion-specific genetic and environmental influences. In that study, "lack of remorse" did not load strongly onto the genetic factors and had a low overall heritability. The differences between this study and the previous study may be due to the range of models tested, the difference in the assessment formats (questionnaire *versus* interview) that can affect heritability estimates (Rhee and Waldman 2002), and sample differences discussed below.

First, the set of models studied by Kendler et al. (2012) did not include common pathway biometric models that would allow statistical dependence between the genetic and
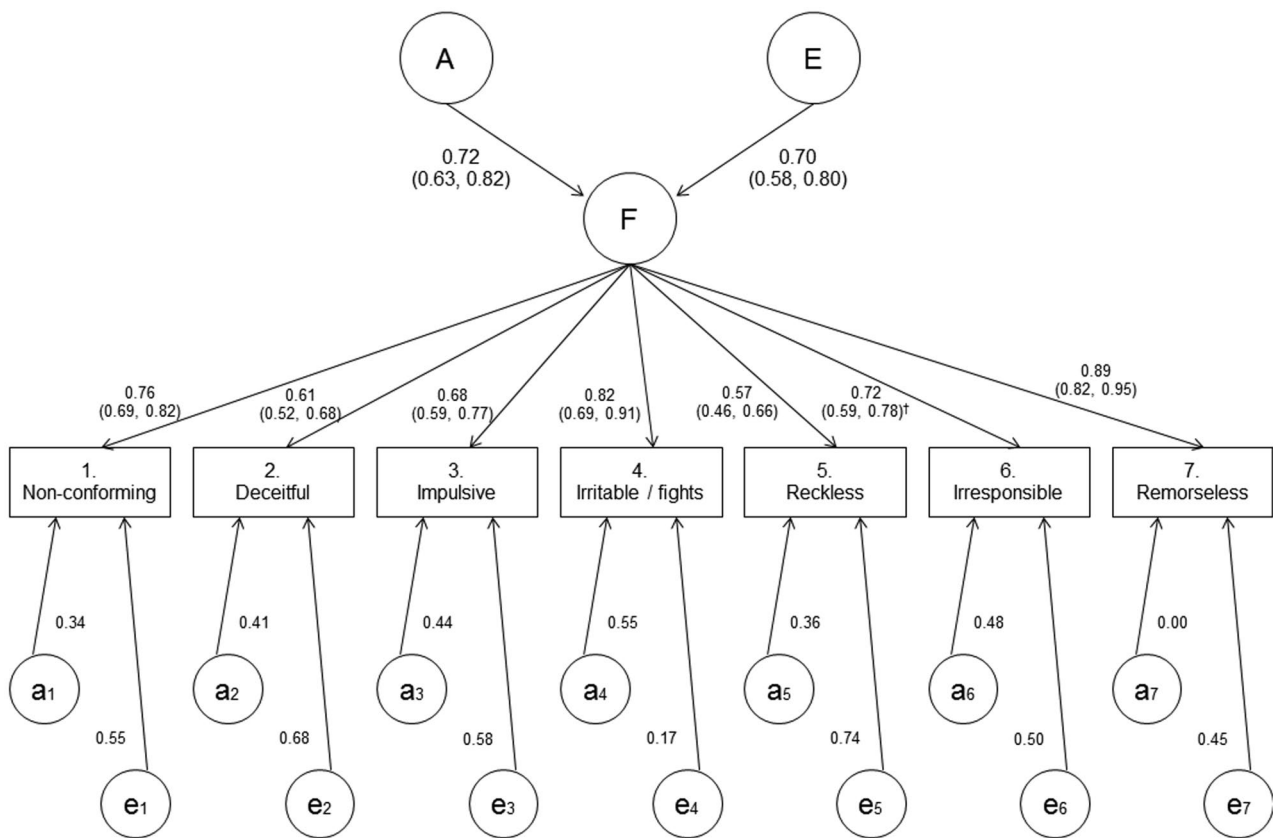
Fig. 3 Path diagram and parameter estimates of the best-fitting common-pathway biometric model. Estimates are from the model with scalar-translated rather than freely estimated threshold parameters, because that model was the best fit to data, but with all data, including separate-sex dizygotic twins. The numbers in parentheses provide 95% likelihood-profile confidence intervals. Superscript "†" refers to the one factor-loading interval estimate that did not properly converge and was estimated as the equivalent supplementary model on the same-sex twins only (see online supplement). Furthermore, reliable confidence intervals for the specific effects were unattainable, but an omnibus test indicated that also the genetic specific effects were significant (p = 0.004). Squares of the path coefficients give corresponding variance proportions: e.g., the common-pathway factor explained $100\% \times 0.89^2 = 79\%$ of the total variance in remorselessness, or criterion 7, of which $100\% \times 0.72^2 = 52\%$ is heritable variation. Table 3 lists the total heritabilities of the criteria

environmental influences, and therefore could not find such dependencies. Second, antisocial individuals may have weak introspective abilities. Co-twin's antisocial behavior is more accurately observed than own behavior; yet, those who are antisocial are generally less likely to perceive others as antisocial (Kendler et al. 2002). In their theoretical analysis of the effects of DIF by zygosity on estimation of heritability, Neale et al. (2005) discussed self-reported antisocial behavior as a possible case for this source of bias. Third, the genetic "aggressive-disregard" factor in the previous study reflected precisely the 3 items that have shown DIF with respect to sex in another study (Jane et al. 2007; Kendler et al. 2012). As we did not find strong indications of DIF by sex, also these differences between the samples could play a role. When multiple items show DIF with respect to the same variable (e.g., sex), the variation in this variable could show up as a common factor for the items. It would be tempting to think that an omnibus test for sex effects is sufficient to guard against all adverse effects of DIF, but significant findings in such a test also depend on its statistical power, whereas the detected factor number depends on another test that may or may not have comparable statistical properties. Here we did not detect DIF for multiple items with respect to the assessed variables, but assessing possible DIF for other assessment formats and/or samples might explain differences across findings.

In general, all the DSM-IV/DSM-5 ASPD criteria tend to load strongly on the same phenotypic factor (Blais and Norman 1997; Warren and South 2009; Huprich et al. 2010). Differential diagnosis has been problematic, however, since many ASPD criteria (e.g., irresponsibility, aggressiveness, impulsivity) may also be associated with other DSM-5 diagnoses, such as borderline PD, schizophrenia, bipolar disorder, or major depression (Blais and Norman 1997; Derefinko and Widiger 2016). Thus, the possibility that ASPD differs phenomenologically between

Table 3 Total genetic and environmental variance of Antisocial Personality Disorder criteria and the percentages attributable to the common factor

Criterion	Genetic variance		Environmental variance	
	a^2	Common factor%	e^2	Common factor%
1. Not conforming	0.41	74	0.59	48
2. Deceitfulness	0.36	54	0.64	28
3. Impulsivity, or failure to plan	0.44	57	0.56	41
4. Irritability/repeated fights	0.67	53	0.33	100
5. Reckless disregard	0.28	61	0.72	22
6. Irresponsibility	0.49	56	0.51	49
7. Lack of remorse	0.42	100	0.58	66

The values correspond the best-fit model illustrated in the Fig. 3. "Common factor%" refers to the percentage of the variance component that is attributable to the biometric common-pathway factor, the rest being attributable to the influences specific to the given criterion

a^2 heritability or proportion of variance in liability to endorse a criterion because of genetic factors, e^2 proportion of variance in liability attributable to environmental factors

individuals who satisfy *versus* do not satisfy a specific characteristic criterion, such as "lack of remorse", has been investigated (Goldstein et al. 2006). Even among those who fulfilled the ASPD diagnostic criteria, lack of remorse was associated with violent behaviors. Among the other criteria, especially criterion #4 ("irritable/fights") was associated with lack of remorse in those who obtained the ASPD diagnosis. Nevertheless, the authors concluded that there was no sufficient evidence to propose that lack of remorse would characterize a subtype of ASPD, instead suggesting "multivariate analytic approaches to examining phenomenologic heterogeneity within the ASPD diagnosis" as a "potentially fruitful avenue for future investigations" (Goldstein et al. 2006). This study represents one such analysis. Our main findings align with these previous phenotypic observations in the sense that we found a clear main factor (no subtypes), with "irritability/aggression" and "lack of remorse" as its strongest representatives.

However, the specific genetic effects we found also imply that some people who are relatively low on the latent ASPD trait can nevertheless have a stable tendency to fulfill the diagnostic criteria. Although the genetic 'residual' liabilities are uncorrelated in the population, some individuals by chance end up having multiple residual contributions. While the specific effects in classic EFA are frequently interpreted as (unstable) measurement errors, such an interpretation does not carry over to specific *genetic* effects in biometric factor analysis because the measurement errors are already contained in the environmental specific effects. This means that simple aggregates of ASPD criteria, such

as sum scores or DSM-IV and DSM-5 diagnoses, will contain both genetic influences related to overall ASPD and genetic influences unrelated to overall ASPD (i.e., influences specific to a single criterion).

Our findings have implications for nosology (Livesley 2005; Kendler 2006), psychometrics (Livesley 2005; Franić et al. 2013), molecular genetics (Tielbeek et al. 2012; Salvatore et al. 2015), developmental psychopathology (Hyde et al. 2016; Viding and McCory 2012), and human behavioral ecology and evolution (Nettle et al. 2013; Ribeiro da Silva et al. 2015; Del Giudice et al. 2011; Colman and Wilson 1997; Ellis 1988). Our study is relevant to all these research fields in providing evidence that a unidimensional ASPD phenotype exists and permeates the domains of genetic and environmental influences. Suggestive of robustness in findings, ASPD and borderline personality share much of their heritability (Kendler et al. 2008; Torgersen et al. 2008; Reichborn-Kjennerud et al. 2015), and a biometric analysis found a common pathway model superior to the tested independent pathway models also in the case of borderline personality disorder (Reichborn-Kjennerud et al. 2013). Furthermore, theoretical arguments distinguish these two personality disorders from other personality constructs that have not shown unambiguous common pathway structure (Brüne 2016; Ribeiro da Silva et al. 2015; Franić et al. 2014). Nevertheless, the findings reported here should be interpreted in light of the following limitations.

Limitations

Finding that a single-factor common pathway model fit the multivariate data for the DSM-IV ASPD criteria best is consistent with the notion of a single mechanism that generates variation along a single dimension, but not a sufficient condition to exclude all other possible explanations. For example, it has been shown by means of theoretical analysis that a causal process of "mutualism" may generate data that fits well with models of underlying latent cause even if there is not one (van der Maas et al. 2006). Dynamic developmental cascades among 'criteria' might create correlation structures that give a false impression of a latent factor. This could also confound genetic correlations if the triggering criteria are partly heritable, causing the entire developmental cascade to reflect the same heritable triggers. However, the most salient indicator item in the common pathway model estimated in this study was "lack of remorse", which appears consistent with a biological mode of strategic behavior that emphasizes exploitation over cooperation (Del Giudice et al. 2011; Ribeiro da Silva et al. 2015).

Another possible limitation is that the attrition in our sample could have had an effect on the structural estimates. In a previous study, nonparticipation in the sample

was predicted by dizygosity, male sex, being married, having children, lower education, and few indicators of poor mental and somatic health and unhealthy lifestyle (Tambs et al. 2009). However, one might generally expect attrition to introduce complex dependencies and thereby weaken rather than strengthen the evidence for a single-factor common pathway structure. Thus, the moderate attrition effects (Tambs et al. 2009) appear to be an unlikely source of bias for our main findings. As a general limitation applicable to all related studies, optimal information criterion for purposes of model selection is still a debated topic (Markon and Krueger 2004; Nylund et al. 2007; Vrieze 2012; Bulteel et al. 2013), and even in the cases where BIC indicated very strong support for a model, SABIC did not always do so (Table 2 and Table S3).

This study was limited to ASPD criteria and the extent to which the common pathway etiology of the ASPD criteria corresponds to the biometric structure of psychopathy remains an open question (Derefinko and Widiger 2016; Wygant et al. 2016). Moreover, a full diagnosis of ASPD requires a presence of conduct disorder before the age of 15 years, and analytic treatment of conduct disorder varies across studies (Jane et al. 2007; Kendler et al. 2012, 2013). However, in the online supplement we present results showing that its inclusion made relatively little difference here. Due to statistical-power considerations, we did not explicitly study genetic sex-limitation in the sense of assessing whether male and female ASPD could be associated with distinct pools of genes (Neale et al. 2006), but previous studies have not found such differences in adolescent or adult antisocial behavior (Jacobson et al. 2002; Larsson et al. 2006). Overall, only few people in our sample exceed the usual diagnostic thresholds and the generalizability of the results is therefore dependent on the dimensional nature of the studied phenomena (Marcus et al. 2006).

When there are unaccounted clustering (dependencies) in data, likelihood-based statistics are not necessarily reliable (Pornprasertmanit et al. 2014). We modeled twin-dependencies similarly to the previous study (Kendler et al. 2012), but neither those nor our phenotypic factor analyses explicitly took into account the *different* phenotypic dependence structures across MZ and DZ twins. Nevertheless, both the studies obtained highly consistent phenotypic and biometric results, albeit different ones.

Compliance with Ethical Standards

Conflict of interest Tom Rosenström, Eivind Ystrom, Fartein Ask Torvik, Nikolai Olavi Czajkowski, Nathan A. Gillespie, Steven H. Aggen, Robert F. Krueger, Kenneth S. Kendler, Ted Reichborn-Kjennerud declare that they have no conflict of interest.

Ethical Approval Approval was received from The Norwegian Data Inspectorate and the Regional Committee for Medical and Health Research Ethics, and written informed consent was obtained from all participants after a complete description of the study.

Human and Animal Rights All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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