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A common genetic factor explains the association between psychopathic personality and antisocial behavior

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ABSTRACT

Background. Both psychopathic personality traits and antisocial behavior are influenced by genetic as well as environmental factors. However, little is known about how genetic and environmental factors contribute to the associations between the psychopathic personality traits and antisocial behavior.

Method. Data were drawn from a longitudinal population-based twin sample including all 1480 twin pairs born in Sweden between May 1985 and December 1986. The twins responded to mailed self-report questionnaires at two occasions: 1999 (twins 13–14 years old), and 2002 (twins 16–17 years old).

Results. A common genetic factor loaded substantially on both psychopathic personality traits and antisocial behavior, whereas a common shared environmental factor loaded exclusively on antisocial behavior.

Conclusions. The genetic overlap between psychopathic personality traits and antisocial behavior may reflect a genetic vulnerability to externalizing psychopathology. The finding of shared environmental influences only in antisocial behavior suggests an etiological distinction between psychopathic personality dimensions and antisocial behavior. Knowledge about temperamental correlates to antisocial behavior is important for identification of susceptibility genes, as well as for possible prevention through identification of at-risk children early in life.

INTRODUCTION

Psychopathy is a serious personality disorder defined by callous and unemotional affects, a grandiose and manipulative interpersonal style, and pervasive impulsive and irresponsible behavior (Cleckley, 1941; Hare, 2003). Psychopathic personality disorder has been linked to antisocial behavior in adults (Hare *et al.* 2000; Hare, 2002). A better understanding of the etiology underlying the personality profile involved in severe antisocial behavior is important for the identification of effective intervention targets.

How to best conceptualize psychopathy and how many symptom dimensions that best describes the disorder is yet unresolved (Cooke & Michie, 2001; Hare, 2003; Cooke *et al.* 2004). Factor analysis of psychopathic traits have found two (e.g. Hare, 1991; Lilienfeld & Andrews, 1996; Frick *et al.* 2000; Benning *et al.* 2003), three (Cooke & Michie, 2001; Andershed

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et al. 2002; Forth *et al*. 2003), and four (e.g. Hare, 2003) dimensions underlying these traits. Nevertheless, contemporary definitions of psychopathy usually include at least three dimensions: an arrogant, grandiose, and deceitful interpersonal style, a deficient affective experience, and an impulsive behavioral style (Cooke & Michie, 2001; Hare, 2003). Regardless of the debate concerning the structure of psychopathy in the adult literature, there is a growing interest in the study of developmental aspects of this personality disorder (Kotler & McMahon, 2005). This research seeks to identify the developmental origins of adult psychopathy and to understand the mechanisms behind the development of antisocial behavior. Thus, the purpose of the downward extension of psychopathy to youths is not necessarily to assign a formal diagnosis of psychopathic personality disorder to children or adolescents, but rather to understand the etiology underlying this socially devastating personality profile.

Existing research suggest that psychopathic-like personality traits can actually be meaningfully assessed in adolescence (e.g. Andershed *et al*. 2002; Forth *et al*. 2003; Vitacco *et al*. 2003; Lynam & Gudonis, 2005). There is an association between these personality traits and antisocial behavior in adolescents, similar to that seen in adults (Lynam & Gudonis, 2005), and the presence of psychopathic-like personality traits identifies a subgroup of antisocial youths with a more pervasive and severe aggressive behavior than that of other youth with conduct problems (Andershed *et al*. 2002).

A number of twin studies have examined the importance of genetic, shared environmental and non-shared environmental factors for psychopathic personality traits and for antisocial behavior. These studies overall suggest that psychopathic personality is highly heritable, and that shared environmental factors are of subordinate importance (Taylor *et al*. 2003; Viding *et al*. 2005; Larsson *et al*. 2006). For the development of antisocial behavior, on the other hand, both genetic and shared environmental factors seem to be of importance (Rhee & Waldman, 2002; Jacobson *et al*. 2002; Tuvblad *et al*. 2006).

To the best of our knowledge, there is only one previous twin study that has examined the genetic and environmental overlap between psychopathic personality traits and antisocial behavior in adolescence, conducted by Blonigen and colleagues (2005). This study used a sample of 17-year-old twins, and a self-report normal range personality measure to index the interpersonal-affective (called Fearless Dominance) and impulsive-antisocial dimensions (called Impulsive Antisociality) of psychopathy. The results suggest a considerable genetic overlap between the impulsive-antisocial dimension and externalizing behavior (antisocial behavior and substance use). There was also a considerable genetic overlap between the interpersonal-affective dimension and externalizing behavior, but in males only (Blonigen *et al*. 2005). Because the study only included same-sex twins, it was not possible to fully explore the etiology of the observed sex-differences.

To further examine the genetic and environmental overlap between psychopathic personality traits and antisocial behavior, we used a population-based sample of adolescent twins. Opposite-sex twins were included to test for qualitative and quantitative sex differences in the genetic and environmental effects. We used a self-report measure, the Youth Psychopathic traits Inventory (YPI; Andershed *et al*. 2002) specifically designed to measure the core psychopathic personality traits among adolescents, with a replicated three-factor structure that includes the grandiose-manipulative, the callous-unemotional and the impulsive-irresponsible dimension suggested for adult psychopathy (Cooke & Michie, 2001). Multivariate twin methods were used to investigate how genetic and environmental factors contributed to the associations between the three psychopathic personality dimensions and antisocial behavior.

MATERIALS AND METHODS

Participants were part of the longitudinal Twin study of Child and Adolescent Development (TCHAD) investigating the development of health and behavior from childhood to adolescence (Lichtenstein *et al*. in press). The TCHAD contains all 1480 twin pairs born in Sweden between 1985 and 1986. Participants were ascertained from the population-based Swedish Twin Registry, which contains information on all twins born in Sweden since 1886 (Lichtenstein *et al*. 2002). The twins and/or their

parents have been contacted at three different occasions: wave 1 in 1994, wave 2 in 1999 and wave 3 in 2002, via mailed questionnaires and/or telephone interviews. Zygosity for same-sex twins was determined using discriminant analysis of twins' and parents' responses to DNA validated questionnaire items concerning the twins' physical similarity and the frequency with which people confuse them (Lichtenstein *et al.* in press).

This study used self-report questionnaire data from waves 2 and 3. At the second time point, 2261 (78%) of the children responded, while 2368 (82%) responded at the third time point. In the present study 2387 twins were included, of which there were 1912 twins with complete information from both wave 2 and wave 3: 362 MZ (monozygotic) male twins, 262 DZ (dizygotic) male twins, 404 MZ female twins, 288 DZ female twins, 298 male opposite-sex twins, and 298 female opposite-sex twins.

Selective attrition may bias estimates in longitudinal analyses (Heath *et al.* 1998). Previously we have shown that there are no significant differences in sex ratio, externalizing behavior or ADHD symptoms between responders and subjects lost to follow-up at wave 2 (Lichtenstein *et al.* in press). We have also tested whether subjects lost to follow-up at wave 3 differed from responders, on baseline measures at wave 2. Multivariate logistic regression analyses showed non-significant odds ratios (OR) for sex [OR 0·69, 95% confidence interval (CI) 0·48–1·00], family socio-economic status (OR 0·42, 95% CI 0·11–1·58), and antisocial behavior (OR 1·22, 95% CI 0·77–1·93).

Measures

Psychopathic personality

The YPI (Andershed *et al.* 2002) is a validated 50-item youth self-report questionnaire designed to measure the core traits of the psychopathic personality constellation (Skeem & Cauffman, 2003). The YPI questionnaire was completed by the twins at age 16–17 (wave 3). The YPI measures each psychopathic trait with five items making up ten internally consistent subscales. Each subscale ranged from 0 to 15 (Andershed *et al.* 2002). These subscales have been shown to form a theoretically meaningful factor structure, corresponding to the three dimensions of psychopathy suggested by Cooke & Michie (2001); (1) a *grandiose/manipulative* dimension including the subscales: dishonest charm, grandiosity, lying, and manipulation; (2) a *callous/unemotional* dimension including the subscales: callousness, unemotionality, and remorselessness; (3) an *impulsive/irresponsible* dimension including the subscales: impulsiveness, thrill-seeking, and irresponsibility. We have previously tested the fit of the three-factor model in the present data, using Confirmatory Factor Analysis. The results suggest that the three-factor model fitted significantly better than the one-factor model and the two-factor model (Larsson *et al.* 2006). The three scales were skewed (skewness: grandiose/manipulative dimension, 0·88; callous/unemotional dimension, 0·91; impulsive/irresponsible dimension, 0·43), they were therefore independently transformed [$\log_{10}(x+1)$] prior to analyses to increase normality of their distributions (skewness: grandiose/manipulative dimension, 0·39; callous/unemotional dimension, 0·36; impulsive/irresponsible dimension, −0·09).

Antisocial behavior

Antisocial behavior at ages 13–14 (wave 2) and 16–17 (wave 3) was measured using a self-report delinquency questionnaire of 34 and 32 items (revised version at wave 3), scores ranged from 0 to 160, and 0 to 170 respectively. The questionnaire served as an indicator of the frequency with which the adolescents had participated in illegal acts in the past 12 months. Items included were, for example, vandalism, breaking and entering, shoplifting, using and selling various types of illicit drugs, assault, fighting, robbery and arson (Tuvblad *et al.* 2005, 2006). The items used were initially derived from a validated instrument used in a project comparing self-reports of delinquency in 13 countries (Junger-Tas *et al.* 1994). It has long been recognized that there is considerable versatility in delinquency. Most offenders commit a wide range of offences; marked specialization is the exception rather than the rule (Klein, 1995; Rutter *et al.* 1998). The most frequently committed acts were theft-related acts and vandalism (shoplifting, theft from school/home, cheating someone for money, destroying public or private property), followed by violent acts (fighting, arson). Least common were drug-related acts. Factor analyses

of the self-reported delinquency items resulted in a single factor with a high internal consistency at both time points (wave 2: Cronbach's alpha, $\alpha = 0{\cdot}87$; wave 3, $\alpha = 0{\cdot}92$). Consequently, we analyzed it as a single composite scale at each wave. The two scales were skewed (skewness: 13–14 years, 4·01; 16–17 years, 5·10), they were therefore independently transformed [$\log_{10}(x+1)$] prior to analyses to increase normality of their distributions (skewness: 13–14 years, 1·03; 16–17 years, 0·78).

To avoid item overlap between the measure of antisocial behavior and psychopathic personality one item (truancy) in the antisocial behavior scale was excluded.

Statistical analyses

The twin method is a natural experiment that relies on the different levels of genetic relatedness between MZ and DZ twin pairs to estimate the contribution of genetic and environmental factors to individual differences in a phenotype of interest. Monozygotic twins are genetically identical, whereas dizygotic twins share on average 50% of their segregating genes. In the basic twin model, total phenotypic variance of a measured trait can be divided into additive genetic factors (A), shared environmental factors (C), and non-shared environmental factors (E). Shared environmental factors refer to non-genetic influences that contribute to similarity within pairs of twins, that is, experiences that twins have in common such as shared familial influences. Non-shared environmental factors are those experiences that make siblings dissimilar (Neale & Cardon, 1992).

We used Mx (Neale, 1997) a structural-equation modeling program to perform the model-fitting analyses by the method of raw maximum-likelihood estimation. This method allows the inclusion of singletons, where information from only one twin in a pair is available, and pairs with data from just one time point, which increases power in the analyses.

Goodness-of-fit of models was assessed by a likelihood-ratio χ^2 test, which is the differences between -2 log likelihood (-2 LL) of the full model from that of the restricted model. This difference is distributed as a χ^2. The degrees of freedom (df) for this test are equal to the difference between the number of estimated parameters in the full model and that in a restricted model. In addition to the likelihood-ratio χ^2 test, Akaike's Information Criterion (AIC $= \chi^2 - 2 \times$ degrees of freedom) was computed. A lower AIC value indicates better fit of the model to the observed data.

We fitted a series of sex-limitation models to test for qualitative sex differences, quantitative sex differences and phenotypic variance differences between the sexes. Significant qualitative sex differences, indicated by genetic correlations between the opposite-sex twins of $<0{\cdot}5$, suggest that different genes are influencing phenotypic variation in the sexes. Opposite-sex twins may also have fewer shared environmental experiences than same-sex twins, indicated by shared environmental correlations between opposite-sex twins of <1. However, it is not possible to estimate specific opposite-sex genetic correlations and shared environmental correlations simultaneously. Quantitative sex differences refer to differences between boys and girls in the magnitude of additive genetic, shared environmental and non-shared environmental influences on the measured phenotypes. The significance of such quantitative sex differences can be tested by allowing the magnitude of the parameter estimates (i.e. ACE) to differ between boys and girls. Potential phenotypic variance differences between the sexes can be tested using a scalar model. Such a model allow the phenotypic variances to differ between boys and girls, whereas the genetic and environmental parameter estimates are equated across sexes and the genetic correlations for opposite-sex twins are constrained to be equal to the genetic correlation for same-sex twins.

In order to investigate the nature of the relationship between psychopathic personality and antisocial behavior, we tested four main types of multivariate models: a Cholesky model, an independent pathway model, a one-factor common pathway model, and a two-factor common pathway model.

The Cholesky model decomposes the individual variance of each phenotype, as well as the covariances between phenotypes into genetic (A), shared (C) and non-shared (C) environmental factors. Because the Cholesky model is fully parameterized, it yields the best possible fit of the data. The Cholesky model can be used to estimate genetic correlations. This statistic varies from $+1{\cdot}0$ to $-1{\cdot}0$ and indicates the

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Table 1. *Prevalence of antisocial behavior, number of respondents (n), means and standard deviations (S.D.) for the three psychopathic personality dimensions and antisocial behavior, by sex*

	Boys		Girls		Boys			Girls		
Measures	Prevalence[a] ⩾1 offence	Prevalence[b] ⩾5 offences	Prevalence[a] ⩾1 offence	Prevalence[b] ⩾5 offences	*n*	Mean	S.D.	*n*	Mean	S.D.
Grandiose/manipulative (age 16–17)					1065	0·87	0·10	1140	0·84	0·10
Callous/unemotional (age 16–17)					1065	0·82	0·07	1136	0·75	0·07
Impulsive/irresponsible (age 16–17)					1064	0·85	0·09	1146	0·84	0·09
Antisocial behavior (age 13–14)	55%	14%	46%	7%	1044	0·37	0·41	1081	0·27	0·35
Antisocial behavior (age 16–17)	66%	21%	58%	10%	1063	0·49	0·46	1149	0·35	0·37

All scales are log-transformed.
[a] Prevalence of having committed at least one offence.
[b] Prevalence of having committed five or more different offences.

extent to which genetic influences in one measure overlap with those on a second measure.

The independent pathway model tests whether the observed covariance between the phenotypes can be adequately explained by a common genetic factor (A), a common shared environmental factor (C) and a common non-shared environmental factor (E). In addition to the common factors, the model includes specific genetic and environmental factors (A's, C's, and E's) that are unique to each measure.

The one-factor common-pathway model includes common genetic and environmental effects (A, C, and E) that load onto one latent variable that in turn loads onto all the phenotypes in the model. Like the independent pathway model, the model also includes specific genetic and environmental factors (A's, C's, E's) that are unique to each measure. This model is easily extended to include multiple intermediate latent variables, such as a two-factor common pathway model (McArdle & Goldsmith, 1990).

We also tested a model that combines elements from the Cholesky model and the independent pathway model. Specifically, this model includes one common genetic factor (A) and one common shared environmental factor (C), in addition to the specific genetic and environmental components (A's, C's). The non-shared environmental factors (E), on the other hand, were Cholesky decomposed.

RESULTS

The prevalence of antisocial behavior, number of respondents, means and standard deviations for antisocial behavior and psychopathic personality dimensions by sex are presented in Table 1. There were significant increases in prevalence from age 13–14 to age 16–17 for both sexes (McNemar test: boys, $p<0{\cdot}001$; girls, $p<0{\cdot}02$). Having committed at least five different offences at both time points was reported by 7·9% of boys, and 2·4% of girls. Mean scores were also consistently higher in boys compared to girls for all measures except for impulsive/irresponsible [grandiose/manipulative: $t=6{\cdot}87$, df = 2, 203, $p<0{\cdot}001$; callous/unemotional: $t=20{\cdot}77$, df = 2, 199, $p<0{\cdot}001$; impulsive/irresponsible: $t=2{\cdot}58$, df = 2, 208, $p<0{\cdot}001$; antisocial behavior (age 13–14): $t=6{\cdot}33$, df = 2, 123, $p<0{\cdot}001$; antisocial behavior (age 16–17): $t=7{\cdot}55$, df = 2, 210, $p<0{\cdot}001$]. Therefore, in our model mean values were allowed to differ between boys and girls. Mean scores for antisocial behavior increased from age 13–14 to age 16–17 for both boys and girls (boys: paired t test, $-8{\cdot}80$, df = 939, $p<0{\cdot}001$; girls paired t test, $-9{\cdot}10$, df = 1, 018, $p<0{\cdot}001$).

There was a substantial interrelationships among the three psychopathic personality dimensions, ranging from $r=0{\cdot}45$–$0{\cdot}63$ for boys, and from $r=0{\cdot}38$–$0{\cdot}59$ for girls. There was a relatively high stability in antisocial behavior

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Table 2. *Intra-class correlations and cross-twin cross-trait correlations for the three psychopathic personality dimensions and antisocial behavior, by zygosity and sex. Boys below the diagonal and girls above the diagonal in grey shade*

Boys/Girls	Grandiose/ manipulative (age 16–17)	Callous/ unemotional (age 16–17)	Impulsive/ irresponsible (age 16–17)	Antisocial behavior (age 13–14)	Antisocial behavior (age 16–17)
Monozygotic (MZ)					
Grandiose/manipulative (age 16–17)	**0·46**[a]/	**0·59**[a]	**0·35**[b]	**0·42**[b]	**0·21**[d]	**0·36**[d]
Callous/unemotional (age 16–17)	**0·24**[b]	**0·46**[a]/	**0·45**[a]	**0·30**[b]	**0·15**[d]	**0·28**[d]
Impulsive/irresponsible (age 16–17)	**0·38**[b]	**0·27**[b]	**0·56**[a]/	**0·55**[a]	**0·33**[d]	**0·47**[d]
Antisocial behavior (age 13–14)	**0·29**[d]	**0·17**[d]	**0·43**[d]	**0·68**[a]/	**0·69**[a]	**0·49**[c]
Antisocial behavior (age 16–17)	**0·31**[d]	**0·19**[d]	**0·45**[d]	**0·54**[c]	**0·56**[a]/	**0·69**[a]
Dizygotic (DZ)					
Grandiose/manipulative (age 16–17)	**0·25**[a]/	**0·26**[a]	**0·16**[b]	**0·18**[b]	0·07[d]	**0·14**[d]
Callous/unemotional (age 16–17)	**0·15**[b]	**0·27**[a]/	**0·27**[a]	**0·19**[b]	−0·01[d]	**0·11**[d]
Impulsive/irresponsible (age 16–17)	**0·13**[b]	**0·20**[b]	**0·15**[a]/	**0·29**[a]	**0·17**[d]	**0·22**[d]
Antisocial behavior (age 13–14)	**0·16**[d]	0·10[d]	**0·19**[d]	**0·51**[a]/	**0·43**[a]	**0·25**[c]
Antisocial behavior (age 16–17)	**0·15**[d]	**0·20**[d]	**0·22**[d]	**0·27**[c]	**0·47**[a]/	**0·45**[a]
Opposite-sexed twins					
Grandiose/manipulative (age 16–17)	**0·27**[a]/	**0·27**[a]	0·10[b]	**0·14**[b]	**0·14**[d]	**0·12**[d]
Callous/unemotional (age 16–17)	**0·19**[b]	**0·14**[a]/	**0·14**[a]	**0·12**[b]	**0·13**[d]	**0·11**[d]
Impulsive/irresponsible (age 16–17)	**0·15**[b]	**0·14**[b]	**0·22**[a]/	**0·22**[a]	**0·25**[a]	**0·18**[c]
Antisocial behavior (age 13–14)	−0·01[d]	−0·03[d]	**0·15**[d]	**0·46**[a]/	**0·48**[a]	**0·19**[c]
Antisocial behavior (age 16–17)	0·10[d]	**0·11**[d]	**0·13**[d]	**0·31**[c]	**0·26**[a]/	**0·26**[a]

Significant correlations are in bold.
[a] Intra-class correlations.
[b] Cross-twin cross-trait correlations within the psychopathic personality constellation.
[c] Cross-twin cross-trait correlations within antisocial behavior across time.
[d] Cross-twin cross-trait correlations between the psychopathic personality constellation and antisocial behaviour.

from age 13–14 to age 16–17 (boys: $r = 0{\cdot}52$; girls: $r = 0{\cdot}48$). The associations between psychopathic personality dimensions and antisocial behavior ranged from $r = 0{\cdot}20$–$0{\cdot}56$ for boys, and from $r = 0{\cdot}12$–$0{\cdot}51$ for girls (data not shown, available from first author).

Table 2 contains the intra-class correlations and cross-twin cross-trait correlation matrices for the psychopathic personality dimensions and antisocial behavior, by zygosity and sex. The diagonal elements contain the intra-class correlations for boys and girls (correlations indicated by superscript a). The difference between the MZ and DZ intra-class correlations can be used for evaluating the relative contribution of genetic and environmental influences. The consistently higher MZ as compared to DZ intra-class correlations shown in Table 2 suggest genetic influences for all measures. For example, the intra-class correlations for the grandiose/manipulative dimension were 0·46 for MZ boys and 0·59 for MZ girls. The corresponding numbers for DZ twins were substantially lower, 0·25 and 0·26.

The off-diagonal elements in Table 2 contain the cross-twin cross-trait correlations. The cross-twin cross-trait correlations can be used to evaluate genetic and environmental influences

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Table 3. *Model fitting results of multivariate analysis of the three psychopathic personality dimensions and antisocial behavior*

Model	Fit of model compared to saturated model							Difference in fit of models		
	−2 LL	df	χ^2	df	*p*	AIC	Compared to model	$\Delta\chi^2$	Δdf	*p*
Saturated model	43559·84	10556	—	—	—	—	—	—	—	—
1. ACE Cholesky										
(a) Sex difference r_a = free, r_c = 1	43800·51	10776	240·67	220	0·16	−199·32	—	—	—	—
(b) Sex difference r_a = 0·5, r_c = free	43801·84	10776	242·01	220	0·147	−197·99	—	—	—	—
(c) Sex difference r_a = 0·5, r_c = 1	**43801·84**	**10781**	**242·01**	**225**	**0·21**	**−207·99**	**1a**	**1·33**	**5**	**0·93**
(d) Scalar r_a = 0·5, r_c = 1	43880·04	10821	320·20	265	<0·05	−209·80	1c	78·20	40	<0·01
2. ACE independent pathway										
(c) Sex difference r_a = 0·5, r_c = 1	43865·99	10811	306·15	255	0·015	−203·85	1c	64·14	30	<0·01
3. AC[a] independent pathway										
(c) Sex difference r_a = 0·5, r_c = 1	**43821·40**	**10801**	**261·56**	**245**	**0·22**	**−228·44**	**1c**	**19·55**	**20**	**0·49**
4. Two-factor common pathway										
(c) Sex difference r_a = 0·5, r_c = 1	43972·40	10817	412·57	261	<0·01	−109·43	1c	170·56	36	<0·01
5. One-factor common pathway										
(c) Sex difference r_a = 0·5, r_c = 1	44213·35	10827	653·51	271	<0·01	111·51	1c	411·5	46	<0·01

Best fitting model indicated in bold.
−2 LL, −2 log likelihood; AIC, Akaike's information criteria; A, additive genetic variance; C, shared environmental variance; E, non-shared environmental variance; r_a, genetic correlation between opposite-sex pairs; r_c, shared environmental correlation between opposite-sex pairs; Sex differences, quantitative sex differences.
[a] E, Cholesky decomposed.

within and between the psychopathic personality dimensions and antisocial behavior. For example, the correlation between the callous/unemotional dimension for one twin and the grandiose/manipulative dimension for his co-twin was 0·24 for MZ boys; for MZ girls this correlation was 0·35. The corresponding cross-twin cross-trait correlations for DZ twins were 0·15 and 0·16. The cross-twin cross-trait correlations were consistently higher in MZ twins compared to DZ twins, suggesting that genetic effects contribute to the covariation among the three psychopathic personality dimensions (correlations indicated by superscript b), between antisocial behavior at age 13–14 and at age 16–17 (correlations indicated by superscript c), and between psychopathic personality dimensions and antisocial behavior (correlations indicated by superscript d).

Model fitting

We fitted four sex-limitation models (Table 3: Models 1a–1d). Model 1c provided the best fit of the data (−2 LL = 43801·84, df = 10781, AIC = −207·99), suggesting that the magnitude of the genetic and environmental effects are different in boys and girls. That is, our results indicate quantitative sex differences rather that qualitative sex differences. The parameter estimates from the best-fitting sex-limitation model was used to calculate the genetic correlations. Genetic correlations for the three psychopathic personality dimensions have already been published (Larsson *et al*. 2006). In short, the results showed genetic correlations between the grandiose/manipulative, the callous/unemotional and the impulsive/irresponsible dimension ranging from 0·59 to 0·78, with the greatest genetic association between the grandiose/manipulative and the impulsive/irresponsible dimension. Table 4 presents genetic correlations between the three psychopathic personality dimensions at age 16–17 and antisocial behavior at ages 13–14 and 16–17. As can be seen, most genetic correlations were significant. The genetic correlations ranged from 0·24 to 0·84, with higher genetic

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Table 4. *Genetic correlations between the three psychopathic personality dimensions and antisocial behavior*

	Antisocial behavior (age 13–14)		Antisocial behavior age (16–17)	
	Boys r_a (95% CI)	Girls r_a (95% CI)	Boys r_a (95% CI)	Girls r_a (95% CI)
Grandiose/manipulative	0·45 (−0·01 to 0·89)	**0·46 (0·21 to 0·78)**	**0·68 (0·35 to 0·93)**	**0·64 (0·49 to 0·87)**
Callous/unemotional	0·24 (−0·33 to 0·72)	**0·38 (0·02 to 0·95)**	0·37 (−0·09 to 0·71)	**0·64 (0·39 to 1·0)**
Impulsive/irresponsible	**0·66 (0·31 to 1·0)**	**0·65 (0·40 to 0·98)**	**0·84 (0·60 to 1·0)**	**0·77 (0·59 to 1·0)**

Significant correlations are in bold.
r_a, Genetic correlations; CI, confidence interval.

associations between the impulsive/irresponsible dimension and antisocial behavior and somewhat lower genetic associations between the callous/unemotional dimension and antisocial behavior.

To further investigate the nature of the relationship between psychopathic personality and antisocial behavior, we fitted a series of multivariate models (Models 2c–5c in Table 3). Specifically, we compared the fit of the best-fitting Cholesky sex-limitation model (Model 1c in Table 3) to an independent pathway model (2c), a model that combine elements from the Cholesky decomposition and the independent pathway model (3c), a one-factor common pathway model (4c) and a two-factor common pathway model (5c). The model that combined elements from the Cholesky decomposition and the independent pathway model (Model 3c) provided the best fit of the data (-2 LL = 43821·40, df = 10801, AIC = $-228·44$). That is, Model 3c provided a more parsimonious representation of the data compared to Model 1c ($\Delta -2$ LL = 19·55, df = 20, $p = 0·49$).

Fig. 1(*a*, *b*) displays estimates for the common (A) and unique (A's) genetic effects as well as for the common (C) and unique (C's) shared environmental effects from the best-fitting model (Model 3c) for boys and girls respectively.

First, the genetic and shared environmental loadings on the three psychopathic personality dimensions were similar for boys and girls. In fact, constraining the 12 loadings ($A_3 + A's_3 + C_3 + C's_3 = 12$) onto the grandiose/manipulative, callous/unemotional and impulsive/irresponsible dimensions to be equal for boys and girls resulted in a non-significant decrease in fit ($\Delta -2$ LL = 15·38, df = 12, $p = 0·22$). However, constraining the corresponding genetic and shared environmental loadings ($A_2 + A's_2 + C_2 + C's_2 = 8$) onto antisocial behavior at ages 13–14 and 16–17 resulted in a significant decrease in fit ($\Delta -2$ LL = 33·18, df = 8, $p < 0·01$). These *post-hoc* analyses suggest, in agreement with previous studies using the same sample, that there are quantitative sex differences in adolescent antisocial behavior (Tuvblad *et al.* 2005, 2006), but not in the three psychopathic personality dimensions (Larsson *et al.* 2006).

Second, summing the genetic paths that load on each measure gives the heritability. The heritability was moderate for the three psychopathic personality dimensions: 33–53% of the variance was explained by genetic effects. The heritability for antisocial behavior was at both time points higher in girls (41%, 62%), than in boys (19%, 31%).

Third, the common additive genetic factor (A) loaded most highly on the impulsive/irresponsible dimension and antisocial behavior at age 16–17, but also significantly on the other measures. Specifically, the common genetic factor explained 20–45% of the variance in the three psychopathic personality dimensions and 19–41% of the variance in antisocial behavior at ages 13–14 and 16–17.

Fourth, the common shared environmental factor (C) loaded significantly on antisocial behavior measures; explaining 14–41% and 4–27% of the variance for boys and girls respectively. However, the common shared environmental contribution to the three

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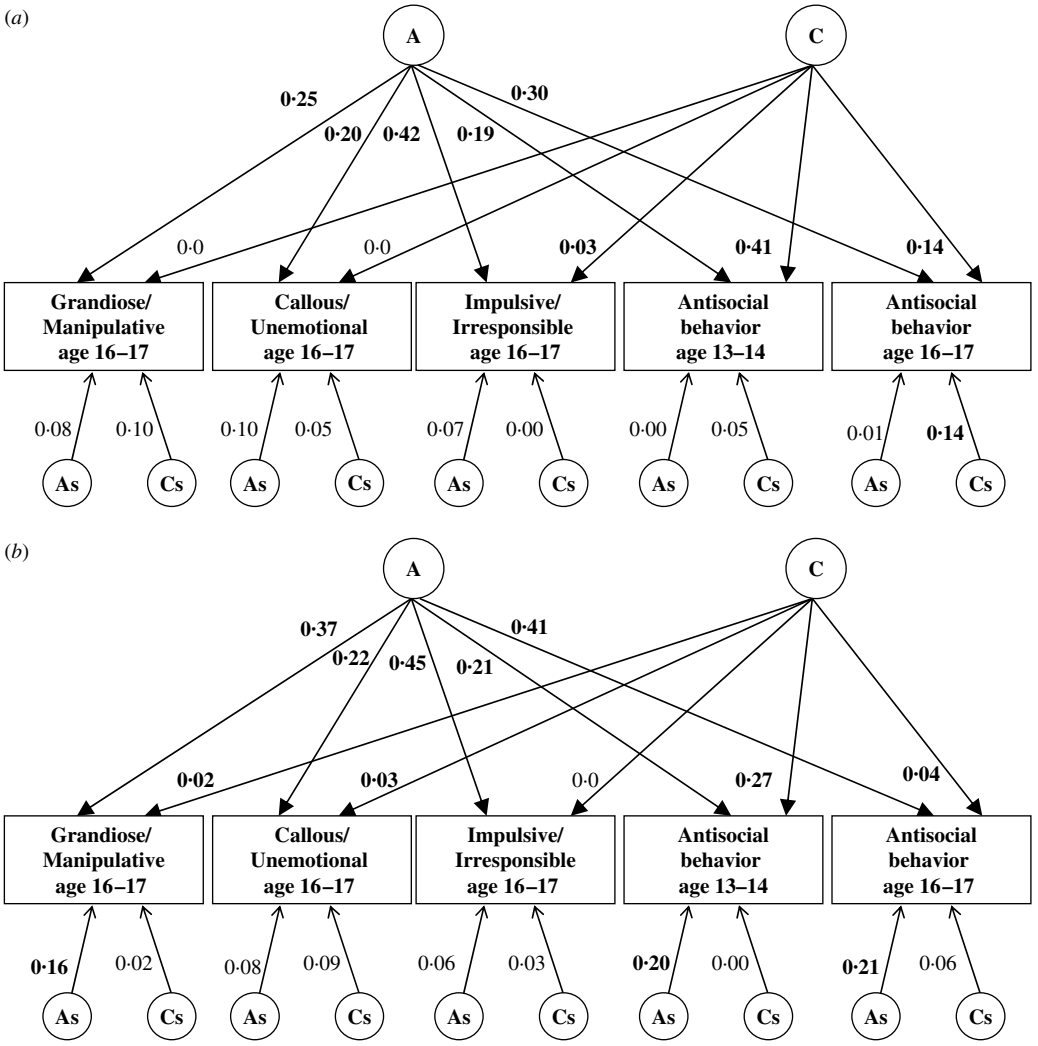

FIG. 1. Standardized squared path estimates from the independent pathway model for *psychopathic personality traits* and *antisocial behavior* in boys (*a*) and girls (*b*) at ages 13–14 and 16–17. The latent variables A (additive genetic factor) and C (shared environmental factor) are depicted in circles. Measured variables are depicted in rectangles (i.e. grandiose/manipulative age 16–17; antisocial behavior age 16–17). As (additive genetic): is residual variance specific to each measure and likewise for Cs (shared environment). The non-shared environmental factors were Cholesky decomposed and are not depicted in the figure. Significant estimates are in bold.

psychopathic personality dimensions were of limited importance.

DISCUSSION

This study found that a common genetic factor contributed substantially to the three psychopathic personality dimensions and to antisocial behavior measured at two time points. Further, a common shared environmental factor was found to influence antisocial behavior, but did not influence psychopathic personality dimensions. Measurement-specific effects were generally only modest.

Our results strongly suggest that genetic factors largely account for the pattern of covariation between the psychopathic personality dimensions and antisocial behavior. This finding is in line with the recent study by Blonigen and colleagues (2005) who reported a genetic

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overlap between self-reported psychopathic personality traits (Fearless Dominance and Impulsive Antisociality) and externalizing psychopathology. This may suggest a common set of genes that influence not only psychopathic-like personality traits and antisocial behavior, but also other disorders, reflecting a genetic vulnerability to externalizing psychopathology (Young *et al*. 2000; Krueger *et al*. 2002; Kendler *et al*. 2003). However, there are other feasible competing explanations to the observed finding. For example, our findings are congruent with genetic influences that act indirectly on antisocial behavior, via psychopathic-like personality traits (Goldsmith & Gottesman, 1996). Personality traits, or temperamental characteristics that antecede psychopathic-like personality traits in adolescents, might therefore be interesting targets for molecular genetic research.

The finding that the shared environment produced a negligible contribution to the three psychopathic personality dimensions is consistent with evidence previously reported across behavioral genetic studies of psychopathic-like personality traits (Taylor *et al*. 2003; Blonigen *et al*. 2005; Larsson *et al*. 2006), as well as other personality dimensions (Bouchard & Loehlin, 2001). In sharp contrast, we found that a common shared environmental factor was important for antisocial behavior measured at two time points. This finding is consistent with a study by Viding *et al*. (2005) who reported that antisocial behavior in children without elevated levels of callous-unemotional traits showed only modest genetic influence but a moderate influence from the shared environment.

Our finding of shared environmental effects in antisocial behavior suggests an etiological distinction between psychopathic-like personality traits and antisocial behavior. It is tempting to speculate that these results may be a manifestation of the distinction between basic tendencies and characteristic adaptations, the former being basic core personality traits and the latter being overt manifestations that have developed as a product of the interplay between the basic tendencies and environmental influences, as outlined by McCrae & Costa (1995). In other words, psychopathic-like personality traits might be more of a basic tendency, whereas antisocial behavior could be viewed as characteristic adaptations (Cooke & Michie, 2001).

The well-known sex difference with boys displaying more antisocial behavior symptoms was observed at both age 13–14 and age 16–17. In line with previous results using the current sample (Tuvblad *et al*. 2005, 2006; Larsson *et al*. 2006), we found that the heritability of antisocial behavior at age 13–14 and age 16–17 was higher for girls than for boys, while no sex differences were found for the three psychopathic personality dimensions. The higher heritability in girls was largely due to age-specific genetic effects that influence antisocial behavior independent of psychopathic personality traits, potentially reflecting genes that are activated at puberty (Jacobson *et al*. 2002), especially at age 13–14. Future multivariate twin studies may benefit from adding other potential key variables that could explain the age and sex-specific genetic effects in the development of antisocial behavior.

Limitations

We also have to consider the limitations of this study and how these might have influenced our findings. First, the psychopathic personality dimensions and antisocial behavior were assessed using self-report questionnaires in a population-based twin sample; hence, our result is not to be extrapolated to adult clinical settings, such as prisons or forensic hospitals, that is, the settings for which the psychopathy construct has been validated (Hare, 2003). It should however, be noted that population-based samples, as opposed to clinical-referred samples, do not introduce referral and selection biases. Also, since there were no overlaps in the items that were used to define the psychopathic personality dimensions and antisocial behavior, the association is not attributable to measurement confounding.

Second, it is generally assumed in twin models that random mating occurs in the parent generation. Assortive mating tends to increase similarity between DZ twins, thereby biasing the heritability estimates downward and the shared environmental estimates upward. Assortive mating for most personality traits has been found to be low in magnitude (Maes *et al*. 1998), suggesting that the effects of positive assortment do not have to be considered when modeling the variance in psychopathic personality dimensions. However, at least on study suggests that

this assumption is invalid in the case of antisocial behavior (Krueger *et al*. 1998). This might suggest that part of the shared environment seen in antisocial behavior may be due to positive assortment.

A third limitation concerns the equal-environment assumption. If MZ twins are exposed to more *similar* environment than DZ twins, this will result in excess similarity for MZ compared to DZ twins. Although the equal-environment assumption is critical to the validity of twin studies, we did not directly test it herein. However, studies that have tested the equal-environment assumption have found it to be valid for anxiety disorder, attention deficit hyperactivity disorder, oppositional defiant disorder, and conduct disorder (Cronk *et al*. 2002).

Finally, like in other twin studies, we have not modeled for the effects of gene–environment interaction; the presence of which has been suggested by other authors (Caspi *et al*. 2002).

Despite these limitations, this study provides evidence of a considerable genetic overlap between psychopathic personality dimensions and antisocial behavior. Future studies should seek to identify early indicators of this personality construct such as temperamental and neuropsychological components. Knowledge from such studies may not only facilitate identification of susceptibility genes, but also provide tools needed to identify at-risk children early in life.

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DECLARATION OF INTEREST

None.

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