Journal of Abnormal Psychology
2006, Vol. 115, No. 2, 221–230

0021-843X/06/$12.00 DOI: 10.1037/0021-843X.115.2.221

A Genetic Factor Explains Most of the Variation in the Psychopathic Personality

Henrik Larsson
Karolinska Institutet

Henrik Andershed
Örebro University

Paul Lichtenstein
Karolinska Institutet

The psychopathic personality can be conceptualized as three interrelated dimensions, (a) an interpersonal style of glibness, grandiosity, and manipulation; (b) an affective disposition of callousness, lack of empathy, and unemotionality; and (c) a behavioral/lifestyle dimension of impulsivity, need for stimulation, and irresponsibility, underpinning a higher order construct, psychopathic personality. The authors used a self-report questionnaire (The Youth Psychopathic Traits Inventory) to study the importance of genetic and environmental influences on psychopathic personality traits in a sample of 1,090 monozygotic and dizygotic twin pairs, aged 16–17 years. Results showed a strong genetic influence behind the higher order "psychopathic personality" factor, underpinned by the three psychopathic personality dimensions. Over and above the effects to the higher order factor, significant unique genetic influences were also found in the callous/unemotional and in the impulsive/irresponsible dimension, but not in the grandiose/manipulative dimension. The authors propose that this latent psychopathic personality factor is a meaningful target for future etiological research.

Keywords: psychopathy, genetic, environment, twins, adolescence

In its adult manifestation the psychopathic personality constellation is referred to as psychopathy and is considered a serious personality disorder that has been linked to a particularly severe and violent pattern of antisocial behavior (Hare, 2002, 2003). Although relatively much is known about what characterizes individuals with psychopathy in adulthood, little empirical work has been done on its etiology. In recent years there has been a growing interest in the study of developmental aspects of this personality disorder (e.g., Frick, Bodin, & Barry, 2000; Lynam, 1996), which seems necessary to identify the individuals with fledgling psychopathy as early as possible and to understand the causes of this socially devastating personality profile.

Psychopathy is characterized in the adult literature by at least three main dimensions: an arrogant, grandiose, and deceitful interpersonal style, a deficient affective experience, and an impulsive behavioral style (Cleckley, 1976; Hare, 1991). In contemporary research and clinical practice, psychopathy is most commonly assessed with the Hare Psychopathy Checklist (the full Psychopathy Checklist–Revised [PCL–R], Hare, 1991, 2003; or the shorter screening version, Hart, Cox, & Hare, 1995), and a substantial amount of literature indicates that psychopathy is a reliable, valid, and meaningful construct in adults.

Given that psychopathy is defined as a personality disorder, it is assumed that psychopathic traits are manifested at an early age (Forth & Burke, 1998; Frick, 2002; Lynam, 2002a). Critics have argued that this assumption must be tested in longitudinal research, given that several features of adult psychopathy are normative and temporary characteristics of adolescence (Edens, Skeem, Cruise, & Cauffman, 2001; Seagrave & Grisso, 2002). Although there is concern, several studies using both the PCL–R (Hare, 1991) and the Psychopathy Checklist–Revised: Youth Version (PCL:YV; Forth, Kosson, & Hare, 2003) have shown that adolescent criminal offenders with pronounced psychopathic traits differ from other antisocial youth in terms of more violent acts committed and the seriousness of their offenses as well as violence while institutionalized (Brandt, Kennedy, Patrick, & Curtin, 1997; Corrado, Vincent, Hart, & Cohen, 2003; Forth & Burke, 1998; Kosson, Cyterski, Steuerwald, Neumann, & Walker-Matthews, 2002; Skeem & Cauffman, 2003; Vincent, Vitacco, Grisso, & Corrado, 2003). Furthermore, studies using a screening measure of psychopathic traits, the Antisocial Process Screening Device (APSD; Frick & Hare, 2001), have shown that the presence of psychopathic-like features (callous/unemotional traits) identifies a subgroup of antisocial youths with a more severe and aggressive pattern of conduct problems than other youths with conduct problems (Caputo, Frick, & Brodsky, 1999; Christian, Frick, Hill, Tyler, & Frazer, 1997; Enebrink, Andershed, & Långström, 2005; Frick et al., 2000).

A related line of research suggests that psychopathy-like features can be meaningfully assessed among nonreferred youths and

Henrik Larsson and Paul Lichtenstein, Department of Medical Epidemiology and Biostatistics, Karolinska Institutet; Henrik Andershed, Department of Behavioral, Social, and Legal Sciences, Örebro University.

This study was funded by the Swedish Council for Working Life and Social Research (Project 2001-2368) and the Swedish Research Council (2001-4231).

Correspondence concerning this article should be addressed to Henrik Larsson, MA, Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, P.O. Box 281, SE-171 77, Stockholm, Sweden. E-mail: henrik.larsson@meb.ki.se

that this kind of research can be important and helpful for identifying risks and protective factors in the development of psychopathy (e.g., Andershed, Kerr, Stattin, & Levander, 2002). Much of this research is conducted on normal populations of youth and is built on the assumption that psychopathy is a dimensional construct. The issue of whether it is correct to treat psychopathy as a dimension rather than as a taxon, has been tested through taxometric methods (Meehl, 1992) in studies focusing on psychopathy-related traits (Harris, Rice, & Quinsey, 1994; Marcus, Edens, & Lilienfeld, 2004; Skilling, Harris, Rice, & Quinsey, 2002; Skilling, Quinsey, & Craig, 2001). The results have been mixed, with some studies showing taxonicity for the behavioral, social deviance dimension of psychopathy only (Harris et al., 1994; Skilling et al., 2002, 2001) and a recent study showing no confirmation that psychopathy or any of its factors are taxonic (Marcus et al., 2004). Also, identifying the presence of a taxon through taxometrics does not preclude the possibility that the behavior pattern, belief, affect, cognition, or attitudes included within the taxon would not be better understood as maladaptive variants along an underlying dimension (Widiger, 2001). In line with this thinking, it has been proposed that psychopathy can be seen as an extreme constellation within the framework of normal personality dimensions such as the five-factor model (FFM) of personality, a proposal that has gained empirical support (e.g., Lynam, 2002a, 2002b; Miller & Lynam, 2003; Widiger, 1998). In addition, results of a recent study, using the APSD (Frick, Cornell, Barry, Bodin, & Dane, 2003), suggest that psychopathy-like features can be reliably identified in nonreferred samples of youths. Research using self-report measures of psychopathic traits has also been quite promising in a number of respects (e.g., Andershed et al., 2002; Caputo et al., 1999; Lilienfeld & Andrews, 1996; Lynam, Whiteside, & Jones, 1999). For example, recent research on 16-year-old adolescents, using a self-report instrument, the Youth Psychopathic Traits Inventory (YPI; Andershed et al., 2002), showed that psychopathic personality traits are related to conduct problem behavior (aggressive and nonaggressive) and identifies a meaningful "psychopathy-like" subgroup of antisocial adolescents (Andershed et al., 2002). Moreover, in another recent study using a sample of serious adolescent offenders, the YPI was shown to (a) predict a range of short-term institutional misbehaviors, (b) manifest good test–retest reliability, (c) be negatively related to anxiety, and (d) be associated with the PCL:YV (Skeem & Cauffman, 2003).

The YPI was designed to measure the core personality characteristics of the psychopathic personality constellation (Andershed et al., 2002) on the basis of contemporary adult models of psychopathy (e.g., Cooke & Michie, 2001; Hare, 1991). More important, the traits measured through the YPI form three distinct but correlated factors (Andershed et al., 2002) similar to those found in previous studies on adult offenders (Cooke, Kosson, & Michie, 2001; Cooke, Michie, Hart, & Clark, 2004; Johansson, Andershed, Kerr, & Levander, 2002; Skeem, Mulvey, & Grisso, 2003; Warren et al., 2003): (a) a grandiose/manipulative dimension; (b) a callous/unemotional dimension; and (c) an impulsive/irresponsible dimension. However, there is an ongoing debate about how to best describe the construct of psychopathy. Factor analysis of these traits have found two (e.g., Benning, Patrick, Hicks, Blonigen, & Krueger, 2003; Frick et al., 2000; Hare, 1991; Lilienfeld & Andrews, 1996), three (Andershed et al., 2002; Cooke & Michie, 2001; Forth et al., 2003), and four (Hare, 2003) factors underlying these traits. Despite this concern, some researchers have emphasized the intercorrelated nature of these dimensions and suggested that psychopathy reflects a broad higher order factor encompassing all of them (e.g., Newman, 1998). Thus, the notion of a superordinate factor of psychopathy has strong empirical support in both adult (Cooke & Michie, 2001; Hare, 2003) and adolescent (Forth et al., 2003) samples. That is, a hierarchical model of psychopathy, in which a higher order factor is overarched by at least two highly correlated trait dimensions, has been suggested (see Cooke & Michie, 2001).

Taken together, a growing body of research suggests that studying psychopathy-like features in youths is important as a part of the search for precursors to the adult disorder and to severe antisocial behavior. In addition, although psychopathic personality characteristics certainly are less extreme among nonreferred youths than among adult offenders, these traits seem to be present, measurable, and manifested in similar ways. Thus, in this sense, it seems reasonable that research on the psychopathic personality in normal populations and in youths can be informative about the clinical adult version of the psychopathic personality.

Etiology of Psychopathic Personality

To date, there are only two published twin studies that have directly investigated the importance of genetic and environmental influences for psychopathic traits (Blonigen, Carlson, Krueger, & Patrick, 2003; Taylor, Loney, Bobadilla, Iacono, & McGue, 2003). One of these used a sample of adult male twins (Blonigen et al., 2003) and examined the genetic and environmental influence on psychopathic personality traits by using a self-report measure, the Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996). Individual differences in all eight dimensions measured by the PPI were associated with genetic and nonshared environmental effects. Genetic effects explained 29%–56% of the variation of the respective dimensions of the PPI. Shared environmental effects were not found for any of the PPI facets (Blonigen et al., 2003). Another recent study on male adolescent twins (Taylor et al., 2003) used a self-report measure that taps the impulsive/antisocial behavior and callous/unemotional interpersonal style of the psychopathic personality constellation (Minnesota Temperament Inventory; Loney, Taylor, Butler, & Iacono, 2002). In this study, genetic effects accounted for approximately 40% of the variation in both the callous/unemotional and the impulsive/antisocial factors. Nonshared environmental effects explained all of the remaining variance, whereas the influences of shared environment seemed to be of no importance (Taylor el al., 2003). Perhaps more interestingly, results from multivariate genetic analysis indicated that there is not only a common genetic effect influencing the callous/unemotional and the impulsive/antisocial factor but also some independence in the etiology underlying these two factors (Taylor et al., 2003). That is, despite a high covariation between the callous/unemotional and the impulsive/antisocial factors, this study suggests that these dimensions can be differentiated etiologically.

Although the two twin studies offer a starting point to a better understanding of the underlying etiology for different psychopathic characteristics, it is important to note that both studies used relatively small samples and they only included male twins. Consequently, it was not possible to assess potential sex differences in

these studies. In addition, none of the studies used a measure with an underlying three-factor structure of interpersonal, affective, and behavioral/lifestyle dimensions that has been shown to describe the psychopathic personality well (e.g., Cooke & Michie, 2001; Johansson et al., 2002). Hence, there is to date no information available on the genetic and environmental influences on the phenotypic association among these three psychopathic personality dimensions. Furthermore, in neither of the previous studies was a "common latent factor" considered as an explanation for the observed phenotypic covariation.

To acquire a better understanding of how genetic and environmental effects influence the psychopathic personality constellation, we assessed a normal population-based sample of 16- to 17-year-old Swedish twins using the self-report YPI (Andershed et al., 2002). The research questions were the following: How much of the variance in the three psychopathic personality dimensions is due to genetic and environmental factors, when considered separately? Is there evidence for sex differences in genetic/environmental effects behind the three dimensions? Is there a common genetic effect influencing the three personality dimensions? and Are there also unique genetic effects for the three separate dimensions?

Method

Sample

The data in this study come from the Twin Study of Child and Adolescent Development, an ongoing prospective longitudinal study on health and behavior in children and adolescents, with data collected from parents when the twins were age 8–9 and both parents and twins at age 13–14 and 16–17 (Eley, Lichtenstein, & Moffitt, 2003; Larsson, Larsson, & Lichtenstein, 2004; Lichtenstein & Svartengren, 1997). The present study is based on data from the third wave of this project, which was conducted during Spring 2002 when the twins were 16–17 years old. All twins born in Sweden between May 1985 and December 1986, if both twins were alive and lived in Sweden at the time of the study, received a mailed questionnaire from which 2,369 (82%) responded. The final sample that was used for the analyses in the present study consisted of twins for whom there were complete data for psychopathic personality traits and for whom zygosity could be diagnosed. Thus, the number of participants in the present analyses is 2,198 twins: (monozygous [MZ] boys = 386, MZ girls = 452; dizygous [DZ] boys = 328; DZ girls = 366, and opposite sex = 666). In addition, a telephone follow-up of the twins who did not participate, using a reduced battery of YPI items, was also completed. The telephone interview sample consisted of 165 twins.

Zygosity determination for the same-sexed twin pairs was based on twin similarity from parent's response (in Waves 1, 2, and 3) and children's response (in Waves 2 and 3). Zygosity determined by these questions has been validated with DNA and has been shown to give >95% and 98% correct classifications, respectively (Lichtenstein et al., 2002). To improve the accuracy of the zygosity classification further, all five assessments of zygosity were used. If there were contradictions between child and parental-derived zygosity, the parental response had priority. In case of disagreement between parent's response in Waves 1, 2, and 3, zygosity was determined as unknown.

Measures

YPI. The YPI is a 50-item youth self-report questionnaire designed to measure the core traits of the psychopathic personality constellation (Andershed et al., 2002). The YPI uses a 4-point Likert-type response scale ranging from *does not apply at all* to *applies very well* and measures each psychopathic trait with 5 self-report items making up 10 internally consistent subscales. The 10 subscales are (with one sample item and Cronbach's alphas in the present data within parentheses) Dishonest Charm ("I have the ability to con people by using my charm and smile," $\alpha = .79$), Grandiosity ("I am more important and valuable than other people," $\alpha = .70$), Lying ("Sometimes I lie for no reason, other than because it is fun," $\alpha = .74$), Manipulation ("I can get almost anyone to believe anything," $\alpha = .77$), Callousness ("When other people have problems it is often their own fault; therefore, one should not help them," $\alpha = .58$), Unemotionality ("I don't let my feelings affect me as much as other people's feelings seem to affect them," $\alpha = .63$), Remorselessness ("I have the ability not to feel guilt and regret about things that I think other people would feel guilty about," $\alpha = .70$), Impulsiveness ("It often happens that I do things without thinking ahead," $\alpha = .68$), Thrill-Seeking ("I like to do things just for the thrill of it," $\alpha = .73$), and Irresponsibility ("It happened several times that I have borrowed something and then lost it," $\alpha = .68$).

The subscales of the YPI have been shown to form a theoretically meaningful and useful three-factor structure consisting of (a) a grandiose/manipulative dimension (including the subscales Dishonest Charm, Grandiosity, Lying, and Manipulation), (b) a callous/unemotional dimension (including the subscales Callousness, Unemotionality, and Remorselessness), and (c) an impulsive/irresponsible dimension (including the subscales Impulsiveness, Thrill-Seeking, and Irresponsibility). This three-factor structure has been supported by both exploratory and confirmatory factor analyses (CFA) (Andershed et al., 2002).

We tested the fit of the three-factor model of the 10 YPI subscales in the present data with CFA (the subscales were created by taking the mean of the five items included in each subscale). We used weighted least squares as our method of estimation with polychoric correlation matrices and its corresponding asymptotic covariance weight matrices (Jöreskog & Sörbom, 1993). The fit indices suggested an acceptable fit of this model to the data among boys, $\chi^2(32, N = 1{,}099) = 233.98$, $p < .001$, comparative fit index = .98, goodness-of-fit index = .99, nonnormed fit index = .98, root-mean-square-error of approximation = .08; and girls, $\chi^2(32, N = 1{,}226) = 282.77$, $p < .001$, comparative fit index = .98, goodness-of-fit index = .98, nonnormed fit index = .97, root-mean-square-error of approximation = .08. We also tested alternative factor structures of the YPI through CFA. We first considered the "traditional" two-factor model (Hare, 1991, 2003). To test this model, we combined the YPI subscales Dishonest Charm, Lying, Grandiosity, Manipulation, Callousness, Unemotionality, and Remorselessness into a first factor and the subscales Impulsiveness, Irresponsibility, and Thrill-Seeking into a second factor. This two-factor model had a significantly poorer fit compared with the three-factor model (as shown through a significantly lower chi-square of the three-factor model) among both boys, $\Delta\chi^2(2, N = 1{,}099) = 82.35$, $p < .001$, and girls, $\Delta\chi^2(2, N = 1{,}226) = 92.05$, $p < .001$. Finally, we considered a one-factor model of the YPI (i.e., all the 10 traits measured by the YPI in one single factor). The results showed that the three-factor model of the YPI had a significantly better fit than this model both among boys, $\Delta\chi^2(3, N = 1{,}099) = 178.40$, $p < .001$, and girls, $\Delta\chi^2(3, N = 1{,}226) = 224.68$, $p < .001$.

On the basis of the above CFA analyses and on the theoretical argument that the three factors gained with the YPI seem similar in content to what has been found with the PCL–R (Cooke & Michie, 2001), we chose to use the three-factor model in the present study. Alpha reliabilities of the three dimensions (that were created by taking the sum of the included subscales) of the YPI in the present sample were .82, .66, and .76 for the grandiose/manipulative, callous/unemotional, and the impulsive/irresponsible dimensions, respectively. The correlations among the three YPI dimensions ranged from .40 to .60, which is in line with previous findings (Andershed et al., 2002; Skeem & Cauffman, 2003).

Telephone Interview. In the telephone follow-up to nonresponders we used a reduced battery of YPI items (20 core YPI items were selected) to

explore potential differences between twins who responded to the self-report questionnaire and twins who did not. Twins who responded to the telephone interview scored significantly higher on the total score of the selected YPI items compared with twins who responded to the questionnaire (responders, $M = 34{,}15$; nonresponders, $M = 37{,}76$; mean difference = 3.61, $t(2310) = -5.92$, $p < .001$, $N = 2{,}312$). In addition, there were also significant differences between responders and nonresponders in socioeconomic status, $\chi^2(4, N = 2{,}217) = 27.63$, $p < .001$. That is, twins who responded to the questionnaire more often came from families with higher socioeconomic status than the twins who responded to the telephone interview.

Statistical Analyses

Univariate analyses. Twin materials are ideal for estimating the genetic and environmental effects of traits and diseases. MZ twins share the same genes, whereas DZ twins share on average half of the segregating genes. A measure of the similarity between twins is the intraclass correlation. Comparisons between the intraclass correlations for MZ and DZ twins provide information about the genetic and environmental effects that are present. A genetic effect is indicated if twin similarity is greater among MZ pairs than DZ pairs. Shared environmental effects reflect twin similarity that is not explained by genetic effects, whereas nonshared environmental effects reflect within-pair differences. The relative importance of genetic and environmental effects can be estimated by model fitting. In general, variance in the phenotype is assumed to be due to three latent factors: (a) additive genetic factors (a^2); (b) shared environmental factors (c^2); and (c) nonshared environmental factors (e^2; which also include measurement error). Within-pair similarity for the phenotype is due to the genetic and the shared environmental factor, as the nonshared environmental factor by definition makes members of a family different from one another. More specifically, the similarity between MZ twin pairs is assumed to be due to both additive genetic and shared environmental influences ($a^2 + c^2$). DZ pairs share only half of their segregated genes, and thus the within-pair similarity is assumed to be due to the sum of half the additive genetic factor plus the shared environmental factor ($.5\ a^2 + c^2$) (Neale & Cardon, 1992).

The path diagram in Figure 1 illustrates the relationship between the measured phenotype in the two members of a pair and the latent factors described earlier. The genetic correlation between MZ twin pairs is set at 1.0, whereas for the DZ twin pairs it is .5. The shared environmental correlation is set to 1.0 in both groups. By definition there is no within-pair correlation between the nonshared environments.

Sex differences. We fitted a series of models to test for sex differences (Neale & Martin, 1989). In the constrained model we assumed equal genetic and environmental variance components for boys and girls. The next step was to test whether there are sex differences in the relative importance of genetic and environmental factors by allowing the magnitude of the parameter estimates to differ between boys and girls, that is, modeling one set of parameters for boys in both like- and unlike-sex twin pairs and similarly another set of parameters for girls. This model is called the common effects sex-limitation model (Neale & Cardon, 1992), and it is assumed that although the same sets of genes and shared environment are important for boys and girls, their relative magnitude may differ. Finally one additional model was fitted to test whether there are different genes or environmental factors influencing phenotypic variation in the sexes. In this model we allowed not only different variance components for boys and girls but also allowed the genetic correlation between the members of opposite-sex twin pairs to vary. This model is called the general sex-limitation model (Neale & Cardon, 1992).

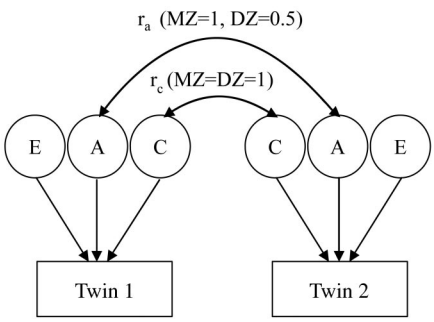

Figure 1. Basic path diagram for univariate twin data: generic (A), shared environmental (C), and nonshared environmental (E) effects on the measured variable are shown for both members of the twin pair. The genetic correlation r_a is set to 1 for MZ and 0.5 for DZ twins. The shared environmental correlation r_c is set to 1 for both MZ and DZ.

Multivariate analyses. Next, we wanted to study the genetic and environmental basis of the covariance between the three psychopathic personality dimensions. A bivariate Cholesky model (Neale & Cardon, 1992) was used to estimate the genetic correlations between each of the specified measures (the three psychopathic personality dimensions). A genetic correlation varies from +1.0 to −1.0 and indicates the extent to which genetic influences in one measure overlap with those on the second measure.

We also fitted a common pathway model (Kendler, Heath, Martin, & Eaves, 1987) to study the variance shared among the three dimensions simultaneously. With the common pathway model it is assumed that both genetic and environmental effects contribute to an intermediate latent variable, which in turn is responsible for the observed pattern of covariation between the measures in the model. Residual variance unique to each measure is also decomposed into unique genetic and environmental factors.

Univariate and multivariate models were fitted to raw data, allowing the inclusion of cases in which information is available from only one twin in a pair and cases in which information is available about a pair from just one measure, which increases the power in the analyses. Modeling was performed with the structural equation modeling package Mx (Neale, Boker, Xie, & Maes, 2003), which provides maximum-likelihood estimates of the different parameters.

Results

Descriptive Statistics and Twin Similarity

Means, standard deviations, number of respondents, and intraclass correlations for the YPI dimensions are presented in Table 1. The YPI data were transformed ($\log_{10}[x + 1]$) before analyses due to the skewness of the data. Means and standard deviations are reported for the raw scale, whereas the intraclass correlations are reported for the transformed scores. There were significant differences in the mean scores between boys and girls for the grandiose/manipulative dimension, $t(2175) = 6.09$, $p < .001$, and the callous/unemotional dimension, $t(2171) = 20.46$, $p < .001$, but not for the impulsive/irresponsible dimension, $t(2179) = 1.81$, $p < .07$. For all three dimensions, the DZ correlations were about half the magnitude of the MZ correlations, suggesting considerable genetic effects and minor shared environmental influences. Moreover, the intraclass correlations were similar for boys and girls and quite similar for same-sex and opposite-sex DZ twins, suggesting no sex differences in the genetic effects.

Univariate Model Fitting

The first two questions to be answered were the following: How much of the variance in the three psychopathic personality dimensions is due to genetic and environmental factors, when considered

Table 1
Descriptive Statistics and Twin Similarities

	Monozygotic twins				Dizygotic twins				Opposite-sex twins			
	n	*M*	*SD*	Intraclass correlation	*n*	*M*	*SD*	Intraclass correlation	*n*	*M*	*SD*	Intraclass correlation
Boys												
Grandiose/manipulative	383	6.4	1.8	.48***	325	6.5	1.8	.30***	332	6.7	1.8	.25***
Callous/unemotional	384	5.6	1.2	.47***	325	5.7	1.1	.26***	332	5.8	1.1	−.06
Impulsive/irresponsible	384	6.0	1.4	.57***	326	6.0	1.4	.20***	329	6.3	1.5	.22***
Girls												
Grandiose/manipulative	444	6.0	1.7	.61***	365	6.1	1.6	.26***	328	6.2	1.7	.25***
Callous/unemotional	445	4.7	1.0	.44***	363	4.8	1.0	.17***	324	4.8	1.0	−.06
Impulsive/irresponsible	449	5.8	1.4	.57***	365	6.0	1.4	.31***	328	6.2	1.4	.22***

*** $p < .001$.

separately? and Is there evidence for sex differences in genetic/environmental effects behind the three dimensions? To investigate this, we fitted a series of different models to test for differences in genetic and environmental influences between boys and girls. Model-fitting results from the different models and the parameter estimates from the best-fitting models for the three YPI-dimensions are presented in Table 2. First, we fitted a model in which we allowed different variance components for boys and girls as well as the genetic correlation between the members of the opposite-sex pairs to vary (Model 1). When we compared this model to the model in which we allowed the magnitude of the parameter estimates to differ between boys and girls (Model 2), Model 1 did not fit better than Model 2 for the grandiose/manipulative dimension (Δ − 2 log likelihood (LL) (1) = 0.0, $p = 1.0$), the callous/unemotional dimension (Δ − 2 LL(1) = 0.2, $p = .68$), or the impulsive/irresponsible dimension (Δ − 2 LL(1) = 0.1, $p = .81$). Next, we compared Model 2 and Model 3, in which we assumed equal genetic and environmental variance components for boys and girls. As shown in Table 2 the differences in −2 LL between Model 2 and Model 3 were not significant for the grandiose/manipulative dimension (Δ − 2 LL(3) = 5.6, $p = .13$), the callous/unemotional dimension (Δ − 2 LL(3) = 2.2, $p = .53$), or the impulsive/irresponsible dimension (Δ − 2 LL(3) = 1.3, $p < .74$), indicating that heritabilites are similar in boys and girls. Thus, we found no evidence of significant sex differences in genetic and/or environmental effects behind any of the three dimensions.

As shown in Table 2, in the best-fitting univariate model for the grandiose/manipulative dimension (with data from both boys and girls), genetic influences (heritability) were estimated to be 51%. Nonshared environmental effects explained almost all of the remaining variance, whereas the influence of shared environmental effects seemed to be of minimal importance. Similar patterns, with considerable heritable and little shared environmental influences were evident for the other two dimensions as well (see Table 2). In the best-fitting univariate models, genetic effects accounted for 43% and 56% of the variation on the callous/unemotional and impulsive/irresponsible dimensions, respectively.

Table 2
Model-Fitting Results and the Parameter Estimates from the Best-Fitting Models for the Three YPI Dimensions

	Variance components (95% CI)			Fit of model		Compared to model	Difference in fit of models		
	A	C	E	−2 LL	*df*		Δ −2 LL	Δ*df*	*p*
Grandiose/manipulative									
Model 1				−4152.41	2168	—	—	—	—
Model 2				−4152.41	2169	1	0	1	1
Model 3	**.51 (.33–.60)**	**.03 (.00–.17)**	**.46 (.40–.53)**	**−4146.81**	**2172**	**2**	**5.6**	**3**	**.13**
Callous/unemotional									
Model 1				−5427.02	2164	—	—	—	—
Model 2				−5426.85	2165	1	.2	1	.68
Model 3	**.43 (.30–.50)**	**.00 (.00–.10)**	**.57 (.50–.64)**	**−5424.64**	**2168**	**2**	**2.2**	**3**	**.53**
Impulsive/irresponsible									
Model 1				−4623.94	2172	—			
Model 2				−4623.88	2173	1	.1	1	.81
Model 3	**.56 (.44–.61)**	**.00 (.00–.09)**	**.44 (.39–.50)**	**−4622.63**	**2176**	**2**	**1.3**	**3**	**.74**

Note. Model 1: general sex-limitation model. Model 2: common effects sex-limitation model. Model 3: constraint model. CI = confidence interval. A = genetic variance component; C = shared environmental variance component; E = nonshared environmental variance component. A p value $< .05$ represents a significant change in −2 LL and thus a significant loss of fit. The best-fitting model is highlighted in boldface type. YPI = Youth Psychopathic Traits Inventory; LL = log likelihood.

Multivariate Model Fitting

The next question to be answered was: Is there a common genetic effect influencing the three personality dimensions? To answer this question we first fitted a series of bivariate Cholesky models to estimate the genetic correlations between each of the three psychopathic personality dimensions. The genetic correlations between the grandiose/manipulative, the callous/unemotional, and the impulsive/irresponsible dimensions ranged from .59 to .78, with the greatest genetic association between the grandiose/manipulative and the impulsive/irresponsible dimensions. These results suggest a substantial genetic overlap among the three dimensions.

Next, we fitted a common pathway model (Kendler et al., 1987) to explore the covariance among all three psychopathic personality dimensions simultaneously. This model is in line with the empirically supported hierarchical model of psychopathy (Cooke & Michie, 2001). That is, we used a model in which a latent construct of what we chose to label "psychopathic personality" (i.e., the covariation of the three factors of the YPI) is underpinned by the three factors of the YPI. Fitting a full common pathway model yielded the following: -2 LL $= -15768.58$; $df = 6511$.

The common pathway model gives a more stringent representation of the data, compared with, for example, the independent pathway model (Kendler et al., 1987; Neale & Cardon, 1992), which is a more general model. When we compare these two models, the results show a nonsignificant change of fit ($\Delta - 2$ LL(4) $= 7.66$, $p = .11$), suggesting that the common pathway model provides a better explanation of the data than the independent pathway model.

Figure 2 presents squared path estimates and confidence intervals for the full common pathway model. In this model, additive genetic factors explained 63% of the variance in the latent psychopathic personality factor, whereas nonshared environmental factors explained the remaining 37%. Shared environmental influences did not contribute to the explanation of psychopathic personality. Furthermore, the common latent psychopathic personality factor loaded most highly on the grandiose/manipulative dimension but also substantially on the other two dimensions. The final question to be answered was: Are there also unique genetic effects for the three separate dimesions? The decomposition of the unique genetic effects revealed that additive genetic factors explained 22% of the variance in both the callous/unemotional dimension and the impulsive/irresponsible dimension, whereas the unique genetic effect in the grandiose/manipulative dimension was negligible.

Discussion

We have shown that each of the three psychopathic personality dimensions (grandiose/manipulative, callous/unemotional, and impulsive/irresponsible) was significantly linked to a highly heritable psychopathic personality factor. Each of the three dimensions contained unique variance, suggesting some etiologic independence within the psychopathic personality constellation.

We found similar genetic influence in all three dimensions (43%–56%), when looking at them separately, which is consistent with two recent twin studies on psychopathic personality traits (Blonigen et al., 2003; Taylor et al., 2003). In addition, our results are consistent with most behavior genetic studies in personality showing that broad heritabilities across various FFM factors usually are in the range of 40%–60% (Bouchard & Loehlin, 2001).

We also investigated the etiological nature of the covariation among the three psychopathic trait dimensions. To date there is only one previous twin study in which the genetic overlap between different dimensions of psychopathy was examined (Taylor et al.,

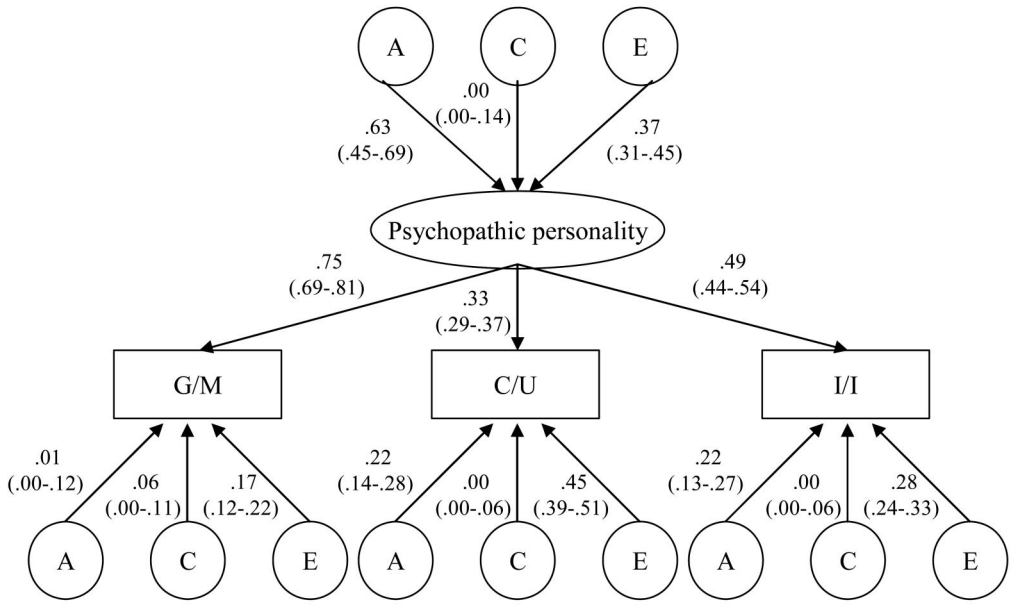

Figure 2. Squared path estimates for the full common pathway model, with 95% confidence intervals. G/M = grandiose/manipulative dimension; C/U = callous/unemotional dimension; I/I = impulsive/irresponsible dimension; A = genetic effects; C = shared environmental effects; E = nonshared environmental effects.

2003). This study showed a sizable genetic correlation ($r = .74$) between the callous/unemotional and impulsive/antisocial factors. In keeping with this previous research, we found genetic correlations among the grandiose/manipulative, callous/unemotional, and impulsive/irresponsible dimensions, ranging between .59 and .78, with the greatest genetic association between the grandiose/manipulative and the impulsive/irresponsible dimensions. These results suggest a strong relationship among genetic factors that are associated with variance in the three dimensions.

In addition, we showed that the three dimensions of the psychopathic personality do covary in a common latent factor, which is consistent with the notion of a hierarchical model of the psychopathic personality construct (Cooke & Michie, 2001; Forth et al., 2003; Hare, 2003). This latent psychopathic personality factor was influenced by genetic factors (63%), which has been assumed for a long time (e.g., Hare, 1970), but until now empirical evidence has been lacking. This relatively large heritability estimate for the latent psychopathic personality factor makes it a novel target for future research, especially because we cannot address specific biological mechanisms associated with the latent psychopathic personality in this study. Thus, to understand the genetic variance seen in the present study, the focus of future studies using samples of related or unrelated individuals should be on the latent psychopathic personality factor and the study of variables that theoretically are closely linked to genetic differences (e.g., Blair, 1999; Frick, 1998; Patrick, 1994).

The hierarchical common pathway model presented in this study also suggests etiologic distinctions among the three psychopathic personality dimensions. We found no evidence of unique genetic effects in the grandiose/manipulative dimension. That is, all the genetic variance for the grandiose/manipulative dimension is shared with the latent psychopathic personality factor. However, we found that unique genetic effects were of importance for the callous/unemotional and the impulsive/irresponsible dimensions. This finding is consistent with one previous twin study, which also showed residual genetic effects in the callous/unemotional dimension of psychopathy (Taylor et al., 2003) and suggests that there are genetic factors that uniquely impact the callous/unemotional and impulsive/irresponsible dimensions, over and above the genetic influence from the psychopathic personality. However, our data do not address the question of why this is the case, so we can only speculate. What is clear is that these two dimensions assess something that can occur independently of each other and also independently of the grandiose/manipulative dimension. One possible interpretation of this finding is that the callous/unemotional dimension also assesses traits that are close conceptually to low neuroticism, whereas the impulsive/irresponsible dimension assesses traits similar to low conscientiousness, that is, dimensions within the FFM of normal personality (see Lynam, 2002b).

Psychopathic Personality Constellation and Nonshared Environment

As would be expected, nonshared environmental influences were found to be significant in all of the analyses conducted. Nonshared environmental factors were shown to be important for explaining 37% of the variance in the latent psychopathic personality factor. If the standard assumption is made that error of measurement does not correlate across measures, the nonshared environmental contribution to the covariance is free of measurement error. Although we cannot directly address the environmental factors associated with the psychopathic personality constellation in the present study, our results clearly point out that it is essential to emphasize the fact that nonshared environmental factors have an important role in explaining the development of these personality traits. One possible interpretation of our results is that differences in peer relationships may account for some of the nonshared environmental influences seen in the latent psychopathic personality factor. It has been shown that during the course of adolescence, changes occur in the close relationships. As children grow older, friends take on an increasingly important role in the lives of adolescents (Savin-Williams & Berndt, 1990). In support of these findings, two previous twin studies (Manke, McGuire, Reiss, Hetherington, & Plomin, 1995; Pike & Atzaba-Poria, 2003) showed a substantial nonshared environmental influence for all aspects of friendship quality, indicating that adolescent siblings experience very different qualities of friendship. These findings highlight the importance of nonshared environmental influences during adolescence and indicate that friendship and peer relationships may be experiences leading to the well-documented sibling differentiation seen during adolescence (Pike & Plomin, 1997). Much less is known about the direct role of peer relationships for the development of psychopathic personality. In fact, this relationship has been explored in only one study, which showed positive associations between the psychopathic traits of the adolescent and levels of psychopathic traits in his or her friends (Andershed, Kerr, Stattin, & Engels, 2003), but more research is needed to address questions about causality.

The results from the present study also indicated that much of the nonshared environmental variance in the psychopathic personality constellation was residual or not attributable to nonshared environmental factors associated with the latent psychopathic personality factor. This might suggest some independence in the unique nonshared environmental effects that contribute to the development of the three personality dimensions. However, it is important to note that the unique nonshared environmental factor includes measurement error.

Psychopathic Personality Constellation and Shared Environmental Influences

The results from the present study suggest that shared environmental factors produce a negligible contribution to the variance in the psychopathic personality constellation. These results replicate those of recent twin studies examining self-reported psychopathic traits in adolescent (Taylor et al., 2003) and adult (Blonigen et al., 2003) twins in finding no evidence of shared environmental influences in psychopathic traits. The results are also consistent with evidence reported from many behavioral genetic studies of psychopathology (Bouchard & McGue, 2003) and personality (Bouchard & Loehlin, 2001; Loehlin, 1992; McGuffin & Thapar, 1992). However, it is important to keep in mind the fact that the genetic and environmental components estimated in our models can vary with age. That is, our findings do not preclude the possibility that shared environmental influences have had a more important role earlier in the development of psychopathic personality traits. For example, twin studies on antisocial behavior (Bouchard & McGue, 2003; Lyons et al., 1995) and personality (Loeh-

lin, 1992) have shown that the shared environmental effects decrease with age. Another issue that needs to be considered is that the classic twin model has limited statistical power to discriminate between the effects of shared genes and shared environments (Hopper, 2000).

No Sex Differences

Some studies have shown that males score higher than females in different measures of psychopathy (Cale & Lilienfeld, 2002), which is consistent with our findings. However, to our knowledge, there are no studies in which potential sex differences in genetic and environmental influences in the psychopathic personality constellation have been investigated. We found no significant sex difference in the genetic and environmental factors for the psychopathic personality dimensions, which is in line with most behavioral genetic studies in the field of normal personality (Bouchard & Loehlin, 2001).

Strengths and Limitations

The present study has a number of strengths, including the use of data from both boys and girls from a large population-based sample of twins with a narrow age span and with a high response rate. The inclusion of opposite-sex twins allowed us to investigate potential sex differences in genetic and environmental influences. Although the YPI has shown promise in a number of ways (Andershed et al., 2002; Skeem & Cauffman, 2003), there are some problems inherent in measuring psychopathic traits through self-report. The main problems are that deceitfulness, lying, and manipulation are symptoms of the psychopathic personality, which makes it difficult to get truthful responses to questions about characteristics such as shallow affect, lack of remorse or guilt, and grandiose sense of self-worth. The YPI was, however, developed with this specifically in mind (Andershed et al., 2002). Another limitation that needs to be considered is that we found evidence of systematic differences in YPI scores among twins who participated in the study versus twins who did not, which might affect generalizability. Also, because we have used a continuous measure of psychopathy in a sample of twins from the general population, our study does not address questions about the genetic and environmental influences on pathological levels of psychopathy. Therefore, it cannot be ruled out that the etiology of extreme range scores, indicative for individuals high in psychopathic traits, is different from that of normal range scores (Livesley, 1998).

There are also issues that need to be considered because of the use of the twin design. The first concerns the equal environmental assumption, which states that MZ and DZ twins share the common environment to the same extent. Therefore, if MZ twins are treated more similarly than DZ twins, this could overestimate the heritability. Although this equal environmental assumption has been criticized, various checks, such as incorporating environmental measures in twin studies and looking at the effects of mistaken zygosity, suggest that the equal environmental assumption generally is valid (Plomin, DeFries, McClearn, & McGuffin, 2001). Second, assortative mating was not taken into account in the present study. However, assortative mating acts to inflate the shared environmental influences, which were very small in our study, suggesting that such bias probably is negligible. Finally, no efforts were made to investigate the presence of gene–environment interactions or gene–environment correlations.

In summary, by using a hierarchical common pathway model, this study offers insights into the etiology of the psychopathic personality constellation in adolescence. We showed that genetic effects accounted for a substantial proportion of variance in the latent psychopathic personality factor, which makes it a promising target for future research. A key question for future research is to examine how genetic effects associated with the latent psychopathic personality factor in turn are related to antisocial behavior in adolescence and adulthood. Additionally, research using longitudinal methods can examine genetic influences on stability and changes of the latent psychopathic personality factor, which is a question that has major implications for the validity of this personality construct in adolescence. Answers to these kinds of questions are likely to increase our understanding of the origin of the psychopathic personality and provide useful information for prevention and treatment.

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Received October 22, 2003
Revision received September 25, 2004
Accepted September 28, 2004 ■