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The Heritability of Psychopathic Personality in 14 to 15 year Old Twins: A Multi-Rater, Multi-Measure Approach

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Abstract

No study has yet examined the genetic and environmental influences on psychopathic personality across different raters and method of assessment. Participants were part of a community sample of male and female twins born between 1990 and 1995. The Child Psychopathy Scale (CPS) and the Antisocial Process Screening Device (APSD) were administered to the twins and their parents when the twins were 14 to 15 years old. The Psychopathy Checklist: Youth Version (PCL:YV) was administered and scored by trained testers. Results showed that a one-factor common pathway model was the best fit for the data. Genetic influences explained 69% of the variance in the latent psychopathic personality factor, while non-shared environmental influences explained 31%. Measurement-specific genetic effects accounted for between 9% and 35% of the total variance in each of the measures, except for PCL:YV where all genetic influences were in common with the other measures. Measure-specific non-shared environmental influences were found for all measures, explaining between 17% and 56% of the variance. These findings provide further evidence of the heritability in psychopathic personality among adolescents, although these effects vary across the way in which these traits are measured, in terms of both informant and instrument used.

Keywords

Psychopathic Personality; Genetics; Parent; Self-Report; CPS; APSD; PCL:YV

Psychopathy in its adult form can be described as a personality disorder comprised of different dimensions. Behaviorally, a psychopath is an impulsive risk-taker with antisocial tendencies. Interpersonally, a psychopath is grandiose and manipulative. Affectively they lack empathy, anxiety, and remorse, and they have difficulties maintaining close relationships (Cleckley, 1941, 1976; Hare, 2002, 2003). Although the prevalence of psychopathy is very low (< 1% among adult males in the community), psychopaths are believed to constitute as much as 15–20% of all prison populations, and are thought to be associated with nearly half of all serious crimes (Blair, Mitchell, & Blair, 2007; Hare, 2003;

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Neumann & Hare, 2008). Recent studies have reported that genetic factors influence psychopathic traits during childhood (Bezdjian, Raine, Baker, & Lynam, 2010; Bezdjian, Tuvblad, Raine, & Baker, 2011; Fontaine, Rijdsdijk, McCrory, & Viding, 2010; Viding, Blair, Moffitt, & Plomin, 2005), adolescence (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2006; Forsman, Lichtenstein, Andershed, & Larsson, 2008; Henrik Larsson, Andershed, & Lichtenstein, 2006; H. Larsson et al., 2007; Taylor, Loney, Bobadilla, Iacono, & McGue, 2003), as well as adulthood (Blonigen et al., 2006; Tuvblad, Bezdjian, Wang, Raine, & Baker, submitted; Brook et al., 2010). A meta-analysis including 10 independent samples published before 2003 provided additional evidence that genetic factors were influential in explaining the variance in psychopathic personality. Results revealed that approximately 49% of the variance in psychopathic personality was due to genetic factors and the remaining 51% was attributable to the non-shared environment (Waldman & Rhee, 2006). However, no study has yet examined the genetic and environmental influences on psychopathic personality across raters and method of assessment. The present study aimed to fill this gap in the literature by examining the genetic and environmental influences on psychopathic personality using three different psychopathy measures including both parent ratings and youth self-reports in a community sample of 14 to 15 year old twins.

At present, the most widely used assessment tool to measure psychopathy in adults is the Hare Psychopathy Checklist-Revised (PCL-R; Hare, 2003). The PCL-R is a rating scale that describes psychopathy as a constellation of interpersonal, affective and behavioral personality traits. The PCL-R was developed to be used predominantly on an incarcerated population. It combines a semi-structured interview comprised of 20 items based on Cleckley's criteria (Cleckley, 1941, 1976) and a detailed review of correctional initiations charts. Since the PCL-R was initially developed for use on an incarcerated population, it contains a large number of items that are related to criminality. The reliability and construct validity for the PCL has been well established (Hare, Clark, Grann, & Thornton, 2000).

More recently, there has been increasing interest in examining psychopathic personality in youth. Similar to most personality disorders diagnosed in adulthood, the construct of psychopathy is assumed to consist of a stable set of maladaptive traits, attitudes and behaviors that begin during early childhood. However, assessment tools used to measure psychopathy in adults were not appropriate for use on children and adolescents (Edens, Skeem, Cruise, & Cauffman, 2001; Kotler & McMahon, 2005). As such, several assessment tools were developed to measure psychopathic personality in youth. Many of these assessment tools have been developed using the PCL-R as a model. This "downward translation" of psychopathy involves the exclusion of some items included in the PCL-R that are inapplicable or inappropriate for children, e.g., promiscuous sexual behavior or many short-term marital relationships and the addition of items that are more developmentally appropriate, e.g., concerning school work (Frick & Hare, 2002) and generally assess personality, e.g., behavioral dyscontrol as a behavioral manifestation of the disorder (Lynam & Gudonis, 2005). Three commonly used assessment tools used to measure psychopathic personality in youth include the Child Psychopathy Scale (CPS; Lynam, 1997), the Antisocial Process Screening Device (APSD; Frick & Hare, 2002), and the Psychopathy Checklist: Youth Version (PCL:YV; Forth, Kosson, & Hare, 2003). The PCL:YV incorporates collateral file review information (when available) along with a semi-structured

interview. The CPS and the APSD are available in both parent and self-report versions; which is an advantage as the use of multiple informants provides both subjective and objective data. Further, several factor structure models of the PCL scales have been suggested, for instance a four factor model comprised of interpersonal, affective, lifestyle and antisocial features; and a three factor model comprised of interpersonal, affective and lifestyle, i.e., omitting the antisocial dimension. Studies using PCL-based measures in adolescent samples reported that a four and a three factor model provide adequate fit in both males and females (Kosson et al., 2013).

To date, there have been only a few studies examining the genetic and environmental influences on psychopathic personality during adolescence. In an early study, 16–18 year old male participants in the Minnesota Twin-Family Study (MTFS) completed the Minnesota Temperament Inventory (MTI). The MTI is a 19-item self-report measure designed to capture the core features of psychopathy (Loney, Taylor, Butler, & Iacono, 2002). Genetic effects explained 39% of the variance in the Antisocial subscale and 42% of the variance in the Detachment subscale. The remaining variances in these two subscales were due to non-shared environmental influences (Taylor et al., 2003). A more recent study, also using data from the MTFS included male and female 17-year-old twins. Psychopathic personality was measured with the Multidimensional Personality Questionnaire (MPQ, 198 items, 11 primary subscales; Tellegen & Waller, 2008). The variance in the Fearless Dominance subscale was explained by genetic (45%) and non-shared environmental (55%) effects. Genetic effects explained 49% of the variance in the Impulsive Antisociality subscale, while non-shared environmental effects explained 51% (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2005). In an additional study, the Youth Psychopathic traits Inventory (YPI; Andershed, Kerr, Stattin, & Levander, 2002), a 50-item self-report questionnaire, was administered to a set of 16–17 year old male and female twins. Results demonstrated that genetic effects explained 63% of the variance in a higher order latent factor, comprised from three psychopathic personality dimensions (callous/unemotional, grandiose/manipulative, and impulsive/irresponsible). The remaining variance in this latent factor was explained by non-shared environmental effects (Larsson et al., 2006). In a follow-up study when these twins were 19–20 years old, the three psychopathic personality dimensions callous-unemotional, grandiose-manipulative and impulsive-irresponsible were found to be highly stable with genetic influences contributing substantially to their stability (Forsman et al., 2008). The aforementioned studies have all relied on self-reports; in addition, none of these studies examined genetic and environmental influences on psychopathic personality as assessed by the CPS (Lynam, 1997), the APSD (Frick & Hare, 2002), or the PCL:YV (Forth et al., 2003).

A recent study analyzed the convergent validity of several psychopathy measures using youth self-reports ($N=160$ incarcerated adolescents), parent-ratings ($N=35$), and interview-based measures. The self-report measures (APSD, CPS, ICU: Inventory of Callous-Unemotional Traits Youth version; Frick, Cornell, Barry, Bodin, & Dane, 2003), and the YPI (Youth Psychopathy Inventory; Andershed et al., 2002) had good convergent validity with the interview-based PCL:YV; the caregiver-report versions of these measures were not significantly correlated with their corresponding self-report versions nor were they

significantly correlated with the PCL:YV. Part of the poor agreement may have been related to the poor psychometric performance of the callous-unemotional subscale for most of these measures as well as the low response rates from caregivers (Fink, Tant, Tremba, & Kiehl, 2012). A shortcoming with this study is that it was not carried out on a genetically informative sample and could therefore not estimate the relative contribution of genetic and environmental influences on psychopathic personality within and across raters and measurement tools.

The present study aimed to expand on previous studies that have found a strong genetic influence on psychopathic personality in adolescents (Blonigen et al., 2005; Forsman et al., 2008; Larsson et al., 2006; Loney et al., 2002) by combining the unique perspectives of both parent ratings and youth self-reports across different measurement tools for psychopathy using a large community sample of 14 to 15 year old twins. The value in using this multi-rater, multi-method approach in assessing the genetic and environmental etiology of psychopathic traits is to incorporate the unique perspectives of different raters and measurement tools within a community sample of both male and female adolescent twins. Thus, based on previous studies that have reported significant genetic influences on psychopathic traits using various measurement instruments, we expected to find strong genetic influences common to all three measurement tools that we were using, i.e., CPS, APSD, PCL:YV. Additionally, based on previous findings investigating the genetic and environmental effects of psychopathic and antisocial behaviors in youth using multiple measures (Bezdjian et al., 2011), we expected to find some unique differences in the genetic and environmental factors influencing psychopathic traits, which may be attributed to each rater's unique perspective. We also expected to find a pattern of higher genetic effects in psychopathic personality based on parent ratings than youth self-reports (Rhee & Waldman, 2002). Further, investigating these traits in mid-adolescence may also be beneficial and add value over the existing literature on psychopathy. The more we can understand the genetic and environmental etiology of antisocial behaviors using different and unique instruments and raters who may provide different perspectives can help inform future studies. In the present study two specific research questions were addressed: (1) To what extent do genetic and environmental factors influence psychopathic personality within raters (parent ratings, youth self-reports) and method of assessment (CPS, APSD, PCL:YV)? (2) Can the variance in psychopathic personality be explained by common genetic and environmental factors across raters and measures?

Method

Participants

The data for the present study come from the University of Southern California (USC) Twin Study of Risk Factors for Antisocial Behavior (RFAB). RFAB is a longitudinal study of the interplay of genetic, environmental, social, and biological factors on the development of antisocial and aggressive behavior from childhood to young adulthood. Participating families were recruited from the Los Angeles community and the sample is representative of the ethnic and socioeconomic diversity of the greater Los Angeles area. To date, four waves of data have been collected, during the first assessment (Wave 1) the twins were 9–10 years

old ($N=614$ twin pairs, $mean\ age=9.60$, $SD=0.59$); during the second assessment (Wave 2) the twins were 11–13 years old ($N=445$ twin pairs, $mean\ age=11.79$, $SD=0.92$); during the third assessment (Wave 3) the twins were 14–15 years old ($N=604$ twin pairs, $mean\ age=14.87$, $SD=0.87$), and during Wave 4, the twins were 16–18 years old ($N=504$ twin pairs, $mean\ age=17.28$, $SD=0.77$). Complete details on study protocol including zygosity determination can be found elsewhere (Baker, Tuvblad, Wang, Gomez, & Raine, 2013). In the present paper, data from Wave 3 when the twins were 14–15 years old were used, since the APSD and the PCL:YV were not included in any of the other waves of assessment.

Procedure

Participants were invited to USC to take part in the study, which involved a ~6 hour laboratory assessment, divided into two 3 hour parts. Part I included both behavioral interviews and neurocognitive testing, while Part II involved psychophysiological assessment. One twin would participate in Part I, while the other would participate in Part II. In the meantime, the parent or primary caregiver, who were typically the biological mother (>90%), would complete all measures and interviews on one twin and then were asked to answer items about the second twin.

Measures

Child Psychopathy Scale (CPS)—The CPS is a well-validated measure for assessing psychopathic personality in youth (Lynam, 1997; Lynam, Derefinko, Caspi, Loeber, & Stouthamer-Loeber, 2007). The CPS was administered to both the twins (CPS-Y) and their parents (CPS-P). Data were available from $N=1,087$ for parent ratings, and $N=1,060$ for the twins' self-reports. The CPS was designed to operationalize psychopathic personality traits found in the Psychopathy Checklist–Revised (PCL–R; Hare, 1991) during childhood and adolescence. The items that make up the scale were drawn from the Child Behavior Checklist (CBCL; Achenbach, 1991) and the Common Language Q Set (a simplified version of the California Child Q Set; Caspi et al., 1992). The revised CPS (Lynam et al., 2005) assesses 13 of the 20 PCL–R constructs using the following two to five item scales: glibness, untruthfulness, manipulation, lack of guilt, poverty of affect, callousness, parasitic lifestyle, behavioral dyscontrol, lack of planning, impulsiveness, unreliability, failure to accept responsibility, and boredom susceptibility. The revision of the CPS was carried out to simplify complex items and to increase the reliability and validity of several constructs that were not optimally operationalized in the original version (i.e., glibness and shallow affect) (Lynam et al., 2005). Not all constructs from the PCL–R were included in the CPS, as they have no childhood counterparts (promiscuous sexual behavior, early behavior problems, many short term marital relationships, and revocation of conditional release) or did not correlate with other items (grandiosity). Two PCL items, criminal versatility and juvenile delinquency, were also excluded so that the CPS might serve as a pure measure of personality uncontaminated by antisocial behavior (Lynam et al., 2005). The psychometric properties of the CPS have been previously examined (Lynam, 1997). Confirmatory factor analyses on the CPS have identified a two-factor solution similar to PCL–R two-factor solution. However, these two factors are highly correlated ($r_p=0.95$) (Lynam, 1997), as such the majority of research using the CPS has used the total scale.

A two-factor solution has also been identified in the present sample using Wave 1 data when the twins were 9–10 years old, including a Callous-Disinhibited and a Manipulative-Deceitful factor (Bezdjian et al., 2010). The same two-factor solution has been identified using the Wave 2 and 3 data as well (analyses available from first author). In the present sample, the internal consistency (Cronbach's alpha: α) of the total scale was acceptable across raters: parent ratings (CPS-P) $\alpha=0.84$, and youth self-reports (CPS-Y) $\alpha=0.78$. The internal consistencies for the Callous-Disinhibited and the Manipulative-Deceitful were, respectively, $\alpha=0.70$ and $\alpha=0.75$ for parent ratings and $\alpha=0.68$ and $\alpha=0.71$ for youth self-reports.

The Antisocial Process Screening Device (APSD)—The APSD (Frick & Hare, 2002) was administered to both the twins (APSD-Y) and their parents (APSD-P) to provide ratings of the youth's psychopathic characteristics. Data were available from $N=1,010$ for parent ratings, and $N=1,076$ for the twins' self-reports. The APSD is a 20-item rating scale designed to assess each of the PCL-R dimensions deemed relevant to children and adolescents. Thus, several of the PCL-R items were not assessed including, leading a parasitic lifestyle, promiscuous sexual behavior, many short-term marital relationships, and revocation of conditional release. In place of these items, the APSD substitutes items assessing more developmentally appropriate behaviors (e.g., keeps same friends, teases others) or additional items (e.g., bragging and thinking oneself more important than others as indicators of grandiosity). Each relevant PCL-R construct was made into a single item that could be scored 0 (not at all true), 1 (sometimes true), or 2 (definitely true). The APSD was initially developed for ratings by parents and teachers, but it has been developed to collect self-report data from adolescents as well (Lynam & Gudonis, 2005). A three-factor solution has been suggested, based on two large samples of children who were administered the APSD, a Callous-Unemotional, a Narcissism and an Impulsivity factor (Kotler & McMahon, 2005). The Callous-Unemotional factor includes items such as feels bad or guilty (reverse scoring), is concerned about feelings of others (reversed scoring), the Narcissism factor includes items such as brags, can be charming, and the Impulsivity factor includes items such as blames others for mistakes, does not plan ahead.

The internal consistency (Cronbach's alpha: α) of the total scale for parent ratings (APSD-P) was $\alpha=0.84$, and $\alpha=0.74$ for youth self-reports (APSD-Y). The internal consistencies for the Callous-Unemotional, Narcissism and Impulsivity were $\alpha=0.69$, $\alpha=0.74$, $\alpha=0.62$ for parent ratings, and $\alpha=0.52$, $\alpha=0.64$, $\alpha=0.54$ for youth self-reports, respectively.

The Psychopathy Checklist: Youth Version (PCL:YV)—The PCL:YV was administered and scored by trained examiners in the context of the half-day visit at the University of Southern California laboratory when interviews and assessments of the twins took place. Data from $N=943$ subjects were collected and scored. The PCL:YV is a 20-item rating scale for the assessment of psychopathic traits in individuals aged 12 to 18 years old. The measure is modeled directly after the PCL-R (Hare, 1991) and is designed as a diagnostic tool rather than as a screening measure (Kotler & McMahon, 2005). The PCL:YV has been modified for an adolescent population by removing items that are inappropriate for adolescents, such as, parasitic lifestyle or many short-term marriages. The 20 items were

scored on a 3-point scale (0: item definitely does not apply; 1 item may or may not apply; and 2: item definitely applies). The items were divided into four factors: an Interpersonal factor, an Affective factor (which together correspond to PCL-R Facet 1), a Behavioral factor and an Antisocial factor (which together correspond to PCL-R Facet 2) which are consistent with the adult PCL-R. These factors can be used separately or summed together for a total scale (Kotler & McMahon, 2005).

The internal reliability (Cronbach's alpha: α) of the total scale was acceptable: $\alpha=0.83$. The internal reliabilities for the Interpersonal, Behavioral, Antisocial, and Affective factors were lower with $\alpha=0.53$, $\alpha=0.66$, $\alpha=0.35$ and $\alpha=0.66$ for the four factors, respectively.

In a subsample of 42 randomly selected participants, inter-rater reliability for the PCL:YV total scale was found to be strong ($r_p=0.89$). These participants were independently rescored by two examiners utilizing all available information, including video recordings of the entire testing day and all questionnaire-based, interview, and neurocognitive measures that were collected and used during the original assessment.

The scales were positively skewed, so each of the included scales were therefore ranked (PROC RANK, Blom option) and normalized (PROC STANDARD) using the statistical software SAS 9.1.3 (SAS, 2005), in order to achieve a more normal distribution within each scale.

Attrition Analyses: Attrition analyses were carried out to examine whether those who discontinued participating during Waves 2 or 3 differed from responders at baseline, i.e., Wave 1. Logistic regression analyses showed non-significant odds-ratios (OR) for socioeconomic status (Hollingshead, 1979) (OR=0.99, 95% CI, 0.98–1.01), gender (OR=.79, 95% CI, .59–1.07), and for psychopathic personality as assessed with the CPS (Lynam, 1997) (OR=1.00, 95% CI, .97–1.03). However, responders and non-responders were significantly different in ethnicity (OR=.70, 95% CI, .50–.99), with Caucasians less likely to drop out. Apart from this slight ethnic difference, those that discontinue participating in the study seemed to be doing so in a random manner.

Statistical Analyses

In the classical twin design co-variances between monozygotic (MZ) and dizygotic (DZ) twins are used to decompose the variance in measured trait to genetic and environmental components. MZ twins share their common environment and as well as 100% of their genes. DZ twins also share their common environment but share, on average, about 50% of their genes. By comparing the resemblance between MZ and DZ twins the total phenotypic variance of a measured trait can be divided into additive genetic (A), shared environmental (C), and non-shared environmental (E) factors. Shared environmental factors refer to non-genetic influences that contribute to similarity within pairs of twins. Non-shared environmental factors are those experiences that make siblings dissimilar, and this parameter also includes measurement error. Heritability is the proportion of total phenotypic variance due to genetic variation (Neale & Cardon, 1992).

Genetic Analyses

Univariate genetic models were first fit to the total scales (i.e., CPS-P, APSD-P, CPS-Y, APSD-Y and PCL:YV) and the subscales (i.e., CPS(P/Y): Callous-Disinhibited, Manipulative-Deceitful; APSD(P/Y): Callous-Unemotional, Narcissism, Impulsivity; PCL:YV: Interpersonal, Affective Behavioral, Antisocial) within each rater (parents and youth) to estimate the relative contributions of genetic (A), shared environmental (C), and non-shared environmental (E) factors to the variance in psychopathic personality. Univariate genetic models were compared to baseline saturated models, which perfectly capture the observed variances, co-variances and means for each twin and zygosity group. By using both male and female same sex twins it is possible to test for quantitative sex differences; that is, to test whether the magnitudes of genetic and environmental effects differ in males and females for a specific trait or disorder or if they can be constrained to be equal.

To further investigate the nature of the relationships across raters (parents and youth) and measures, two multivariate models were fit to the total scales (i.e., CPS-P, CPS-Y, APSD-P, APSD-Y and PCL:YV) simultaneously: a multivariate Cholesky decomposition and a one-factor common pathway model.

A multivariate Cholesky decomposes the variance of each phenotype (i.e., CPS-P, APSD-P, CPS-Y, APSD-Y and PCL:YV), as well as the co-variances among the phenotypes into genetic (A), shared environmental (C) and non-shared (E) environmental factors. A Cholesky decomposition has the same number of factors in each of the A, C, and E components as the number of observed variables. That is, the first genetic factor loads on all five measures, the second genetic factor loads on all measures (APSD-P, CPS-Y, APSD-Y and PCL:YV) except the first measure (CPS-P) and so on, until the last genetic factor only loads on the final remaining measure (PCL:YV); this same procedure repeats for the shared environmental (C) and non-shared (E) environmental components.

In a one-factor common pathway model, common genetic (A), common shared environmental (C), and common non-shared environmental (E) factors are mediated through the a shared latent factor that represents the variance shared among the five total scales (i.e., CPS-P, APSD-P, CPS-Y, APSD-Y and PCL:YV). The model estimates fewer parameters than the multivariate Cholesky decomposition and therefore, is more parsimonious. In addition to the genetic and environmental effects on the shared latent factor; As, Cs, and Es (which include measurement error) parameters that are specific to each measure are also estimated (McArdle & Goldsmith, 1990; Neale & Cardon, 1992).

All genetic models were fit with the structural equation program Mx (Neale, Boker, Xie, & Maes, 2003). The goodness of fit was compared through the difference in the chi-square statistic (χ^2). The suitability of the models was also determined by comparing the model's Akaike Information Criterion (AIC). The AIC represents the balance between model fit and the number of parameters (parsimony), with lower values indicating the most suitable model (Akaike, 1987). A last model-selection statistic was the Bayesian Information Criterion (BIC), where increasingly negative values correspond to increasingly better fitting models (Raftery, 1995).

Results

Descriptive Statistics

Mean sex differences were found with males displaying higher scores on average than females based on both parent ratings (CPS-P $t_{(1087)}=4.22, p<.001$, Cohen's $d = 0.25$; APSD-P $t_{(1008)}=5.57, p<.001$, Cohen's $d = 0.35$) and for youth self-reports (CPS-Y $t_{(1059)}=2.62, p<.001$, Cohen's $d = 0.16$; APSD-Y $t_{(1074)}=6.76, p<.001$, Cohen's $d = 0.41$; PCL:YV $t_{(941)}=4.44, p<.001$, Cohen's $d = 0.29$).

Twin Correlations and Univariate Genetic Models

Twin correlations within each rater (parents and youth) and measure (total- and subscales) are shown in the Appendix, Table A1. All significant MZ correlations were higher than DZ correlations, which indicate the importance of heritable influences. Note that for the PCL:YV Interpersonal subscale, boys' MZ correlation was non-significant, and girls' MZ and DZ correlations were significant and almost equal in magnitude, suggesting shared environmental influences.

Further, the DZ male correlations for parent rated CPS were low, which may be due to several reasons including bias in the parent reports of the CPS or perhaps due to non-additive or dominant genetic effects. However, model fitting analyses of the data revealed that non-additive genetic effects were non-significant and could be dropped from the overall saturated model (CPS-P: $\chi^2=0.15; df=2; p=0.93$). Even though the low DZ male correlations might suggest dominant genetic effects at work, the moderate to strong resemblance among DZ opposite sex pairs suggests otherwise and may influence any possible effects from the low DZ correlations.

Univariate genetic models were fit separately within each rater (parents and youth) and measure (total- and subscales), see Appendix Table A2. Based on AIC and BIC criteria, the best fitting model was an AE model in which the shared environmental influences could be dropped and the remaining parameters were constrained to be equal across sexes (e.g., parent ratings CPS Callous/Disinhibited subscale, AIC=816.66, BIC=-1906.82). Genetic influences accounted for about half of the variance in psychopathic personality based on both parent ratings and youth self-reports, with non-shared environmental effects accounting for the remaining portion of the variance. Note, for the PCL:YV Interpersonal subscale a full ACE (boys=girls) model is presented as it was difficult to determine whether an AE or CE model provided a better fit. These effects are displayed in Figure 1.

Correlations

Phenotypic correlations across raters (parents and youth) and measures (total scales) are presented in Table 1. The correlations among these measures of psychopathic personality and across raters ranged from $r_p=.35$ to $.80$.

Further, the two CPS subscales were moderately correlated (in parent ratings: $r_p=.55$; and in youth self-report: $r_p=.52$). Similarly, the APSD subscales were also moderately correlated (Callous-Unemotional and Narcissism in parent ratings: $r_p=.40$; and in youth self-report:

$r_p=.22$; Callous-Unemotional and Impulsivity in parent ratings: $r_p=.44$; in youth self-report: $r_p=.20$; Impulsivity and Narcissism in parent ratings: $r_p=.56$; and in youth self-report: $r_p=.48$); as well as the PCL:YV subscales (Interpersonal and Affective: $r_p=.50$; Interpersonal and Behavioral: $r_p=.43$; Interpersonal and Antisocial: $r_p=.37$; Affective and Behavioral: $r_p=.58$; Affective and Antisocial: $r_p=.46$; Behavioral and Antisocial: $r_p=.48$).

Multivariate Genetic Model Fitting

Next, a series of multivariate models were fit to the data across raters (parents and youth) and measures (total scales: CPS-P, CPS-Y, APSD-P, APSD-Y, PCL-YV) simultaneously. A one-factor common pathway model provided a better fit to the data than a multivariate Cholesky decomposition based on the AIC and BIC criteria (Table 2, Model 3, AIC=1893.71, BIC=-10003.37), and did not significantly differ from the saturated model ($\chi^2=248.62$; $df=215$; $p=.06$). Similar to the univariate analyses, estimates could be constrained to be equal in boys and girls (Model 3a, AIC=1861.26, BIC=-10086.48). This one-factor common pathway model could be further reduced by dropping all common and measurement specific shared environmental influences (Model 3b, $\chi^2=4.16$; $df=6$; $p=.66$).

Figure 2 displays standardized estimates for the best-fitting one-factor common pathway model. Squaring the standardized parameter estimates presented in Figure 2 provides the relative genetic and environmental contributions to the phenotypic variance. In the latent psychopathic personality factor 69% ($p<.05$) of the variance was due to genetic factors and the remaining 31% ($p<.05$) of the variance was due to non-shared environmental factors.

Significant measurement-specific genetic influences were found for CPS-P (35%), APSD-P (17%), CPS-Y (15%), and APSD-Y (9%), while genetic influences in the PCL:YV were entirely explained by the variance in the latent common factor. Significant measurement-specific non-shared environmental influences (which include measurement error) were found for all five measures: CPS-P (32%), APSD-P (17%), CPS-Y (38%), APSD-Y (36%), and PCL:YV (56%).

Discussion

The purpose of the present study was to examine the genetic and environmental influences on psychopathic personality across raters (parents and youth) and measures using both total score and subscales of the CPS (Child Psychopathy Scale; Lynam, 1997), the APSD (Antisocial Process Screening Device; Frick & Hare, 2002), and the PCL:YV (Psychopathy Checklist: Youth Version; Forth et al., 2003). Four conclusions can be drawn from the present study's results. First, our univariate results showed that genetic and non-shared environmental influences each explained about half of the variance in psychopathic personality based on the CPS and the APSD. Genetic influences only explained about one third of the variance in each of the three subscales Affective, Behavioral and Antisocial of the PCL-YV and non-shared environmental influences explained the remaining two thirds of the variance. Second, the univariate results also indicated higher heritability based on parent ratings than youth self-reports. Third, when submitting the total scales, i.e., CPS-P, APSD-P, CPS-Y, APSD-Y and PCL-YV, in a multivariate genetic design, a one-factor common pathway model best described the data. Genetic influences explained as much as two thirds

of the variance in this latent psychopathic personality factor and the non-shared environment explained the remaining one third of the variance. Fourth, measurement-specific genetic influences were only found for CPS-P, APSD-P, CPS-Y and APSD-Y, highlighting the unique aspects of these individual measures of psychopathy. Measure-specific non-shared environmental influences (including measurement error) were observed for all measures.

As expected, our analyses revealed higher mean values for psychopathic personality in males than in females. The prevalence of psychopathy has also been found to be lower among female than males in correctional, psychiatric and community samples (Blair et al., 2007; Nicholls, Ogloff, Brink, & Spidel, 2005). Consistent with previous research (Forsman et al., 2008; Larsson et al., 2007), no differences in the magnitude in genetic and environmental components were found across boys and girls and variance components could be constrained to be equal. In spite of the consistent sex difference in mean levels of psychopathic personality, the underlying etiologies of psychopathic personality appear to be similar for both sexes. There may still be biological and social differences between the sexes that might explain the greater mean levels of psychopathic personality seen in males. Future studies need to disentangle the circumstances or experiences that may lead to greater expression of psychopathic personality in males.

Further, the twin intraclass correlations for the PCL-YV Interpersonal subscale indicated the importance of the shared environment in the development of interpersonal traits among girls. Even though studies using PCL-based measures have suggested the same factor structure in both adolescent boys and girls (Kosson et al., 2013), there could be scalar equivalence, as item mean scores are typically ignored in factor analysis. A previous study using item response theory found that some PCL-R items performed differently in female offenders than in male offenders. Specifically, female offenders were achieving higher scores on Item 5 (“conning/manipulative”) which is part of the PCL-R Interpersonal subscale (Bolt, Hare, Vitale, & Newman, 2004). Future studies need to confirm our initial finding that shared environment may play an important role in the development of interpersonal psychopathic traits in females. If this finding proves to be robust, this suggests that familial experiences common for both twins in a pair (e.g., parenting, common peers) may partly explain or contribute to the development of these types of traits, specifically interpersonal traits in females.

Visual inspection of the overall pattern of the genetic and environmental variance components (see Figure 1) indicated that parents’ ratings tended to yield higher heritability estimates in these traits than did youth self-reports. Previous research has shown higher heritability estimates when parent or teacher ratings have been used as compared to self-reports (Rhee & Waldman, 2002). It is also possible that different raters are reporting on unique features of the same traits as individuals are likely to behave differently in different situations. Further, among the three different measures used, the lowest heritability and the largest non-shared environmental effects were found for the PCL:YV scales. In univariate genetic modeling, non-shared environmental effects also include measurement error. Since the test-retest reliability for the PCL:YV was strong, $r_p = .89$, the most likely explanation for these effects may be that the items for PCL-YV were endorsed by very few adolescents (the response rate across the 20 PCL:YV items varied from 0.11% (i.e., serious criminal

behavior) to 9.45% (i.e., poor anger management), with the majority of items being endorsed by about 2% of the youth).

Additionally, since the present study was the first to implement a clinical diagnostic measure such as the PCL:YV in a community sample, it may be more difficult to capture psychopathic traits in a community setting using a more structured diagnostic clinical tool. Thus, these effects may have been influenced by the low variability of psychopathy in a community sample, whereas the other self-report and interview measures more easily capture psychopathic personality in adolescents. Nevertheless, these measures present interesting and unique pieces of information for the further understanding of the development of psychopathic personality traits in adolescents.

Arguably, the most interesting result from this study is the clear preference for a one-factor common pathway model when the five total scales, i.e., CPS-P, APSD-P, CPS-Y, APSD-Y, PCL:YV, were analyzed simultaneously. Genetic influences explained 69% of the variance in this latent psychopathic personality factor and the non-shared environment explained 31%. It should be noted that the multivariate model used in the present study partitions measurement error away from variance in the latent psychopathic personality factor. The non-shared environment in the latent psychopathic personality factor was found to account for about one third of the variance, thus accounting for a significant amount of variance independent of measurement error. In our univariate analyses the non-shared environmental component accounted for about half of the variance. The non-shared environment includes experiences that are unique to each twin in a pair, although in univariate genetic models this effect also includes measurement error.

The results of the multivariate analyses further showed that measurement-specific genetic influences were found for CPS-P, APSD-P, CPS-Y and APSD-Y. This suggests that there were genetic influences that were not captured by the latent psychopathic personality factor, indicating that these assessment tools are measuring common as well as unique features of psychopathic personality traits. Measure-specific non-shared environmental influences were found for all measures, explaining between 17% and 56%. It should be noted that multivariate genetic modeling always includes measure-specific non-shared environmental influences, as this term also includes measurement error.

These findings should be viewed with a few limitations in mind. First, the sample used in the present study was a community sample of adolescent twins; thus, our findings cannot be extrapolated to clinical settings, such as prisons or forensic hospitals, the settings for which the construct of psychopathy has been previously validated (Hare, 2003). It should, however, be noted that community samples, as opposed to clinical-referred samples, do not introduce referral and selection biases. Second, there are several assumptions related to the classical twin design, for example the heritability estimate is population and time specific. For a review on these and other assumption in the classical twin design in relation to psychopathological disorders and behaviors, see (Plomin, DeFries, McClearn, & McGuffin, 2001; Tuvblad & Baker, 2011). We only used the subscales in our univariate modeling (i.e., CPS(P/Y): Callous-Disinhibited, Manipulative-Deceitful; APSD(P/Y): Callous-Unemotional, Narcissism, Impulsivity; PCL:YV: Interpersonal, Affective, Behavioral,

Antisocial), but not in the multivariate modeling, since no clear factor structure could be identified either within or across informants using confirmatory factor analyses as well as biometric multivariate modeling (results available from first author). Finally, the items for PCL:YV were endorsed by only a few adolescents. Low endorsements rates or homogenous responding to psychopathic personality traits can lead to range restrictions in the data, which in turn may influence correlation patterns and present a more muddled picture of the construct.

In conclusion, the total scales of the CPS-P, APSD-P, CPS-Y, APSD-Y and the PCL:YV were best explained by a one-factor common pathway model. Genetic influences explained about two thirds of the variance in this latent psychopathy factor. Measurement-specific genetic influences were found only for CPS-P, APSD-P, CPS-Y and APSD-Y. Measurement-specific non-shared environmental influences (including measurement error) were observed for all measures. These findings further suggest that different raters provide their own unique perspective on these traits. These various assessment tools seem to be measuring similar as well as different aspects of psychopathy; future research should identify the core items measuring psychopathic personality.

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Figure 1. Genetic and Environmental Influences for Psychopathic Personality, Age 14–15 Years
 Note: CPS=Child Psychopathy Scale, APSD=Antisocial Process Screening Device, PCL:YV=Psychopathy Checklist: Youth Version, P=parent ratings, Y=youth self-reports. A and C estimates for the PCL:YV Interpersonal subscale are non-significant, see Tables A1 and A2.

Figure 2. Best Fitting One-Factor Common Pathway for Psychopathic Personality, Age 14–15 Year

Notes: Standardized path estimates and 95% confidence intervals from the best-fitting One-Factor Common Pathway model for CPS-P (Parent rated Child Psychopathy Scale), CPS-Y (Youth Self-Reported Child Psychopathy Scale), APSD-P (Parent rated Antisocial Process Screening Device), APSD-Y (Youth Self-Reported Antisocial Process Screening Device), and PCL:YV (Youth – Psychopathy Checklist: Youth Version). Common additive genetic factors (A_c) and non-shared environmental factors (E_c) are depicted in circles. The latent factor is labeled Psychopathic Personality. Measured variables are depicted in rectangles. A_s : additive genetic residual variance specific to each measure, likewise for and non-shared environment (E_s).

Table 1

Phenotypic Correlations for Psychopathic Personality, Age 14–15 Years

	CPS-P	APSD-P	CPS-Y	APSD-Y	PCL:YV
CPS-P	1				
APSD-P	.80	1			
CPS-Y	.39	.40	1		
APSD-Y	.35	.40	.68	1	
PCL-YV	.35	.36	.52	.52	1

Note. CPS=Child Psychopathy Scale, APSD=Antisocial Process Screening Device, PCL:YV=Psychopathy Checklist: Youth Version, P=parent ratings, Y=youth self-reports, all correlations were significant at $p<.05$.

Table 2
Fit Indices for Multivariate Genetic Models of Psychopathic Personality, Age 14–15 Years

#		Overall fit				Chi-square difference			
		-2LL	df	AIC	BIC	χ^2	Df	p	χ^2 df p
1	Saturated model	11795.09	4860	2075.09	-9448.78				
2	Multivariate Cholesky ACE, males	12010.04	5045	1920.04	-9925.47	214.95	185	.07	
2a	Multivariate Cholesky ACE, males = females	12053.45	5090	1873.45	-10045.86	258.36	230	.10	43.41 45 .54
2b	Multivariate Cholesky ACE, males = females, Drop all C	12066.21	5105	1856.21	-10086.85	271.12	245	.12	12.76 15 .62
3	One-Factor Common Pathway, males	12043.71	5075	1893.71	-10003.37	248.62	215	.06	
3a	One-Factor Common Pathway, males = females, correlate unique effects within each rater	12073.26	5106	1861.26	-10086.48	278.17	246	.08	
3b	One-Factor Common Pathway, males = females, correlate unique effects within each rater, Drop all C	12077.42	5112	1853.42	-10103.34	282.33	252	.09	4.16 6 .66

Note. -2LL=-2(log-likelihood), AIC=Akaike's Information Criterion, BIC=Bayesian Information Criterion, χ^2 =difference in log-likelihoods between models, df= degrees of freedom. Included measures, total scales: CPS-P, CPS-Y (Child Psychopathy Scale), APSD-P, APSD-Y (Antisocial Process Screening Device), and PCL:YV=Psychopathy Checklist: Youth Version, P=parent ratings, Y=youth self-reports.

Table 3

Twin Correlations for Psychopathic Personality, Age 14–15 Years

	MZ boys	DZ boys	MZ girls	DZ girls	OS
<u>CPS-P</u>					
Callous/Disinhibited	.51	.05	.42	.11	.41
Manipulative/Deceitful	.40	.14	.50	.31	.41
Total	.44	.04	.57	.21	.46
<u>APSD-P</u>					
Callous-Unemotional	.67	.38	.63	.32	.47
Narcissism	.41	.34	.37	.28	.35
Impulsivity	.54	.27	.53	.32	.38
Total	.52	.38	.54	.38	.43
<u>CPS-Y</u>					
Callous/Disinhibited	.33	.19	.48	.10	.22
Manipulative/Deceitful	.49	.21	.49	.20	.23
Total	.39	.26	.53	.15	.27
<u>APSD-Y</u>					
Callous-Unemotional	.55	.19	.46	.28	.16
Narcissism	.23	.14	.38	.18	.18
Impulsivity	.27	.23	.39	.09	.33
Total	.39	.23	.54	.24	.33
<u>PCL:YV</u>					
Interpersonal	.09	.01	.24	.25	.13
Affective	.33	-.04	.25	.09	.09
Behavioral	.28	.26	.38	-.05	.16
Antisocial	.28	.17	.32	-.04	-.07
Total	.27	.13	.42	.15	.18

Note. CPS=Child Psychopathy Scale, APSD=Antisocial Process Screening Device, PCL:YV=Psychopathy Checklist: Youth Version, P=parent ratings, Y=youth self-reports. In the first four columns, values .20 and higher are significant at $p<.05$; in the final column, values .18 and higher are significant at $p<.05$.