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Psychopathic Personality and Negative Parent-to-Child Affect: A Longitudinal Cross-lag Twin Study

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Abstract

Purpose—Previous studies that have explored the relationship between parenting style and children's antisocial behavior have generally found significant bidirectional effects, whereby parenting behaviors influence their child's antisocial outcomes, but a child's behaviors also lead to changes in parenting style.

Methods—The present study investigated the genetic and environmental underpinnings of the longitudinal relationship between negative parent-to-child affect and psychopathic personality in a sample of 1,562 twins. Using a biometrical cross-lag analysis, bidirectional effects were investigated across two waves of assessment when the twins were ages 9–10 and 14–15, utilizing both caregiver and youth self-reports.

Results—Results demonstrated that negative parental affects observed at ages 9–10 influenced the child's later psychopathic personality at ages 14–15, based on both caregiver and youth self-reports. For these 'parent-driven effects', both genetic and non-shared environmental factors were important in the development of later psychopathic personality during adolescence. There were additional 'child-driven effects' such that children's psychopathic personality at ages 9–10 influenced negative parent-to-child affect at ages 14–15, but only within caregiver reports.

Conclusions—Thus, children's genetically influenced psychopathic personality seemed to evoke parental negativity at ages 14–15, highlighting the importance of investigating bidirectional effects in parent-child relationships to understand the development of these traits.

Keywords

longitudinal; psychopathic personality; negative parental affect; adolescent twins

Psychopathy in its adult manifestation is considered to be a complex personality disorder characterized by a constellation of interpersonal, affective, and behavioral traits (Hare, 2002). The prevalence rate of psychopathy in the general population ranges between 0.5% and 1% (Hare, 2003; Neumann & Hare, 2008) and between 15 and 25% in the incarcerated population (Hare, 1996). Psychopaths not only lack feelings of remorse, empathy and guilt, but they also lack responsibility and fear of punishment, and have difficulty regulating their

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emotions. Psychopathic traits seem to present with similar symptomologies across the lifespan (i.e., childhood, adolescence, adulthood) (Blair, 2005). Psychopathic personality has been found to be heritable in childhood (Bezdjian, Raine, Baker, & Lynam, 2010) and adolescence (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2005). Even though an increasing number of studies have examined psychopathic personality during early childhood (e.g., Viding, Blair, Moffitt, & Plomin, 2005), surprisingly few have focused on the relationship between conflictive/negative parent-child relationships and child and adolescent psychopathic tendencies. This is perhaps explained by the fact that many researchers have rejected the role of early environmental influences on the development of psychopathic personality (Marshall & Cooke, 1999). The present study aimed to fill this gap in the literature by examining the relationship between negative parental affect and psychopathic personality in a sample of twins studied from pre- to middle adolescence. By examining these relationships in a genetically informative design, it is possible to investigate the direction as well as the genetic and environmental etiology of parenting and child behaviors.

The Relationship between Parenting Style and Child Psychopathic Personality

Studies examining the relationship between parenting style and child antisocial behavior emphasize the importance of parenting characteristics in the development of children's antisocial behavior. Specifically, harsh and inconsistent parenting is associated with the development of aggression, delinquency, and conduct problems. In this "parent effects model" negative parenting style is believed to influence a child's antisocial behavior (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000; Maccoby, 2000; Moffitt, 2005). The few studies that have examined the relationship between parenting style and psychopathic personality have found evidence for an association between parenting style and children's later psychopathic personality during adulthood. Studies using a retrospective study design have shown that male psychopathic prisoners had more adverse experiences during childhood (i.e., neglect, abuse, poor supervision) than non-psychopathic prisoners (Marshall & Cooke, 1999). Individuals high on psychopathic personality had experienced higher levels of childhood neglect and/or abuse compared to those low on psychopathic personality (Lang, af Klinteberg, & Alm, 2002). A more recent study using a prospective study design reported that poor parental bonding (lack of maternal care and low paternal overprotection) and childhood physical abuse at age 3 were both associated with psychopathic personality at age 28 (Gao, Raine, Chan, Venables, & Mednick, 2010). While most of these previous studies suggest a strong relationship between parenting style and psychopathic personality, there is also evidence that children with psychopathic personality are not affected by the type of parenting received. For example, a study including 136 Hispanic females found that parenting style was unrelated to the presence of psychopathic traits, including callousness, suggesting that these traits have a unique developmental pathway independent from parenting style or the quality of parenting (Vitacco, Neumann, Ramos, & Roberts, 2003). Taken together, however, the majority of these studies suggest that parenting style has an important influence on the development of psychopathic personality, suggesting parent-driven effects.

There is also evidence that the association between parenting style and children's antisocial behavior is not solely driven by parental affects, but may also be instigated by the child ('child effects model'). Specific child characteristics (e.g., child's oppositional, irritable behavior) elicit (negative) responses from the environment. That is, some parental aggression may be evoked or may be a reaction to-rather than a cause of-antisocial and aggressive behavior exhibited from their children (Moffitt, 2005). Adoption studies of antisocial behavior have yielded results supporting the hypothesis that aggressive children

elicit more negative environments from their parents (Ge et al., 1996; O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998). For example, O'Connor et al. (1998) found that children at genetic risk (based on the biological mother's characteristics) received more negative parenting from their adoptive parents than children not at genetic risk. A more recent study showed an association between parental negativity and genetic risk for psychopathic personality traits (Beaver, Barnes, May, & Schwarz, 2011). Thus, bidirectional effects most likely exist between parenting characteristics and children's antisocial behavior, in which both child and parent driven effects operate in a reciprocal manner.

Using a Genetically Informative Longitudinal Design to Examine Bidirectional Effects

Based on previous research we know that children's genotypes can influence parenting behaviors. However, this is not to say that parenting is driven entirely by children's genotypes or by their behaviors. Parents' own genetic predispositions can also influence the ways in which they parent their children. Although it is still possible that parents can have an important influence on child outcomes through environmental mechanisms. Using a bidirectional effects model (i.e., a cross-lag model) in a genetically informative design makes it possible to examine the etiology of parenting-child outcome relationships by testing for both environmental and genetic effects simultaneously. Understanding the direction and genetic and environmental etiology of the association between parenting style and psychopathic personality will advance our understanding of the development of psychopathic personality. This could ultimately have consequences for intervention and prevention strategies for reducing psychopathy and other externalizing behavior problems.

To date, only a handful of studies using a longitudinal and genetically informative design have investigated bidirectional effects between parenting style and children's antisocial behavior. In a study including a sample of same-sexed twins from the Minnesota Twin Family Study at ages 11 and 14 years, caregiver and youth-self reported data were combined. Parent-child-conflict was partly a result of parent's responses to their child's heritable antisocial behavior, but also due to contributions from the child's antisocial behavior via environmental (shared and non-shared) mechanisms (Burt, McGue, Krueger, & Iacono, 2005). Similar findings were reported in a sample of 4 and 7 years old twins participating in the Twins Early Development Study (TEDS). Parents' negative feelings towards their children environmentally (shared and non-shared) influenced the risk of antisocial behavior in their children, and heritable antisocial behavior evoked parental negativity towards the child (Larsson, Viding, Rijdsdijk, & Plomin, 2008). An additional study including adoptive siblings and twins, the Nonshared Environment in Adolescent Development (NEAD) at ages 10–18 and 13–21 years showed that the cross-lag association between parental conflict-negativity and adolescent antisocial behavior/depressive symptoms was primarily explained by genetic factors (Neiderhiser, Reiss, Hetherington, & Plomin, 1999). Taken together, these findings showcase the importance of a bidirectional relationship between parenting style and antisocial (and internalizing) behavior. Based on these findings, parenting style seems to be partly contributing to the development of antisocial behavior. In turn, this relationship is partly explained by the child's heritable antisocial behavior. However, these studies did not specifically examine the etiology of bidirectional effects between parenting style and child psychopathic personality.

Research has further demonstrated that both caregiver ratings and youth self-report are beneficial in assessing complex traits (Bezdjian, Tuvblad, Raine, & Baker, 2011). A study using parent ratings of psychopathic personality reported intraclass correlation coefficients of .80–.88 in a 2–4 year follow-up (mean age at intake: 10.65) (Frick, Kimonis, Dandreaux, & Farell, 2003). Muñoz and Frick (2006) investigated the 2-year stability of self-reported

psychopathic personality in the same sample and reported slightly lower coefficients of .43–.58 (Muñoz & Frick, 2007). These findings stress the importance of utilizing different informants when trying to understand complex traits. Different informants are likely to report different aspects of a behavior, partly because individuals behave differently in different situations and partly because some behaviors are more likely to be encountered and witnessed than certain other behaviors (Bezdjian et al., 2011). In the present study, data based on parent ratings and youth self-reports were utilized and analyzed separately in order to provide a more comprehensive account of the relationship between parenting style and psychopathic personality.

The Present Study

The present study used data from a longitudinal study of twins, with data collected from both caregiver and youth self-reports at two waves of assessment when the twins were 9–10 and 14–15 years of age. Using these longitudinal data, a cross-lagged (or bidirectional effects) model was employed to investigate the relationship between parenting style (negative parent-to-child affect) and psychopathic personality in children. Specifically, we used a biometric cross-lagged model to address the following research questions: (1) How much of the variance in psychopathic personality and negative parent-to-child affect is explained by genetic, shared environmental and non-shared environmental influences within each wave? (2) Is the association between negative parent-to-child affect and psychopathic personality mainly explained by parent-driven effects, or child-driven effects, or a combination of both? (3) How do genetic and environmental influences contribute to parent-driven effects, and/or child-driven effects? Based on previous findings from Burt et al., (2005) and Larsson et al. (2008) examining parenting style and antisocial behavior, we would expect to find significant genetic and environmental effects influencing the relationship between negative parent-to-child affect and psychopathic personality at ages 14–15. Specifically, we predicted that negative parent-to-child affect would contribute to children's psychopathic personality through environmental mechanisms. We did not have a specific hypothesis on whether these effects would be driven by shared or non-shared environmental influences. Additionally, we hypothesized that childhood psychopathic personality might influence and instigate negative parenting via genetic mechanisms (that is, children's psychopathic behavior may evoke negative parenting). We also examined sex differences and based on previous studies we did not expect to find any significant sex differences in the genetic and environmental contribution to the associations between negative parent-to-child affect and psychopathic personality (Burt et al., 2005; Larsson et al., 2008).

Method

Participants

The sample was drawn from participants in the University of Southern California (USC) Risk Factors for Antisocial Behavior (RFAB) twin study. RFAB is a prospective study of the interplay of genetic, environmental, social, and biological factors on the development of antisocial and aggressive behavior from childhood to emerging adulthood. Participating twins and their families were identified through the Los Angeles, Orange and Ventura County public school districts, direct contact of private schools, as well as through advertisements and referrals (e.g., by other participants, Mothers of Twins clubs). Families of identified twins were sent letters inviting them to participate in the study, and those interested were asked to return postage-paid information forms or contact us for participation in the study. The twins were evaluated using an extensive protocol, including cognitive, behavioral, psychosocial and psychophysiological measures. Likewise, the caregivers (biological mother >90%) were interviewed using questionnaires and clinical

interviews aimed at assessing the behavior, psychological status, and personality of each twin, as well as his/her psychosocial tendencies, prenatal and life experience. The sample is representative of the ethnic and socioeconomic diversity of the greater Los Angeles area (Baker, Tuvblad, Wang, Gomez, & Raine, 2013).

The present study utilizes data from two separate waves of assessment; the first (time-1) and the third wave of assessment (time-2) (see Table 1), as parent-to-child-affect was not collected during Wave-2. During 2001–2004 (Wave-1) when the twins were 9–10 years old (*mean age*=9.60, *SD*=0.59), and during Wave-3 when the twins were 14–15 years old (*mean age*=14.87, *SD*=0.87), assessed from 2006–2010. The total study sample includes 1,562 subjects, including 169 monozygotic (MZ) male, 171 MZ female, 121 dizygotic (DZ) male, 120 DZ female, and 200 DZ opposite-sex twin pairs.

Zygosity Determination

Zygosity was based on DNA microsatellite analysis (>7 concordant and zero discordant markers=MZ; one or more discordant markers=DZ) for 87% of the same-sex twin pairs. For the remaining same-sex twin pairs, zygosity was established by questionnaire items about the twins' physical similarity and the frequency with which people confuse them. The questionnaire was used only when DNA samples were insufficient for one or both twins. When both questionnaire and DNA results were available, there was a 90% agreement between the two (Baker, Barton, Lozano, Raine, & Fowler, 2006).

Measures: Children's Psychopathic Personality

Psychopathic personality in the children was assessed with the Child Psychopathy Scale (CPS), a well-validated measure for assessing psychopathic personality in youth (Lynam, 1997). The CPS was administered to the twins and their caregivers at Waves 1 and 3. The CPS was designed to measure psychopathic traits found in the Psychopathy Checklist–Revised (PCL–R; Hare, 1991) during childhood and adolescence. The revised CPS (Lynam et al., 2005) assesses 13 of the 20 PCL–R constructs as 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 undertaken 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 not included so that the CPS might serve as a pure measure of personality uncontaminated by antisocial behavior (Lynam et al., 2005). In the present sample, the internal reliability (Cronbach's α) of the total score was acceptable, caregiver reports, Wave-1, α =0.80, and Wave-3, α =0.84, and youth self-reports, Wave-1: α =0.75, and Wave-3: α =0.78.

Negative Parent-to-Child Affect

Negative Parent-to-Child Affect was assessed in both the children and their parents using an adaptation of the validated Parent Feelings Questionnaire (Deater-Deckard, 1996). Both Positivity and Negativity were assessed using a 30-item questionnaire assessing parental affect. The format of responses ranged from never, rarely, sometimes, often or always. Parallel instruments were used for youth and caregiver reports, and administered using survey design software which allowed slight wording changes tailored to the perspective of each informant (e.g., How often do you criticize [name of child]? vs. How often does [your

caregiver] criticize you?). The Negative Affect score utilized in the current study was composed of 5 items—a composite score was calculated summing together the following items assessing negative affect in caregiver reports (Cronbach's α for the 5 negativity items in caregiver reports were moderately acceptable Wave-1, $\alpha=0.63$; Wave-3 $\alpha=0.61$):

- How often do you wish that [child name] would leave you alone for a few minutes?
- How often do you nag [child name] about what s/he is doing wrong?
- How often do you criticize [child name]?
- How often do you find yourself wishing that [child name] were different in some ways?
- Are there things [child name] avoids talking about with you?

Similarly, youth self-reported items were also summed together to create a composite of negative parental affect using the same 5 items (Cronbach's alphas for the 5 negativity items in youth self-report were moderately acceptable Wave-1, $\alpha=0.56$; Wave 3 $\alpha=0.66$).

The measures (psychopathic personality, negative parent-to-child affect) were skewed and were therefore ranked (PROC RANK using the BLOM option) and normalized (PROC STANDARD) using the statistical software SAS 9.1.3 (SAS, 2005).

Statistical Analyses

Descriptive statistics (means and standard deviations) were first computed for the psychopathic personality and negative parent-to-child affect at the two waves. Next, phenotypic correlations and cross-time correlations (e.g., Time-1 and Time-2) were calculated.

The classical twin design is a natural experiment that relies on the different levels of genetic relatedness between MZ and DZ twins in order to estimate the relative contribution of genetic and environmental factors to individual differences in a phenotype of interest. The total phenotypic variance of a measured trait can be divided into additive genetic factors (A), shared environmental factors (C), and non-shared environmental factors (E). Since MZ twins share all their alleles, additive genetic factors are perfectly correlated in MZ twins. DZ twins correlate 0.5, because DZ twins share on average half of their segregating alleles. Shared environmental factors refer to non-genetic influences that contribute to similarity within pairs of twins. Shared environmental influences contribute equally to the similarity in MZ and DZ twins, and thus shared environmental factors correlate 1.0 in both MZ and DZ twins. Non-shared environmental factors are those experiences that make siblings dissimilar. There is no correlation for the unique environment by definition, and this parameter also includes measurement error (Neale, Boker, Xie, & Maes, 2003).

A cross-lagged model was utilized to examine the association between psychopathic personality and negative parent-to-child affect between childhood (ages 9–10) and adolescence (ages 14–15). In a cross-lagged model all phenotypic associations across the two time-points are expressed as partial regression coefficients. The cross-age (stability) parameters (b_{11} , b_{22} , see Figure 1) estimate the stability of negative parent-to-child affect and psychopathic personality across the two time points, while controlling for the preexisting association between the two phenotypes at time-1 (ages 9–10 years), see Table 1. The two cross-lag parameters (b_{12} , b_{21}) estimate the independent contribution of negative parent-to-child affect (or psychopathic personality) at time-1 on psychopathic personality (or negative parent-to-child affect) at time-2 (age 14–15) years while controlling for the stability and preexisting association at time-2. That is, did negative parent-to-child affect at ages 9–

10 years impact psychopathic personality at time-2 above and beyond its relationship with psychopathic personality during time-1?

The model also estimates univariate biometrical parameters which decompose variance in both psychopathic personality in the children and the parent-to-child affect measures, as well as their covariance within each assessment; (1) *total* effects of genetic (A), shared (C) and non-shared (E) environmental effects on individual traits at time-1; (2) additional genetic, shared and non-shared environmental effects *unique* to time-2, as well as (3) the genetic, shared and non-shared environmental overlap between negative parent-to-child affect and psychopathic personality within each time-point, i.e., genetic (r_g), shared (r_c) and non-shared (r_e) environmental correlations.

The cross-lagged associations (i.e., between early parenting with later child psychopathy, and vice versa) can also be decomposed into genetic, shared and non-shared environmental factors, in order to explain the sources of variance in both later parental negativity and child psychopathic personality. Variance in parental negativity during adolescence (time-2) can be decomposed into: (1) cross-age (stability) effects: genetic and environmental influences on negative parent-to-child affect at time-1 contributing to the heritability in negative parent-to-child affect at time-2 (e.g., genetic effects: $b_{11}^2 * a_1^2$); (2) cross-lagged effects: genetic and environmental influences unique to children's psychopathic personality at time-1 explaining the heritability in parental negativity at time-2, i.e., *child-driven effects* (e.g., genetic influences: $b_{12}^2 * a_2^2$); (3) genetic and environmental effects common to negative parent-to-child affect and psychopathic personality at time-1 (e.g., genetic influences: $2 * [b_{11} * a_1 * r_{g1} * a_2 * b_{21}]$), and (4) residual effects: genetic and environmental effects unique to negative parent-to-child affect at time-2 (e.g., genetic influences: a_3^2), see Table 1.

Similarly, the genetic and environmental variance for children's psychopathic personality during adolescence (time-2) can be decomposed into these four sources: (1) cross-age (stability) effects from early psychopathic personality at time-1 (e.g., genetic effects: $b_{22}^2 * a_2^2$); (2) cross-lagged effects from early parental negativity at time-1 representing 'parent driven effects' (e.g., genetic effects: $b_{21}^2 * a_1^2$), (3) common effects shared by parental negativity and child psychopathic personality at time-1 (e.g., genetic effects: $2 * [b_{22} * a_2 * r_{g1} * a_1 * b_{12}]$); and (4) residual effects unique to child psychopathic personality at time-2 (e.g., genetic effects: a_4^2).

To test for sex differences in the variance components, we compared a model in which the magnitudes of genetic and environmental effects were allowed to differ between males and females, against a model in which the estimates were constrained to be equal.

All models were fit with the structural equation program Mx (Neale et al., 2003). The adequacy of fit was assessed by computing twice the difference between the log-likelihood of a full model and that of a submodel, in which parameters are fixed to be zero or constrained to be equal. This difference follows a χ^2 distribution with the difference in the number of estimated parameters in the two models as the degrees of freedom. A significant χ^2 indicates that the model with fewer parameters to be estimated fits the data worse. The suitability of the models was also determined by comparing the model's Akaike Information Criterion (AIC) (Akaike, 1987) and Bayesian Information Criterion (BIC) (Raftery, 1995), where increasingly negative values correspond to increasingly better fitting models.

Results

Descriptive Statistics

Table 2 presents number of participants, means and standard deviations for the raw (untransformed) psychopathic personality and negative parent-to-child affect scales. Significant mean differences across sex were found for caregiver reports of psychopathic personality at time-1 ($t_{(1217)}=5.66, p<.001$) and time-2 ($t_{(1087)}=4.22, p<.001$), as well as for negative parent-to-child affect at time-2 ($t_{(879)}=3.23; p<.001$) with higher mean scores observed for boys in all cases. Similarly, significant mean differences across sex were found for youth self-reports of psychopathic personality at time-1 ($t_{(1208)}=5.28, p<.001$) and time-2 ($t_{(1059)}=2.62, p<.001$) with boys showing higher mean scores. Boys and girls did not differ, however, in their own reports of negative parent-to-child affect.

Phenotypic Correlations

The phenotypic stability across the two time points for caregiver reports was moderately strong and significant ($r_p = 0.46$ and 0.62 for ratings of psychopathic personality in boys and girls, respectively, and $r_p = 0.48$ and 0.50 in their ratings of negativity towards boys and girls (all $p < 0.05$; see Table 3). For youth self-reports of psychopathic personality the phenotypic stability was also moderate and significant ($p < 0.05$): $r_p = 0.37$ in boys and 0.27 in girls, while stability of parental negativity appeared less strong and non-significant: $r_p = 0.11$ for boys and 0.10 for girls. The phenotypic associations *between* the parental negativity and children's psychopathic personality *within* each time were moderate and significant for caregiver reports in both boys ($r_p = 0.41$ for time-1 and 0.44 for time-2), and in girls ($r_p = 0.43$ for time-1 and 0.54 for time-2). These parenting-child outcome associations were also significant across the two time points: $r_p = 0.21$ and 0.36 for early parenting and later psychopathic personality in boys and girls, respectively; $r_p = 0.33$ and 0.39 for early psychopathic personality and later parental negativity in boys and girls, respectively. For youth self-reports, the phenotypic correlations *between* the two measures *within* were $r_p = 0.25$ (time-1) and 0.30 (time-2) in boys, and $r_p = 0.34$ (time-1) and 0.43 (time-2) in girls, and somewhat lower *across* the two time points: $r_p = 0.10$ and 0.22 in boys; $r_p = 0.05$ and 0.17 in girls.

Cross-lagged Model Fitting Analyses

Model-fitting results for psychopathic personality and negative parent-to-child affect are displayed in Table 4. Based on AIC and BIC criteria, a model constraining male and female components to be equal best described the data (caregiver reports, AIC=2232.519, BIC=8796.78, youth self-reports, AIC=2962.25, BIC=-8070.12). Parameter estimates from the best-fitting cross-lagged models are displayed in Figure 1 for caregiver ratings and (youth self-reports). We will go over the results for caregiver ratings and present the results for youth self-reports in brackets.

Univariate ACE Estimates: Caregiver Reports [Youth Self-Reports]

Genetic influences at time-1 accounted for 21% (A_1^2) (95% CI, .04–.39) [26%; 95% CI, .02–.40] of the phenotypic variance in negative parent-to-child affect, shared environmental factors accounted for 42% (C_1^2) (95% CI, .27–.56) [4%; 95% CI, .00–.23], and non-shared environmental factors accounted for 36% (E_1^2) (95% CI, .31–.43) [70.5%; 95% CI, .60–.81]. For psychopathic personality, genetic influences accounted for 53% (A_2^2) (95% CI, .37–.60) [39%; 95% CI, .18–.48] of the phenotypic variance, and non-shared environmental factors accounted for 47% (E_2^2) (95% CI, .40–.55) [61%; (95% CI, .52–.71)].

Univariate (residual) estimates of genetic and environmental influences *unique* to time-2 showed that genetic influences accounted for 1% (A_3^2) (95% CI, .00–.23) [49%; 95% CI, .

22–.60] of the phenotypic variance in negative parent-to-child affect, shared environmental factors accounted for 38% (C_3^2) (95% CI, .20–.47) [1.2%; 95% CI, .00–.23], and non-shared environmental factors accounted for 40% (E_3^2) (95% CI, .32–.48) [46%; 95% CI, .38–.57]. For psychopathic personality, genetic influences accounted for 27% (A_4^2) (95% CI, .05–.43) [41%; 95% CI, .14–.52] of the phenotypic variance, shared environmental factors accounted for 8% (C_4^2) (95% CI, .00–.26) [.01%; CI, .00–.21], and non-shared environmental factors accounted for 39% (E_4^2) (95% CI, .32–.48) [50%; 95% CI, .41–.61]. As some of the variance at time-2 is contributed from time-1 the variance components estimates do not sum to 100%. That is, negative parent-to-child affect (1+38+40) sum to 79 and psychopathic personality sum to (27+8+39) 74. About 21% of the variance in negative parent-to-child affect and 26% in psychopathic personality at time-2 were explained by negative parent-to-child affect and psychopathic personality at time-1. [Similarly for youth self-report: negative parent-to-child affect (49+1+46) sums to 96% and psychopathic personality sum to (41+0.01+50) approximately 91%. About 4% of the variance in negative parent-to-child affect and 9% in psychopathic personality at time-2 were explained by negative parent-to-child affect and psychopathic personality at time-1].

Total genetic and environment estimates at time-2 is computed by adding on the genetic, shared and non-shared environmental contribution from negative parent-to-child conflict and psychopathic personality at time-1. Thus, total negative parent-to-child conflict at time-2 was A=8% (95% CI, .03–.30), C=44% (95% CI, .26–.52), E=48% (95% CI, .40–.54); and for psychopathic personality A=41% (95% CI, .18–.55), C=9% (95% CI, .001–.26), E=50% (95% CI, .43–.60). [Similarly for youth self-report: total negative parent-to-child conflict at time-2 was A=51% (95% CI, .23–.61), C=1% (95% CI, .00–.23), E=48% (95% CI, .39–.58); and for psychopathic personality A=44% (95% CI, .17–.54), C=1% (95% CI, .00–.21), E=55% (95% CI, .46–.66)], (see Table 5).

Age-Specific Genetic and Environmental Correlations: Caregiver Reports [Youth Self-Reports]

At time-1, there was a significant genetic correlation between negative parent-to-child affect and psychopathic personality, $r_{g1}=0.74$ (95% CI, .40–1.00) [$r_{g1}=0.58$; 95% CI, .16–1.00], while non-shared environmental influences also showed a significant correlation $r_{e1}=0.35$ (95% CI, .25–.45) [$r_{e1}=0.24$; 95% CI, .14–.35] suggesting that the unique experiences contributing to parental negativity toward one child may also be influencing their psychopathic traits. The correlations at the later age reflect correlations between the residual effects at time-2, and thus, index the relationship between negative parent-to-child affect and psychopathic personality that is independent of their relationship at ages 9–10 years. At time-2, neither residual genetic nor residual shared environmental influences were correlated with one another, although there was a significant non-shared environmental correlation: $r_{e2}=0.45$ (95% CI, .32–.56) [$r_{e2}=0.26$; 95% CI, .11–.40]. Thus, any shared genetic etiology between psychopathic personality and parent-to-child affect at the later age is likely the result of the earlier association between these variables at ages 9–10 years, and any stability in these influences from childhood to later adolescence.

Cross-Age and Cross-lag Paths: Caregiver Reports [Youth Self-Reports]

The cross-lag estimates in Figure 1 are presented as partial regression coefficients, i.e., paths b_{11} , b_{22} , b_{12} , b_{21} , located in the center of the path diagram. All four partial regression paths were significant, suggesting both continuity in these variables across time (b_{11} and b_{22}) as well as a bidirectional relationship between negative parent-to-child affect and psychopathic personality. The partial regression coefficients estimate the strength of the longitudinal associations between any two measures when controlling for preexisting associations between negative parent-to-child affect and psychopathic personality at time-1. Specifically,

the *cross-age stability path* (b_{11}) between negative parent-to-child affect at time-1 and time-2 estimates the association between negative parent-to-child affect at the two time points when controlling for psychopathic personality at time-1. Similarly, the *cross-age stability path* (b_{22}) between psychopathic personality at time-1 and time-2 estimates the association between psychopathic personality when controlling for negative parent-to-child affect at time-1.

Further, the significance of both b_{12} and b_{21} is suggestive of the bidirectional relationship between negative parent-to-child affect and child psychopathic personality across time. The *cross-lagged path* (b_{21}) indicates that early negative parent-to-child affect at time-1 independently contributed to children's later psychopathic personality at time-2 while controlling for the relationship with psychopathic personality at time-1. Likewise, early psychopathic personality at time-1 independently contributed to later negative parent-to-child affect at time-2 (b_{12}).

Squaring these partial regression coefficients index the percentage of the total variance explained in each measure at the later age. Thus, the cross-age stability path from negative parent-to-child affect independently explained 14% (b_{11}^2) (95% CI, .29–.45) [0.16%; 95% CI, .00–.12] of the variance in negative parent-to-child affect at time-2. The cross-age stability path from psychopathic personality independently explained 21% (b_{22}^2) (95% CI, .39–.53) [8%; 95% CI, .20–.34] of the variance in psychopathic personality at time-2. Further, the cross-lag path from negative parent-to-child affect at time-1 independently explained 0.81% (b_{21}^2) (95% CI, .02–.17) [2%; 95% CI, .06–.23] of the variance in psychopathic personality at time-2. The cross-lag path from psychopathic personality at time-1 independently explained 1.96% (b_{12}^2) (95% CI, .06–.22) [0%; 95% CI, 0–.04] of the variance in negative parent-to-child affect at time-2.

Finally, the phenotypic cross-lag estimates in Figure 1, i.e., paths b_{11} , b_{22} , b_{12} , b_{21} and the biometrical parameters were used to decompose the variance of both time-2 variables into their genetic and environmental components, using the expectation equations provided in Table 1. The first section of Table 5 displays the total genetic, shared and non-shared environmental influences on *negative parent-to-child affect*, while the second section shows these relative effects for *child psychopathic traits*.

Explaining Parental Negativity at Time-2: Caregiver Reports [Youth Self-Reports]

Cross-Age Stability Effects—Genetic influences unique to negative parent-to-child affect explained 42% [0%] of the heritability in negative parent-to-child affect at ages 14–15 years (calculated as: $b_{11}^2 * a_1^2 = .03$, see Table 1 and divided by the total genetic variance of .07). Stable effects explained 13% [0%] of the shared environment and 11% [0%] of the non-shared environmental variance components in later parental negativity.

Cross-lag Effects—Genetic influences unique to early psychopathic personality explained 14% [2%] of the heritability in negative parent-to-child affect at ages 14–15 years ($b_{12}^2 * a_2^2 = .01$, see Table 1), reflecting the genetic basis of child-driven effects of early psychopathic personality in the children on later parental negativity during adolescence. These cross-lag effects also explained 2% [0%] of the shared and 2% [4%] of the non-shared environment components, respectively.

Common and Residual Effects—The remaining part of the heritability in negative parent-to-child affect at age 14–15 years was explained by genetic factors *common* to negative parent-to-child affect and psychopathic personality at ages 9–10 years ($2 * [b_{11} * a_1 * r_{g1} * a_2 * b_{21}]$, see Table 1) (29% [0%] of the total genetic variance in later parental negativity) and *specific* genetic effects (14%) [97%] of negative parent-to-child

affect at age 14–15 years. *Common* effects (between the two measures at time-1) explained none of the shared environment [0%] (due to the zero estimate for shared environmental variance in time-1 CPS) and only 2% [0%] of the non-shared environmental components of parental negativity at time-2. Residual effects (specific to time-2) explained the majority (85%) [100%] of both the shared and non-shared environment (85%) [96%], respectively.

Explaining Psychopathic Personality at Time-2: Caregiver Reports [Youth Self-Reports]

The second section of Table 5 displays the relative effects of stable, cross-lagged, common and residual (time-2 specific) effects on the genetic, shared and non-shared environmental influences on *psychopathic personality*.

Cross-Age Stability Effect—Genetic influences unique to psychopathic personality explained 27% [5%] of the heritability in psychopathic personality at ages 14–15 years. These stable effects explained 18% [8%] of the non-shared environmental effects in children's later psychopathic personality. Given that no shared environmental effects in psychopathic personality were found at either time-1, there is correspondingly no explanation of the time-2 variance by the stable effects of early psychopathic personality.

Cross-lag Effects—Genetic influences on early negative parent-to-child affect explained 1% [0%] of the heritability in children's later psychopathic personality at ages 14–15 years, indicating a trivial influence of children's genetic differences on these 'parent-driven' effects. These cross-lag effects also explained correspondingly little or none of the shared environment (0%) [0%], and the non-shared environment (1%) [0%]. Thus, in spite of the significant overall parent-driven effect (b_{21}) in the biometrical cross-lag model, these effects are quite small especially in comparison to the child-driven effects. Still, it is worth noting that the cross-lagged effects of early parenting on later child behavior appear to be split evenly between genetic and (non-shared) environmental sources.

Common and Residual Effects—The remaining part of the heritability in psychopathic personality at age 14–15 years was explained by genetic factors *common* to psychopathic personality and negative parent-to-child affect at ages 9–10 years (7%) [2%] and *specific* genetic effects (65%) [93%] of psychopathy at ages 14–15 years. Common effects explained none of the shared environmental variance in later parental negativity (again, due to the zero estimate of shared environment for CPS). In fact, the shared environment for later parental negativity was entirely explained by the residual (time-2 specific) effects. *Common* shared environment and residual shared environment explained 0% [0%] and 100% [100%], respectively. The non-shared environment for adolescent psychopathic personality was almost entirely explained by the time-2 specific (residual) effects (78%) [91%], with a small contribution from the common effects between early parental negativity and child psychopathic personality (3%) [1%].

Discussion

The purpose of the present study was to examine the direction and the genetic and environmental etiology of the association between negative parent-to-child affect and psychopathic personality from ages 9–10 years to 14–15 years. Specifically, we examined (1) whether the association between parenting and child behavior across time was driven by parental effects (i.e., early parenting affecting later child behavior), child effects (early child behavior affecting later parenting), or both; and (2) the extent to which genetic and environmental influences contribute to these associations. Utilizing both caregiver and youth self-report measures of parent-to-child negative affect and children's psychopathic personality, two main conclusions were drawn from the results. First, significant parent-

driven effects were found, based on caregiver and youth self-reports: negative parent-to-child affect at ages 9–10 years influenced psychopathic personality at ages 14–15 years, independently of early heritable child psychopathic personality. Second, significant child driven effects were also found. Based on caregiver reports, psychopathic personality at age 9–10 years influenced negative parent-to-child affect at age 14–15 years. Thus in part, children's genetically influenced psychopathic personality seemed to evoke parental negativity at ages 14–15 years, which lends support to our expected direction of effects and hypothesis.

Consistent with previous literature, our results demonstrated that both genetic and shared environmental influences were significant in caregiver reported negative parent-to-child affect (Larsson et al., 2008; Neiderhiser et al., 1999). In contrast, the variance in youth self-reports on negative parent-to-child affect was primarily explained by genetic and non-shared environmental factors, with shared environmental factors being of minimal importance. The greater and significant effect of shared environment in caregiver reports of parent-to-child affect could be the result, at least in part, of rater bias since the same caregiver completed ratings of both twins. Similar to other studies examining the direction and genetic and environmental etiology of parenting and antisocial behavior (Burt et al., 2005; Larsson et al., 2008; Neiderhiser et al., 1999), we did not find any significant sex differences in the genetic and environmental variance components.

For both caregiver and youth self-reports at both time points, about half of the variance in psychopathic personality was explained by genetic factors, with the majority of the remaining variance being explained by non-shared environmental influences. These results fall in line with previous twin studies examining psychopathic personality in childhood (Bezdjian et al., 2010) and adolescence (Blonigen et al., 2005).

Furthermore, our results showed a significant genetic overlap between negative parent-to-child affect and psychopathic personality at ages 9–10 years (caregiver, $r_g=0.74$; youth self-report, $r_g=0.58$). Significant non-shared environmental overlap was also found at both time points. It should be noted that the non-shared environmental correlation (r_e) could also reflect correlated measurement error between the two measures.

Moderate stability was observed in negative parent-to-child affect across the 5-year time lag. Similar to previous studies, the cross-age stability path independently explained 14% of the variance in negative parent-to-child affect at ages 14–15 years for caregiver reports (Burt et al., 2005; Larsson et al., 2008). For youth self-reports the stability was of minimal importance with respect to the total variance in negative parent-to-child affect at ages 14–15 years.

In line with previous findings (Frick et al., 2003; Muñoz & Frick, 2007), the stability in psychopathic personality was slightly stronger in caregiver ratings than youth self-reports from ages 9–10 to 14–15. The cross-age stability path independently explained 21% of the variance in psychopathic personality at ages 14–15 years. For youth self-reports the cross-age stability path independently explained approximately 8% of the variance in psychopathic personality at ages 14–15 years.

Our results further demonstrated a significant bidirectional relationship between negative parent-to-child affect and psychopathic personality across time. Negative parent-to-child affect at ages 9–10 years independently contributed to psychopathic personality at ages 14–15 years, even after taking the stability in psychopathic personality into account (caregiver, 0.81%; youth self-report, 2%). These findings emphasize the importance of *parent-driven* effects in the relationship over time between parenting and psychopathic personality. Thus, findings from the present study confirm that negative parenting contributes to the

development of psychopathic personality (Patrick, 2005), although the effect sizes are small. Other factors that are likely to impact the development of psychopathy include genetic influences (Bezdjian et al., 2010; Blonigen et al., 2005) and deficits in neurocognitive and affective-emotional processing (Gao, Glenn, Schug, Yang, & A., 2009). Psychopathic individuals have also been found to show reduced prefrontal structure and function, including the right orbitofrontal cortex, right anterior cingulate cortex, and left dorsolateral prefrontal cortex (Gao et al., 2009). Critical functions of the amygdala have been found to be compromised in individuals scoring high on psychopathic traits (Blair, 2008). Regardless, individual differences in psychopathic traits within this young sample are more likely driven by normal variation in personality traits than by any abnormalities or neurological insults.

Likewise, psychopathic personality at age 9–10 years independently contributed to negative parent-to-child affect at age 14–15 years, but only in caregiver reports (1.96%). Thus, *child-driven* effects appear to be of some importance in the relationship over time between psychopathic personality and negative parent-to-child affect. Interestingly, this was only true for the caregivers' ratings, as no significant relationship was found for youth self-reports. Differences in findings between informants also stress the importance of utilizing both caregiver and self-report data when trying to understand complex processes. Although caregivers may not always be privy to their child's behaviors or motives or even whereabouts, they may be able to understand difficult and complex constructs better than children can (Bezdjian et al., 2011). In contrast, self-reporters are privy to their own behaviors and motives. As children get older, they may become better at masking their psychopathic behaviors; thus, yielding a unique set of results from each informant.

The bidirectional effects found in the present study may seem small, but are well in line with what others have reported between antisocial behavior and parenting style (Burt et al., 2005; Larsson et al., 2008). The few previous studies that have investigated parenting style and psychopathic personality have shown that parenting style has an important influence on the development of psychopathic personality, suggesting the importance of parent-driven effects (Gao et al., 2010; Lang et al., 2002; Marshall & Cooke, 1999). However, the findings in the present study emphasize the importance of bidirectional effects, i.e., both child-driven and parent-driven effects in the relationship between psychopathic personality and negative parent-to-child affect. These bidirectional effects suggest that parenting both influence and is influenced by child behavior. Related to this is the fact that antisocial behavior tends to cluster in families; however, the reason for this is not very well understood. The family concentration of antisocial behavior could be explained by genetic influences on antisocial behavior, but it may also be explained by non-genetic social or cultural transmission across generations. It has been suggested that this intergenerational transmission of antisocial behavior is explained by the fact that antisocial behavior is learned (Bandura, 1973), children learn and perform antisocially simply by watching a role model behave in antisocial ways. However, psychopathic personality is highly heritable and parents and children share part of the genetic make-up (~50%). Findings from the current study indicates that there is a reciprocal relationship between parents and a child, specifically how these processes evolve over time and across generations needs to be further examined.

Based on caregiver reports negative parent-to-child affect at ages 9–10 years independently explained 1% of the genetic influences and 1% of the non-shared environmental influences in psychopathic personality at ages 14–15 years. Thus, negative parent-to-child affect contributed to children's psychopathic personality through both genetic and non-shared environmental mechanisms; although, the effects, albeit significant were quite small in comparison to other sources. In contrast, psychopathic personality at ages 9–10 years independently explained 14% of the genetic influences in negative parent-to-child affect at ages 14–15 years. This finding indicates that the child's psychopathic personality—which is

in part genetically influenced—appears to evoke parental negativity at ages 14–15 years. This finding differs from previous results in which Burt et al., and Larsson et al., reported a much lower genetic influence, 1.75% and 3.1% respectively. In addition, psychopathic personality at ages 9–10 years independently explained 2% of the shared environmental influences and 2% of the non-shared environmental influences in negative parent-to-child affect at ages 14–15 years.

Based on youth self-reports, negative parent-to-child affect at ages 9–10 years independently explained 2% of the genetic influences in psychopathic personality at ages 14–15 years and 4% of the non-shared environmental influences. Negative parent-to-child affect contributed to the children's psychopathic personality through genetic and non-shared environmental mechanisms.

Our findings indicate that regardless of the child's heritability of psychopathic personality, negative parent-to-child affect may contribute to the child's psychopathic personality. At the same time, earlier heritable psychopathic personality seems to be influencing parenting style. These results stress the importance of taking child factors into account when investigating parenting style on the development of psychopathic personality. This lends further support to the importance of considering heritable influences in the development of psychopathology. In addition, psychopathic traits (e.g., callous and unemotional traits) can be identified early on in development (Viding et al., 2005), suggesting that findings from the current study should be considered when designing prevention strategies in form of parental training and guidance.

Limitations

The results of this study are subject to several potential limitations. First, when using a bidirectional effects model it is not possible to distinguish between passive vs. evocative/reactive gene-environment correlations (r_{GE}) (Scarr & McCartney, 1983). Passive r_{GE} occurs mainly because of genetic overlap between parents and their children. For example, children of aggressive parents inherit genetic susceptibility for aggression as well as experience an adverse rearing environment. Passive-gene-environment correlation for mother's positivity and monitoring toward her child was recently reported by (Neiderhiser et al., 2004). Evocative/reactive r_{GE} occurs when a child's characteristics elicit a particular response from the environment. This occurs when the parents are responding to a genetically influenced trait or behavior in the child. Previous adoption studies on antisocial behavior have shown that aggressive children tend to elicit more negative environments from their parents (Ge et al., 1996; O'Connor et al., 1998).

Second, there are several assumptions related to the classical twin design, which possibly may not have been met. For example, if the equal environment assumption is not met, then higher correlations among MZ twins may be due to environmental factors, rather than genetic factors and heritability estimate may be overestimated. Also, it is assumed that there is no interaction between genetic and environmental influences. For a review on these and other assumption in the classical twin design and whether they apply to psychopathological disorders and behaviors, see (Tuvblad & Baker, 2011). In addition, twins have been found not to differ from singletons on rates of psychopathology (including ODD, CD, and antisocial behavior), cognitive ability, academic performance, and brain development (Barnes & Boutwell, 2013; Hjern, Ekeus, Rasmussen, & Lindblad, 2012; Moilanen et al., 1999; Ordaz et al., 2010). This suggests that findings from twin samples may be generalized to the general population.

Third, the negative-parent-to-child affect scale (parent ratings, youth self-report) had low internal consistency based on Cronbach's alphas. However, this is most likely explained by

the fact that each scale contained a low number of items (5 items each). Nevertheless, the negative-parent-to-child affect scale comes from a well validated measure (Deater-Deckard, 1996).

Fourth, the present study only utilized data from two time points, as such, certain longitudinal models could not be utilized (e.g., latent growth curve model). In addition, these effects were observed during pre-adolescence and early adolescence. It is possible that the strength of these bidirectional relationships and the heritability of these constructs (and the parent-child dynamic) would be different at early ages when these child behaviors are emerging and at later ages when they are more canalized. Future studies should examine the longitudinal relationships between parenting and psychopathic personality.

Conclusions

In summary, the results from this study support the notion that the association between negative parenting and psychopathic personality in pre- and mid-adolescent twins was due to bidirectional effects (i.e., the relationship between negative parent-to-child affect and psychopathic personality is due to both parent-driven and child-driven effects). Furthermore, we found support for the hypothesis that children's genetically influenced psychopathic personality evoked negative parenting during adolescence. To better understand the underlying relationship between negative parenting (style) and the development of psychopathic personality throughout the course of adolescence, genetic and environmental interactions between negative parenting and psychopathic personality need to be further explored.

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References

- Akaike AC. Factor analysis and AIC. *Psychometrika*. 1987; 52:317–332.
- Baker L, Tuvblad C, Wang P, Gomez K, Raine A. The Southern California Twin Register at the University of Southern California: III. Twin Research and Human Genetics. 2013; 16(1):336–343. [PubMed: 23394193]
- Baker LA, Barton M, Lozano DI, Raine A, Fowler JH. The Southern California Twin Register at the University of Southern California: II. Twin Research and Human Genetics. 2006; 9:933–940. [PubMed: 17254433]
- Bandura, A. *Aggression: a social learning theory analysis*. New York: Prentice-Hall; 1973.
- Barnes JC, Boutwell BB. A Demonstration of the Generalizability of Twin-based Research on Antisocial Behavior. *Behavior Genetics*. 2013; 43(2):120–131. [PubMed: 23274656]
- Beaver K, Barnes JC, May J, Schwarz JA. Psychopathic Personality Traits, Genetic Risk, and Gene-Environment Correlations. *Criminal Justice and Behavior*. 2011; 38(9):896–912.
- Bezdjian S, Raine A, Baker LA, Lynam DR. Psychopathic personality in children: genetic and environmental contributions. *Psychological Medicine*. 2010; 41(3):589–600. [PubMed: 20482945]
- Bezdjian S, Tuvblad C, Raine A, Baker LA. The genetic and environmental covariation among psychopathic personality traits, and reactive and proactive aggression in childhood. *Child Development*. 2011; 82(4):1267–1281. [PubMed: 21557742]
- Blair RJ. Applying a cognitive neuroscience perspective to the disorder of psychopathy. *Development and Psychopathology*. 2005; 17(3):865–891. [PubMed: 16262996]
- Blair RJ. The amygdala and ventromedial prefrontal cortex: functional contributions and dysfunction in psychopathy. *Philosophical Transactions of the Royal Society B: Biological Sciences*. 2008; 12(363 (1503)):2557–2565.

- Blonigen DM, Hicks BM, Krueger RF, Patrick CJ, Iacono WG. Psychopathic personality traits: heritability and genetic overlap with internalizing and externalizing psychopathology. *Psychological Medicine*. 2005; 35:637–648. [PubMed: 15918340]
- Burt AS, McGue M, Krueger R, Iacono WG. How are parent-child conflict and childhood externalizing symptoms related over time? Results from a genetically informative cross-lagged study. *Development and Psychopathology*. 2005; 17(1):145–165. [PubMed: 15971764]
- Collins WA, Maccoby EE, Steinberg L, Hetherington EM, Bornstein MH. Contemporary research on parenting: The case for nature and nurture. *American Psychologist*. 2000; 55:218–232. [PubMed: 10717969]
- Deater-Deckard, K. *The Parent Feelings Questionnaire*. London: Institute of Psychiatry; 1996.
- Frick PJ, Kimonis ER, Dandreaux DM, Farell JM. The 4 year stability of psychopathic traits in non-referred youth. *Behavioral Science and the Law*. 2003; 21(6):713–736.
- Gao Y, Glenn AL, Schug RA, Yang Y, A R. The neurobiology of psychopathy: a neurodevelopmental perspective. *Canadian Journal of Psychiatry*. 2009; 54(12):813–823.
- Gao Y, Raine A, Chan F, Venables PH, Mednick SA. Early maternal and paternal bonding, childhood physical abuse and adult psychopathic personality. *Psychological Medicine*. 2010; 40(6):1007–1016. [PubMed: 20441692]
- Ge X, Conger RD, Cadoret RJ, Neiderhiser JM, Yates WR, Troughton E, et al. The developmental interface between nature and nurture: A mutual influence model of child antisocial behavior and parent behaviors. *Developmental Psychology*. 1996; 32(4):574–589.
- Hare, RD. *Manual for the Hare Psychopathy Checklist-Revised*. North Tonawanda, NY: Multi-Health System; 1991.
- Hare RD. Psychopathy: a clinical construct whose time has come. *Criminal Justice and Behavior*. 1996; 23:25–54.
- Hare, RD. Psychopathy and risk for recidivism and violence. In: Gray, J.; Laing, J.; Noaks, L., editors. *Criminal Justice, Mental Health, and Politics of Risk*. London: Cavendish Publishing; 2002. p. 27-47.
- Hare, RD. *The Hare Psychopathy Checklist-Revised (PCL-R)*. 2nd edition. Toronto, Ontario, Canada: Multi-Health Systems; 2003.
- Hjern A, Ekeus C, Rasmussen F, Lindblad F. Educational achievement and vocational career in twins a Swedish national cohort study. *Acta Paediatrica*, epub ahead of print. 2012
- Lang S, af Klinteberg B, Alm PO. Adult psychopathy and violent behavior in males with early neglect and abuse. *Acta Psychiatry Scandinavia Supplement*. 2002; 412:93–100.
- Larsson H, Viding E, Rijdsdijk F, Plomin R. Relationships between parental negativity and childhood antisocial behavior over time: a bidirectional effects model in a longitudinal genetically informative design. *Journal of Abnormal Child Psychology*. 2008; 36(5):633–645. [PubMed: 17602294]
- Lynam DR. Pursuing the psychopath: capturing the fledgling psychopath in a nomological net. *Journal of Abnormal Psychology*. 1997; 106(3):425–438. [PubMed: 9241944]
- Lynam DR, Caspi A, Moffitt TE, Raine A, Loeber R, Stouthamer-Loeber M. Adolescent psychopathy and the big five: results from two samples. *Journal of Abnormal Child Psychology*. 2005; 33(4): 431–443. [PubMed: 16118990]
- Maccoby EE. Parenting and its effects on children: On reading and misreading behavior genetics. *Annual Review of Psychology*. 2000; 51:1–27.
- Marshall LA, Cooke DJ. The childhood experiences of psychopaths: a retrospective study of familial and societal factors. *Journal of Personality*. 1999; 13(3):211–225.
- Moffitt TE. The new look of behavioral genetics in developmental psychopathology: gene-environment interplay in antisocial behaviors. *Psychological Bulletin*. 2005; 131(4):533–554. [PubMed: 16060801]
- Moilanen I, Linna S-L, Kumpulainen K, Tamminen K, Piha J, Almqvist F. Are twins' behavioural/emotional problems different from singletons'? *European Child and Adolescent psychiatry*. 1999; 8:62–67. [PubMed: 10654135]

- Muñoz LC, Frick PJ. The reliability, stability, and predictive utility of the self-report version of the Antisocial Process Screening Device. *Scandinavian Journal of Psychology*. 2007; 48(4):299–312. [PubMed: 17669220]
- Neale, MC.; Boker, SM.; Xie, G.; Maes, H. Mx: Statistical modeling. Sixth ed. Richmond, VA: Department of Psychiatry, Medical College of Virginia; 2003.
- Neiderhiser JM, Reiss D, Hetherington EM, Plomin R. Relationships between parenting and adolescent adjustment over time: genetic and environmental contributions. *Developmental Psychology*. 1999; 35(3):680–692. [PubMed: 10380859]
- Neiderhiser JM, Reiss D, Pedersen NL, Lichtenstein P, Spotts EL, Hansson K, et al. Genetic and environmental influences on mothering of adolescents: a comparison of two samples. *Developmental Psychology*. 2004; 40(3):335–351. [PubMed: 15122961]
- Neumann CS, Hare RD. Psychopathic traits in a large community sample: Links to violence, alcohol use intelligence. *Journal of Consulting and Clinical Psychology*. 2008; 76(5):893–899. [PubMed: 18837606]
- O'Connor TG, Deater-Deckard K, Fulker D, Rutter M, Plomin R. Genotype-environment correlations in late childhood and early adolescence: antisocial behavioral problems and coercive parenting. *Developmental Psychology*. 1998; 34:970–981. [PubMed: 9779743]
- Ordaz SJ, Lenroot RK, Wallace GL, Clasen LS, Blumenthal JD, Schmitt JE, et al. Are there differences in brain morphometry between twins and unrelated singletons? A pediatric MRI study. *Genes, Brains and Behavior*. 2010; 9(3):288–295.
- Patrick, CJ. *Handbook of Psychopathy*. Guilford Press; 2005.
- Raftery AE. Bayesian model selection in social research. *Sociological Methodology*. 1995; 25:111–163.
- SAS. SAS/STAT software: changes and enhancements through release 9.2. Cary, NC: SAS Institute Inc.; 2005.
- Scarr S, McCartney K. How people make their own environments: A theory of genotype-environment effects. *Child Development*. 1983; 54:425–435.
- Tuvblad, C.; Baker, LA. Human Aggression across the Lifespan: Genetic Propensities and Environmental Moderators. In: Huber, R.; Brennan, P.; Bannasch, D., editors. Vol. 75 of *Advances in Genetics (Aggression)*. Vol. Vol. 75. 2011. p. 171-214.
- Viding E, Blair JR, Moffitt TE, Plomin R. Evidence of substantial genetic risk for psychopathy in 7-year-olds. *Journal of Child Psychology and Psychiatry*. 2005; 46(6):592–597. [PubMed: 15877765]
- Vitacco MJ, Neumann CS, Ramos V, Roberts MK. Ineffective parenting: a precursor to psychopathic traits and delinquency in Hispanic females. *Annals of the New York Academy of Sciences*. 2003; 1008:300–303. [PubMed: 14998901]

Highlights

- Negative parenting at ages 9–10 influenced child's psychopathic personality at ages 14–15
- These effects were independent of early heritable child psychopathic personality
- Children's genetically influenced psychopathic personality seemed to evoke parental negativity at ages 14–15

Figure 1.

Caregiver Reports (Youth Self-Reports in parentheses), Cross-lag model Psychopathic Personality (CPS, Child Psychopathy Scale) and Negative Parent-to-Child Affect. Significant path estimates are denoted with an asterisk *.

Table 1

Equations Used in the Cross-lag Model

	A	C	E	Total
Negative Parent-to-Child Affect at Ages 14–15 Years				
Negative parent-to-child affect (b_{11})	$(b_{11}^2 * a_1^2)$	$(b_{11}^2 * c_1^2)$	$(b_{11}^2 * e_1^2)$	$(b_{11}^2 * a_1^2) + (b_{11}^2 * c_1^2) + (b_{11}^2 * e_1^2)$
Cross-Age (stability) parameter (time-1 to time-2)				
Psychopathic Personality (b_{12})	$(b_{12}^2 * a_2^2)$	$(b_{12}^2 * c_2^2)$	$(b_{12}^2 * e_2^2)$	$(b_{12}^2 * a_2^2) + (b_{12}^2 * c_2^2) + (b_{12}^2 * e_2^2)$
Cross-lag parameter (<i>child driven effect</i> : child's psychopathic traits negative parent-to-child affect)				
Common effects at time-1 (9–10 yr)	$(2 * [b_{11} * a_1 * rg_1 * a_2 * b_{21}])$	$(2 * [b_{11} * c_1 * rc_1 * c_2 * b_{21}])$	$(2 * [b_{11} * e_1 * re_1 * e_2 * b_{21}])$	$(2 * [b_{11} * a_1 * rg_1 * a_2 * b_{21}]) + (2 * [b_{11} * c_1 * rc_1 * c_2 * b_{21}]) + (2 * [b_{11} * e_1 * re_1 * e_2 * b_{21}])$
Residual (specific) effects at time-2 (14–15 yr)	a_3^2	c_3^2	e_3^2	$a_3^2 + c_3^2 + e_3^2$
Total A, C, or E components of negative parent-to-child affect at time-2 (14–15 yr)				1.00(100%)
Psychopathic Personality at Ages 14–15 Years				
Psychopathic personality (b_{22})	$(b_{22}^2 * a_2^2)$	$(b_{22}^2 * c_2^2)$	$(b_{22}^2 * e_2^2)$	$(b_{22}^2 * a_2^2) + (b_{22}^2 * c_2^2) + (b_{22}^2 * e_2^2)$
Cross-Age (stability) parameter (time-1 to time-2)				
Negative parent-to-child affect (b_{21})	$(b_{21}^2 * a_1^2)$	$(b_{21}^2 * c_1^2)$	$(b_{21}^2 * e_1^2)$	$(b_{21}^2 * a_1^2) + (b_{21}^2 * c_1^2) + (b_{21}^2 * e_1^2)$
Cross-lag parameter (<i>parent-driven effect</i> : parent's negative parent-to-child affect psychopathic personality)				
Common effects at time-1 (9–10 yr)	$(2 * [b_{22} * a_2 * rg_1 * a_1 * b_{12}])$	$(2 * [b_{22} * c_2 * rc_1 * c_1 * b_{12}])$	$(2 * [b_{22} * e_2 * re_1 * e_1 * b_{12}])$	$(2 * [b_{22} * a_2 * rg_1 * a_1 * b_{12}]) + (2 * [b_{22} * c_2 * rc_1 * c_1 * b_{12}]) + (2 * [b_{22} * e_2 * re_1 * e_1 * b_{12}])$
Residual (specific) effects at time-2 (14–15 yr)	a_4^2	c_4^2	e_4^2	$a_4^2 + c_4^2 + e_4^2$
Total A, C, or E components of psychopathic personality at time-2 (14–15 yr)				1.00(100%)

Note. A=additive genetic influences, C=shared environmental influences, E=non-shared environmental influences

Table 2

Means, Standard Deviations, and Number of Participants (n) for Psychopathic Personality and Negative Parent-to-Child Affect at Ages 9–10 and 14–15 Years, by Sex and Zygosity, Caregiver and Youth Self-Reports

	Males			Females		
	Means (Standard Deviations)			Means (Standard Deviations)		
	MZ	DZ	OS	MZ	DZ	OS
Caregiver Report						
Psychopathic Personality 9–10 years	13.42(7.33) n=272	15.34(7.17) n=172	14.44(7.29) n=147	11.91(6.00) n=288	12.19(6.93) n=184	11.95(6.98) n=147
Psychopathic Personality 14–15 years	12.23(7.50) n=223	13.51(7.93) n=169	13.26(8.28) n=137	10.42(6.63) n=216	11.29(7.77) n=200	11.46(6.99) n=137
Negative Parent-to-Child Affect 9–10 years	2.27(.57) n=272	2.42(.56) n=172	2.37(.66) n=147	2.29(.62) n=288	2.33(.59) n=184	2.27(.59) n=147
Negative Parent-to-Child Affect 14–15 years	2.41(.54) n=168	2.50(.57) n=131	2.52(.67) n=113	2.35(.62) n=188	2.33(.56) n=163	2.35(.58) n=113
Youth Self-Report						
Psychopathic Personality 9–10 years	13.90(5.87) n=268	14.16(6.33) n=172	14.44(6.52) n=146	12.39(5.34) n=278	12.13(5.63) n=184	12.44(5.78) n=144
Psychopathic Personality 14–15 years	16.04(7.07) n=220	17.33(7.06) n=166	16.09(7.45) n=132	14.86(7.55) n=207	15.84(6.73) n=195	15.22(7.42) n=132
Negative Parent-to-Child Affect 9–10 years	2.24(.71) n=263	2.20(.70) n=172	2.31(.75) n=146	2.21(.72) n=282	2.14(.75) n=183	2.18(.80) n=147
Negative Parent-to-Child Affect 14–15 years	2.44(.64) n=166	2.73(.68) n=132	2.69(.68) n=105	2.46(.77) n=187	2.66(.77) n=155	2.65(.68) n=104

Note. MZ=monozygotic, DZ=dizygotic, OS=opposite sex, n=number of participants, untransformed data

Phenotypic Correlations for Psychopathic Personality and Negative Parent-to-Child at Ages 9–10 and 14–15 Years, Caregiver and Youth Self-Reports

Table 3

	1	2	3	4	5	6	7	8
1. Caregiver Psychopathic Personality 9–10 years	1	.20*	.62*	.30*	.43*	.15*	.39*	.22*
2. Self-Report Psychopathic Personality 9–10 years	.26*	1	.16*	.27*	.14*	.34*	.05	.17*
3. Caregiver Psychopathic Personality 14–15 years	.46*	.20*	1	.43*	.36*	.12*	.54*	.35*
4. Self-Report Psychopathic Personality 14–15 years	.25*	.37*	.34*	1	.16*	.04	.34*	.43*
5. Caregiver Negative Affect 9–10 years	.41*	.06	.21*	.12*	1	.13*	.50*	.21*
6. Self-Report Negative Affect 9–10 years	.18*	.30*	.12*	.10	.14*	1	.13*	.11
7. Caregiver Negative Affect 14–15 years	.33*	.17*	.44*	.19*	.48*	.16*	1	.32*
8. Self-Report Negative Affect 14–15 years	.14*	.22*	.24*	.25*	.18*	.10	.26*	1

Note. $p < .05$, untransformed data; estimates for boys are on the below diagonal and girls above the diagonal, cross-informant correlations are bolded

Table 4

Multivariate Model Fit Indices, Caregiver and Youth Self-Reports

Model #	Overall fit				Model difference test			
	-2LL	Df	AIC	BIC	χ^2	df	p	χ^2
Caregiver								
1 Saturated (means constrained)	10545.88	4096	2353.88	-8367.92	--	--	--	--
2 Cross – lag (males females)	10723.22	4232	2259.22	-8732.17	177.34	136	0.010	--
3 Cross – lag (males=females)	10740.52	4254	2232.52	-8796.78	194.64	158	0.025	17.30
Youth Self-Report								
1 Saturated (means constrained)	10977.29	3943	3091.29	-7637.62	--	--	--	--
2 Cross – lag (males females)	11144.25	4079	2986.25	-8006.89	166.96	136	0.037	--
3 Cross – lag (males=females)	11164.25	4101	2962.25	-8070.12	186.96	158	0.057	20.00
								22
								.58

Note. -2LL=-2(log-likelihood); AIC=Akaike's Information Criterion; BIC=Bayesian Information Criterion; χ^2 =difference in log-likelihoods between nested models; df=change in degrees of freedom.

Table 5

Squared, Standardized Estimates: Caregiver Ratings [Youth Self-Reports]

	A	C	E	Total
Negative Parent-to-Child Affect at Ages 14–15 Years				
Negative Parent-to-Child Affect (b_{11})	.03 (42%) [0]	.06 (13%) [0]	.05 (11%) [0]	
Psychopathic Personality (b_{12})	.01 (14%) [.01 (2%)]	.01 (2%) [0]	.01 (2%) [.02 (4%)]	
Common Ages 9–10 Effects	.02 (29%) [0]	.00 (.00%) [0]	.01 (2%) [0]	
Residual (Specific) Effects	.01 (14%) [.495 (97%)]	.38 (85%) [.01 (100%)]	.40 (85%) [.46 (96%)]	.79 (79%) [.96 (96%)]
Total ACE Components of Negative Parent-to-Child Affect at Ages 14–15 Years	.07 [.51]	.45 [.01]	.47 [.48]	1.00 (100%) [1.00 (100%)]
Psychopathic Personality at Ages 14–15 Years				
Stable Effects of Psychopathic Personality (b_{22})	.115 (27%) [.025 (5%)]	.00 [0]	.09 (18%) [.047 (8%)]	
Negative Parent to Child Affect (b_{21})	.005 (1%) [0]	.00 [0]	.005 (1%) [0]	
Common Ages 9–10 Effects	.03 (7%) [.01 (2%)]	.00 [0]	.015 (3%) [.01 (1%)]	
Residual (Specific) Effects	.27 (65%) [.41 (93%)]	.08 (100%) [.01 (100%)]	.39 (78%) [.50 (91%)]	.74 (74%) [.91 (91%)]
Total ACE Components of Psychopathic Personality at Ages 14–15 years	.42 [.44]	.08 [.01]	.50 [.55]	1.00 (100%) [1.00 (100%)]

Note. b_{12} denotes child-driven; b_{21} denotes parent-driven, please also see Table 1, A=genetic influences; C=shared environmental influences; E=non-shared environmental influences. The sum of effects may sum slightly above or below 1.0 due to rounding of decimals. Youth Self-Reports in brackets.