Published in final edited form as:

Child Dev. 2011 July ; 82(4): 1267–1281. doi:10.1111/j.1467-8624.2011.01598.x.

The Genetic and Environmental Covariation Among Psychopathic Personality Traits, and Reactive and Proactive Aggression in Childhood

Serena Bezdjian,

Washington University School of Medicine

Adrian Raine,

University of Pennsylvania

Catherine Tuvblad, and

University of Southern California

Laura A. Baker

University of Southern California

Abstract

The present study investigated the genetic and environmental *covariance* between psychopathic personality traits with reactive and proactive aggression in 9- to 10-year-old twins ($N = 1,219$). Psychopathic personality traits were assessed with the Child Psychopathy Scale (D. R. Lynam, 1997), while aggressive behaviors were assessed using the Reactive Proactive Questionnaire (A. Raine et al., 2006). Significant common genetic influences were found to be shared by psychopathic personality traits and aggressive behaviors using both caregiver (mainly mother) and child self-reports. Significant genetic and nonshared environmental influences specific to psychopathic personality traits and reactive and proactive aggression were also found, suggesting etiological independence among these phenotypes. Additionally, the genetic relation between psychopathic personality traits and aggression was significantly stronger for proactive than reactive aggression when using child self-reports.

A significant link between psychopathy and aggressive behaviors has been established in antisocial youths and adults (Hare & Jutai, 1983; Harpur & Hare, 1994; Porter & Woodworth, 2006). During adulthood, psychopaths are charged with violent crimes twice as often as nonpsychopathic offenders (Hare & Jutai, 1983). Meta-analyses have demonstrated that psychopathy has an overall effect size of $r_s = .27-.37$ in predicting violence or violent behaviors (Hemphill, Templeman, Wong, & Hare, 1998; Salekin, Rogers, & Sewell, 1996).

There is growing evidence that psychopathic personality traits are related to aggression much earlier in life. Precursors to psychopathy have been said to emerge in early childhood in the form of “callous–unemotional” traits (Lynam, 1997, 2002), which closely resemble adult psychopathic personality traits (particularly Revised Psychopathy Checklist [PCL-R] Factor 1; Hare, 1991). These characteristics or traits are usually associated with aggressive and violent behavior (Dodge, 1991; Fite, Stoppelbein, & Greening, 2009; Frick, 1998; Lynam, 1997, 2002). Comprehension and greater understanding of the link between

psychopathy and aggression could greatly benefit and aid in the prediction of future violence by adult offenders (Salekin et al., 1996).

Aggressive individuals who are said to be *reactive* in nature have been described as having high emotional arousal, difficulties with impulse control, and an inability to regulate or control their emotions or affect (Dodge & Coie, 1987). Proactive aggression, on the other hand, has been described as an organized and instrumental form of aggression, with little evidence of autonomic arousal (Berkowitz, 1993; Dodge, 1991). Although both reactive and proactive aggression are statistically related constructs (Dodge & Coie, 1987) and often co-occur and correlate at about $r = .70$ (Vitaro & Brendgen, 2005), evidence also suggests that reactive and proactive aggression may be separate and distinct types of aggression (Crick & Dodge, 1996; Dodge & Coie, 1987; Fite, Stoppelbein, Greening, & Gaertner, 2009) with unique correlates to both types of aggression. For example, reactive aggression is related to peer rejection and victimization (Lamarche et al., 2007; Salmivalli & Helteenvuori, 2007; Schwartz et al., 1998), while proactive aggression is related to psychopathic personality traits (Raine et al., 2006), and later delinquent behavior (Brendgen, Vitaro, Tremblay, & Lavoie, 2001; Pulkkinen, 1996; Vitaro, Gendreau, Tremblay, & Olinny, 1998). In addition, both exploratory and confirmatory factor analyses have shown that reactive and proactive aggression are factorially distinct (Crick & Dodge, 1996; Poulin & Boivin, 2000; Raine et al., 2006; Salmivalli & Nieminen, 2002).

Criminals identified as committing proactive violent offenses have higher scores on the PCL than those with a history or background of reactive aggression or violence (Cornell et al., 1996). Some have argued that psychopathy is more associated with proactive rather than reactive aggression (Blair, 2001, 2007; Patrick & Zempolich, 1998) and even early in life, Frick, Cornell, Barry, Bodin, and Dane (2003) have found that children with callous–unemotional traits have higher proactive aggression scores (Frick et al., 2003). Along similar lines, Raine et al. (2006) also found a strong association between psychopathic personality traits (as measured by the Child Psychopathy Scale [CPS]; Lynam, 1997) and residualized proactive aggression, but found no significant relation between psychopathy and reactive aggression. Accordingly, we would expect a stronger relation between psychopathic personality traits and proactive than reactive aggression in community samples of children. However, research has also established that psychopaths are impulsive and the most violent of offenders (Hare & Jutai, 1983; Harpur & Hare, 1994), suggesting that there may also be a significant relation between psychopathic personality traits and *reactive* aggression (Flight & Forth, 2007). Although phenotypic relations can be hypothesized for both reactive and proactive aggression with psychopathy, the etiological roots of either relation is not yet well understood.

Substantial estimates for the heritability of psychopathic personality traits in children and adolescents have been reported (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2006; Larsson, Andershed, & Lichtenstein, 2006; Taylor, Loney, Bobadilla, Iacono, & McGue, 2003; Viding, Blair, Moffitt, & Plomin, 2005), and reactive and proactive forms of aggression have also been found to be heritable (Baker, Raine, Lui, & Jacobson, 2008; Brendgen, Vitaro, Boivin, Dioone, & Perusse, 2006; Tuvblad, Raine, Zheng, & Baker, 2009). To the authors' knowledge no study has examined the underlying common genetic and environmental etiological covariation between psychopathic personality traits and reactive and proactive aggression in children. As both psychopathic and aggressive traits have been found to be heritable and covary with one another, we would expect to find significant genetic overlap among these constructs. Based on previous studies examining the relation between psychopathy and proactive behaviors, we hypothesized a genetic overlap between psychopathic personality traits and *proactive* aggression (Patrick & Zempolich, 1998). However, we would also expect a significant relation between psychopathic

personality traits and *reactive* aggression because of the fact that both constructs encompass difficulties in impulse control (Flight & Forth, 2007).

When examining violent and aggressive behaviors, it is critical to consider and understand the motivation of the violence—that is, whether the violent and aggressive act was “defensive,” meaning an immediate reaction to an emotional stimulus, or “offensive,” a planned and instrumental act (Berkowitz, 1983; Porter & Woodworth, 2006, p. 483). It is also possible that a violent act may be associated with both *reactive* and *proactive* aggression (Barratt, Stanford, Dowdy, Liebman, & Kent, 1999). Caregivers may not fully know or understand the motive behind their child’s aggressive act; therefore, (child) self-report data may be especially informative. Both caregiver and child self-report data of psychopathic personality traits and aggressive behaviors were examined in the present study, as both caregivers and their children know crucial and unique pieces of information regarding behaviors. Self-reporters are privy to their own motives and behaviors that may go undetected by their caregivers or other reporters, while caregivers may be able to understand difficult and complex constructs better than children can (Bartels et al., 2003); thus, both types of reporters are utilized in the current study. Predicting whether psychopaths will commit more reactive violence (as a result of poor behavioral controls) or more premeditated instrumental violence (because of a callous nature) is difficult (Porter & Woodworth, 2006). Thus, there is paucity in the literature and a need for a greater understanding of the relation between violent acts and aggressive behaviors using both caregiver and twin self-reports.

Previously in our sample, we have demonstrated that psychopathic personality traits measured using the CPS (Lynam, 1997) yield a two-factor solution: Factor 1 (callous & disinhibited) and Factor 2 (manipulative & deceitful). The present study investigates the genetic and environmental covariation of these two CPS factors with reactive and proactive aggression in 9- and 10-year-old twins. The purpose of the current study was to expand the existing literature on psychopathic personality traits and aggressive behaviors, and examine the genetic and environmental overlap between the two dimensions of psychopathic personality traits (callous & disinhibited and manipulative–deceitful) and reactive and proactive aggression employing both child self-report and caregiver reports of these traits. Specifically, we expect to find a genetic overlap between psychopathic personality traits and *proactive* aggression (Patrick & Zempolich, 1998). We also expect a significant relation between psychopathic personality traits and *reactive* aggression to emerge because of the fact that both constructs encompass difficulties in impulse control (Flight & Forth, 2007).

Method

Participants

The sample was composed of participants from the Southern California Twin Project at the University of Southern California (USC). Complete information regarding the design and procedures of the project has been provided in Baker, Barton, Lozano, Raine, and Fowler (2006). Briefly, the Southern California Twin Project is a longitudinal study assessing the development of aggressive and antisocial behaviors from childhood to young adulthood. The present study used data from the first wave of assessment that was conducted from 2000 to 2004.

The twins and their families were recruited from Los Angeles County. The sample is representative of the ethnic and socioeconomic diversity of the greater Los Angeles area and includes 605 families ($N = 1,219$ children). The mean age of the children in the first wave assessment was 9.60 years ($SD = 0.60$), and the gender distribution was approximately equal with 48.7% boys ($n = 594$) and 51.3% girls ($n = 625$). The twins and their primary

caregivers (mainly the biological mothers) participated in a 6–8 hr laboratory assessment at USC. The caregivers were administered self-report questionnaires and interviewed about their twins' behaviors. The mean age of the caregivers was 40.14 years ($SD = 6.61$).

Zygosity was established through DNA micro-satellite analyses. Details on the determination of twins' zygosity have been provided in Baker, Jacobson, Raine, and Bezdjian (2007). The sample is composed of the following zygosity groups: monozygotic (MZ) males ($N = 138$ pairs), MZ females ($N = 139$ pairs), dizygotic (DZ) males ($N = 84$ pairs), DZ females ($N = 97$ pairs), and DZ opposite-sex pairs ($N = 147$ pairs).

Measures

Child Psychopathy Scale—Psychopathic personality traits were measured using a slightly extended version of the revised CPS–Revised Extended (Lynam, 1997; Lynam et al., 2005; see also Falkenbach, Poythress, & Heide, 2003). The CPS is a well-validated instrument measuring psychopathic personality traits in children and adolescents (see Lynam, 1997) and is composed of 14 subscales (based on 58 *yes* or *no* items scored 1 = *no*, 2 = *yes*), which include assessments of glibness, untruthfulness, lack of guilt, callousness, impulsiveness, boredom susceptibility, manipulation, poverty of affect, parasitic lifestyle, behavioral dyscontrol, lack of planning, unreliability, failure to accept responsibility, and grandiosity. The CPS was administered to the twins (self-report) and their caregivers in interview form. Internal reliabilities for the total composite score was acceptable with Cronbach's $\alpha = .83$. The CPS total score (mean of all 58 items) demonstrates moderate to strong correlations with caregiver reports of children's aggressive and antisocial behaviors (see Baker et al., 2007).

The CPS was primarily developed to operationalize, in childhood and adolescence, the *personality* traits found in the PCL–R (Hare, 2000). Two PCL–R items, criminal versatility and juvenile delinquency, were not included so that the CPS might serve as a pure measure of personality uncontaminated by antisocial behavior. Further validation of the instrument is provided in Lynam (1997).

Previously, we have demonstrated through exploratory principal component analyses and confirmatory factor analyses that caregiver ratings of CPS subscales have a two-dimensional solution within our sample. That is, a two-factor solution was the best fitting and most appropriate solution and fit better than a one-, three-, and four-factor solution. The two factors found were callous & disinhibited and manipulative–deceitful (Bezdjian, Raine, Baker, & Lynam, 2011). Based on this finding, confirmatory factor analyses were also conducted in child self-reported data of psychopathic personality traits using the CPS. Results indicated that the same factor structure found in adults was also the best fitting solution for child self-reported data (two-factor solution: $\chi^2 = 362.59$, $df = 78$, comparative fit index [CFI] = .84, Akaike's information criterion [AIC] = 22916.29, root mean square error of approximation [RMSEA] = .07; one-factor solution: $\chi^2 = 494.39$, $df = 77$, CFI = .77, AIC = 22953.52, RMSEA = .09; three-factor solution: $\chi^2 = 531.35$, $df = 76$, CFI = .75, AIC = 22992.49, RMSEA = .10; the three-factor model is based on the factor structure tested in Salekin, Brannen, Zalot, Leistico, & Neumann, 2006). Thus, two factor scores were computed for each child within each rater, by using means of items for scales loading on each factor.

Reactive and Proactive Aggression Questionnaire—To measure aggressive behavior in the twins, the Reactive and Proactive Aggression Questionnaire (RPQ) was completed by both the twins (self-report) and their caregivers (Raine et al., 2006). The RPQ is a 23-item validated questionnaire designed to measure reactive and proactive aggression in young children and adolescents (Raine et al., 2006). The RPQ includes 11 reactive items

(e.g. hitting others when teased, gets angry when frustrated) and 12 proactive items (e.g., damages things for fun, uses force to get things from others). The RPQ is scored on a 3-point response scale (0 = *never*, 1 = *sometimes*, 2 = *often*). Mean scores for the item responses were computed to form reactive and proactive aggression subscales (obtained from principal component analyses of the 23 items). Caregiver (mainly the biological mothers) reports of the twins' aggressive behaviors as well as the twins' self-reports were used in the current study. It has been shown in at least two prior independent studies that the RPQ has a two-factor reactive–proactive solution (Baker et al., 2008; Raine et al., 2006).

Data Analyses

The genetic analyses carried out in the current study are based on traditional twin designs and the general assumptions of twin studies. MZ twins share 100% of their genes, while DZ twins share on average 50% of their genes. The total phenotypic variance of a measured trait can be divided into additive genetic factors (*A*), shared environmental factors (*C*), and nonshared environmental factors (*E*). In general, heritability is the proportion of total phenotypic variance because of genetic variation (Neale & Cardon, 1992; Plomin, DeFries, McClearn, & McGuffin, 2001). For a particular trait or measure, MZ correlations that are higher than DZ correlations are indicative of genetic influences, whereas a DZ correlation more than half the MZ intraclass correlation signifies the presence of both genetic and shared environmental effects. The cross-twin cross-trait correlations yield information about the genetic and environmental correlations between traits. Cross-twin cross-trait correlations are interpreted the same way as intraclass correlations—that is, greater values for MZ compared with DZ pairs suggest shared or correlated genetic influences for two traits.

Bivariate Cholesky decompositions were fitted to estimate genetic correlation (r_g), shared environmental correlation (r_c), and nonshared environmental correlation (r_e) between CPS factors and reactive and proactive aggression. A genetic correlation indicates the extent to which genetic effects on one measure are associated with genetic effects on another measure (Posthuma et al., 2003).

A series of multivariate models were also fitted to investigate further the nature of the relations among reactive and proactive aggression and the CPS factors. These models included: (a) saturated covariance models to estimate the phenotypic thresholds and variance–covariance matrices within each of the five zygosity groups, and (b) multivariate models to determine the magnitude and significance of genetic (*A*), shared twin environment (*C*), and nonshared environment (*E*) variance for each factor, as well as to understand the nature of the covariance between each factor, that is, the extent to which genetic and environmental aspects may influence the shared genetic and environmental influences between the factors. In particular, we fitted a Cholesky decomposition in which correlated genetic and environmental effects were included for both factors, and an independent pathway model in which genetic and environmental influences were uncorrelated for the two factors. In addition to estimating genetic and environmental influences common to the measures, an independent pathway estimates genetic and environmental influences specific to each measure.

All models were fitted with the structural equation program Mx (Neale, Boker, Xie, & Maes, 2003) using a maximum likelihood estimation procedure for raw data. Raw maximum likelihood yields a goodness-of-fit index called log-likelihood. The fit of the models was assessed and determined by comparing the AIC between different models. The AIC represents the balance between model fit and the number of parameters (parsimony), with lower values (i.e., larger negative) of AIC indicating the most statistically acceptable or suitable model (Akaike, 1987). Finally, a third model-selection statistic was the Bayesian information criterion (BIC), where increasingly negative values also correspond to

increasingly better fitting models (Raftery, 1995). All measures were ranked and standardized to normalize the skewed data and estimates displayed are after transformations.

Results

Descriptive Statistics

Means and standard deviations (by sex and zygosity) for the two CPS factors (callous & disinhibited and manipulative & deceitful) as well as the two RPQ factors (reactive and proactive aggression) for both caregiver and child self-reports are provided in Table 1. No mean or variance differences were found between Twins 1 and 2 or between MZ and DZ twins. Mean sex differences were found using caregiver reports of both psychopathic dimensions and reactive and proactive aggression with boys showing significantly higher mean values than girls on all four factors: manipulative & deceitful ($t = 3.65$, $df = 1,213$, $p < .01$), callous–disinhibited ($t = 5.99$, $df = 1,189$, $p < .01$), reactive aggression ($t = 4.32$, $df = 1,213$, $p < .01$), and proactive aggression ($t = 2.79$, $df = 1,196$, $p < .01$). Sex differences of reactive and proactive aggression have been previously provided in Baker et al. (2008). Likewise, mean sex differences were also found using child self-reports with boys showing significantly higher mean values than girls on all four factors: manipulative & deceitful ($t = 3.96$, $df = 1,197$, $p < .01$), callous & disinhibited ($t = 3.34$, $df = 1,197$, $p < .01$), reactive aggression ($t = 3.13$, $df = 1,203$, $p < .01$), and proactive aggression ($t = 3.99$, $df = 1,203$, $p < .01$).

For caregivers' report, the phenotypic correlations for the two psychopathic dimensions with reactive and proactive aggression were as follows: manipulative & deceitful and reactive aggression: $r_p = .50$, $p < .01$; manipulative & deceitful and proactive aggression: $r_p = .47$, $p < .01$; callous & disinhibited and reactive aggression: $r_p = .51$, $p < .01$; callous & disinhibited and proactive aggression: $r_p = .43$, $p < .01$. For twins' self-report, the phenotypic correlations for the two psychopathic dimensions with reactive and proactive aggression were as follows: manipulative & deceitful and reactive aggression: $r_p = .39$, $p < .01$; manipulative & deceitful and proactive aggression: $r_p = .46$, $p < .01$; callous & disinhibited and reactive aggression: $r_p = .45$, $p < .01$; callous & disinhibited and proactive aggression: $r_p = .46$, $p < .01$. Additionally, cross-report correlations among the two psychopathy dimensions and reactive and proactive aggression are reported in the Appendix.

Twin Correlations

Intraclass and cross-twin cross-trait correlations for the two psychopathic personality factors and reactive and proactive aggression within each of the five zygosity groups are reported in Table 2 for caregiver reports and Table 3 for child self-reports. The MZ intraclass correlations were consistently greater than the corresponding DZ intraclass correlations, both in male and female twin pairs, suggesting that genetic influences are important for manipulative & deceitful and callous & disinhibited personality traits and for reactive and proactive aggression. In general, the cross-twin cross-trait correlations between the psychopathic personality traits and aggressive behavior scales were also greater for MZ than for DZ pairs, indicating that genetic effects are important for the relation between manipulative & deceitful and callous & disinhibited traits with reactive and proactive aggression.

Bivariate Modeling

Table 4 displays the genetic and environmental correlations between the psychopathic personality dimensions and reactive and proactive aggression, separately for child and caregiver reports. In bivariate genetic analyses of each CPS factor paired with each of the two aggression scales, a model that constrained genetic and environmental parameter

estimates to be equal in males and females fitted best. Also, the shared environmental correlation could be dropped in all four pairings without a decrease in fit (results available upon request).

For caregivers, the genetic and nonshared environmental correlations between the callous & disinhibited factor with reactive aggression were $r_g = .49$ and $r_e = .47$, respectively. The genetic and environmental correlations between callous & disinhibited and proactive aggression were $r_g = .41$ and $r_e = .37$, respectively. Similarly, the genetic and environmental correlations between the manipulative & deceitful factor with reactive aggression were $r_g = .57$ and $r_e = .34$, respectively, while $r_g = .35$ and $r_e = .41$ for manipulative & deceitful with proactive aggression. Both genetic and non-shared environmental correlations were significant ($p < .05$) in all cases except for the genetic correlation between proactive aggression and either psychopathic factor using caregiver reports of the twins' behavior.

The twins' self-report data also revealed significant genetic and nonshared environmental correlations between the two psychopathic personality traits and reactive and proactive aggression. Specifically, the genetic and nonshared environmental correlations between the callous & disinhibited factor with reactive aggression were $r_g = .59$ and $r_e = .37$, respectively. The genetic and environmental correlations between callous & disinhibited and proactive aggression were $r_g = .76$ and $r_e = .30$, respectively. Correspondingly, the genetic and environmental correlations between the manipulative & deceitful factor with reactive aggression were also significant ($r_g = .57$ and $r_e = .29$, respectively), as were those for manipulative & deceitful with proactive aggression ($r_g = .81$ and $r_e = .29$).

Additionally, the difference in correlations for reactive and proactive scales were also tested and results revealed for twins' self-report that the genetic correlation between callous & disinhibited and proactive aggression was significantly greater than the genetic correlation between callous & disinhibited and reactive aggression ($\chi^2_{\text{diff}} = 140.30$, $df = 112$, $p = .04$; the genetic correlation could not be constrained to be equal across types of aggression). Similarly, the genetic correlation between manipulative & deceitful traits and proactive aggression was also significantly greater than the genetic correlation between the manipulative & deceitful traits and reactive aggression in the twins' self-report data ($\chi^2_{\text{diff}} = 137.13$, $df = 112$, $p = .05$; i.e., the genetic correlation could not be constrained to be equal for manipulative & deceitful with the two types of aggression). The genetic relation between psychopathic personality traits and aggression was significantly stronger for proactive than reactive aggression, but only in child self-reports. The genetic correlation between proactive aggression with either the callous & disinhibited or manipulative & deceitful psychopathic personality dimension was not significant in caregiver reports of the twins' behaviors.

Multivariate Modeling

A summary of the various multivariate genetic models fit to the two psychopathic personality traits and reactive and proactive aggression in both caregiver and child self-reports is provided in Table 5. Specifically, a fully saturated model (Model 1 in Table 5) was used as a baseline comparison for a Cholesky decomposition (Model 2), and an independent pathway model (Model 3a–f). The one-factor independent pathway model demonstrated the best fit to the data for both caregiver (Model 3c) and child self-reported data (Model 3d). Constraining parameters to be equal for males and females did not result in a significant loss in fit ($\Delta\chi^2 = 31.15$, $\Delta df = 24$, $p = .15$; Model 3a); therefore, we constrained the sexes to be equal in subsequent models. Additional constraints on the independent pathway model revealed that both common (C_c) and specific shared environmental (C_s) effects could be dropped without a significant loss in fit as demonstrated by the lower AIC and BIC indices (Model 3c in caregiver reports: $\Delta\chi^2 = 14.58$, $\Delta df = 8$, $p = .07$; in caregiver reports and Model 3d in twin self-reports: $\Delta\chi^2 = 9.16$, $\Delta df = 12$, $p > .10$). Additionally, genetic effects

(common or specific) could not be dropped from either caregiver reports ($\Delta\chi^2 = 60.82$, $\Delta df = 8$, $p < .01$), or twin self-reported data ($\Delta\chi^2 = 13.19$, $\Delta df = 4$, $p < .001$) without a significant loss in fit to our model.

Squared standardized parameter estimates from the one-factor AE independent pathway model with equal effects across gender are provided in Figure 1 (for both caregiver and child self-reports). As shown, all shared effects from common genetic (A_c) and environmental (E_c) factors were significant ($p < .05$) for both caregiver and child reports, illustrating the overlapping etiologies for psychopathic personality traits and aggression. Specific effects for each measure were also significant in all cases except for genetic influences (A_s) on the callous & disinhibited factor in child reports. While a shared etiology for psychopathic personality traits and aggression is clearly evident, there are also measure-specific influences for each construct, which are both genetic and environmental in origin.

Summing the genetic paths for each measure yields the heritability estimate. On the same note, summing the nonshared environmental paths yields the relative influence of the environmental effects. For caregiver reports, the heritability for the two psychopathic personality factors and for reactive and proactive aggression was moderate but significant: genetic effects explained 53%–57% ($p < .05$) of the variance in each of these psychopathic and aggressive traits. Similarly, the nonshared environmental effects for the two psychopathic personality traits and reactive and proactive aggression were also moderate, explaining 43%–48% ($p < .05$) of the variance in these traits (see Figure 1).

Likewise, for child self-report significant genetic and nonshared environmental effects for both psychopathic personality traits and reactive and proactive aggression were found. Genetic effects (both common and specific) explained 33%–38% ($p < .05$) of the variance in psychopathic and aggressive traits, while nonshared environmental influences explained 48%–62% ($p < .05$) of the variance in psychopathic and aggressive behavioral traits in children (see Figure 1).

Discussion

This study provides one of the first reports from a large twin study examining the genetic and environmental covariation among childhood psychopathic personality traits and reactive and proactive aggressive behaviors. Our results demonstrated, most importantly, a significant genetic and non-shared environmental correlation among psychopathic personality traits and reactive and proactive aggressive behaviors in children using both caregiver reports and child self-reports. We also found unique genetic and nonshared environmental influences in each of the measures, suggesting some independence among these behaviors and constructs. Furthermore, we also found a significant association between psychopathic personality traits and proactive aggression, but only in child self-reports. This may in part be due to the fact that children may have more insight into their own actions and behaviors (at least more so than their parents).

Common genetic estimates for psychopathic personality traits and aggression ranged from 21% to 46% using caregiver reports and 15% to 26% using the twins' self-reported data. Bivariate analyses also revealed significant genetic correlations between both psychopathic personality factors with reactive and proactive aggression indicating further that genetic influences may be common to psychopathy and aggression during childhood. This shared influence could arise because of a common set of genes influencing both types of behaviors and in turn may influence the susceptibility to both psychopathic personality traits and aggressive behaviors and thus their covariation. Evidence of genetic influence on these traits, however, does not signify that the children in our sample are not capable of change or

that they are impervious to intervention methods. This result falls in line with other twin studies on psychopathic personality traits that have found that a common genetic factor influences psychopathic personality traits and different types of externalizing behaviors in adolescents (Larsson et al., 2006).

In comparing shared genetic influences between psychopathic personality traits with different forms of aggression, we found that the genetic correlation between the callous & disinhibited factor and proactive aggression was significantly greater than callous & disinhibited and reactive aggression, but only using the twins' self-reports. Similarly, the twins' self-reports also indicated that the genetic correlation between the manipulative & deceitful factor and proactive aggression was significantly greater than with reactive aggression. These results fall in line with what has been previously found phenotypically in the psychopathy and aggression literature (Patrick & Zempolich, 1998). Previous analyses within this sample have also demonstrated that children might be better reporters of their own aggressive behaviors in that they have better knowledge of their motivation for perpetrating different aggressive acts as compared to other reporters including caregivers (see Baker et al., 2008; Raine et al., 2006). Relatedly, we have found a better fit of the two-factor proactive-reactive model to self-report data compared to caregiver or teacher reports, indicating somewhat greater validity and differentiation between these two forms of aggression when assessed by self-report (Baker et al., 2008).

Furthermore, it may be difficult for a single rater to provide a complete or thorough picture of a desired behavior. Data collected from multiple informants has been previously found to be more informative (Baker et al., 2006; Baker et al., 2008). In general, interrater agreement has been found to be low (Youngstrom, Loeber, & Stouthamer-Loeber, 2000). Moreover, meta-analyses conducted on informant data demonstrated that a correlation of $r = .28$ ($p < .05$) was found between different types of informants, and a correlation of $r = .22$ ($p < .05$) was found between self-reporters and other informants or reporters (Achenbach, McConaughy, & Howell, 1987). These findings suggest that different raters may be privy to different aspects of a behavior. Additionally, individuals are also likely to behave differently in different situations. For example, parents may be less aware of their children's proactive aggression than their reactive aggression. It is also possible that parents may not fully know every detail of their children's behaviors.

Another source of rater disagreement may be rater bias. Rater bias may occur in twin studies because a single informant (mainly the biological mothers) is reporting on both twins (either as being more similar than they actually are or exaggerating the differences; Bartels et al., 2003; Hudziak et al., 2003). Additionally, greater shared environmental influences found in parental reports may be indicative of rater bias; however, no shared environmental influences were found for the current study indicating that rater bias may be low.

Moreover, the genetic relation between psychopathic personality traits and aggression was significantly stronger for proactive than reactive aggression, but only in child self-reports. This finding is in line with the prediction that psychopathic personality traits should have more of a core relation with proactive than reactive aggression (Patrick & Zempolich, 1998). The lack of a significant difference in genetic and environmental correlations for psychopathic personality traits with reactive versus proactive aggression using caregiver reports may stem in part from parents' inability to distinguish proactive from reactive aggressive behaviors in their children, as previously reported in this study (Baker et al., 2008).

We also found a significant nonshared environmental factor commonly influencing psychopathic personality traits and reactive and proactive aggression. It should also be noted

that the model used in the present study partitions measurement error away from variance in the latent nonshared environmental factor. When doing this, common nonshared environmental estimates ranged from 15% to 19% in caregiver reports, and 14% to 30% in twins' self-reports. These nonshared environmental influences in psychopathic personality traits and aggression may involve experiences or circumstances that are unique to psychopathic personality traits or aggression. They may also include influences that are unique to the individual (not shared by his or her cotwin). These may involve the young twins having different friends and influences.

In addition to finding common genetic and environmental factors among psychopathic personality traits and aggression, our study also found substantial and significant specific genetic and nonshared environmental influences, including measurement error, on psychopathic personality traits and reactive and proactive aggression. This implies that there are etiological differences in psychopathic personality traits and aggressive behaviors in our sample of young twins. As the genetic correlations between psychopathic personality traits and aggression were significantly less than 1.0 using both caregiver and twins' self-reported data, this lends further support to the notion that there are multiple factors that capture psychopathic personality traits and aggression within this sample.

In line with previous research on psychopathic personality traits (Larsson et al., 2006) and aggressive behavior (Eley, Lichtenstein, & Moffitt, 2003) we did not find any quantitative sex differences in the covariation among psychopathic personality traits and reactive and proactive aggression using either twins' self-reported data or the caregiver reports of the twins' behaviors. Evidence suggests that psychopaths seem to choose to engage in instrumental or proactive aggression and antisocial behavior (Blair, 2007), and that psychopathy is a relatively good predictor of violence (Hart, 1998; Salekin et al., 1996). Others have also suggested that psychopathy is significantly correlated with future violence (Grann, Langstrom, Tengstrom, & Kullgren, 1999; Hemphill et al., 1998). Knowledge and understanding of the link between psychopathic personality traits with aggression may greatly aid in the prediction of future antisocial and violent behavior in adult offenders (Salekin et al., 1996). Research has demonstrated that psychopathic offenders were approximately five times more likely than nonpsychopathic offenders to engage in violent recidivistic behaviors (Serin & Amos, 1995). The results from the current study extend prior knowledge of the relation between psychopathic personality traits and aggression in examining the genetic overlap of psychopathic personality traits with reactive and proactive aggression. Results demonstrated a similar pattern between psychopathic personality traits and reactive and proactive aggression in this sample. Thus, it may be that it is difficult to differentiate these types of aggressive behaviors at such a young age when using caregiver reports.

Although most research on psychopathy has focused thus far on adults, there is increasing evidence that maintains that psychopathic personality traits are related to aggression much earlier in life (Frick, Bodin, & Barry, 2000; Lynam, 2002). For example, several researchers have purported that psychopathic personality traits emerge early in life (in childhood) in the form of "callous-unemotional" traits (Lynam, 1997, 2002). These traits resemble adult psychopathic personality traits and may be associated with serious aggressive behavior and may signal a pattern of antisocial and violent behavior (Dodge, 1991; Frick, 1998). The current findings do provide evidence that these traits may be associated early in life. Fully understanding antisocial and aggressive behaviors is crucial for designating a subgroup of antisocial youth who showed more severe aggression and violence, especially youth who were more likely to show both instrumental (e.g., for gain) and reactive (e.g., in response to perceived provocation) aggression (Enebrink, Andershed, & Langstrom, 2005; Frick et al., 2003; Kruh et al., 2005).

The current findings of significant genetic and environmental covariance between psychopathic personality traits and aggressive behaviors may have implications for future molecular genetic studies. Understanding the covariance among complex behaviors including antisocial and aggressive behaviors may aid in trying to identify susceptibility genes as well as aid in the possible prevention and intervention of externalizing disorders through identification of at-risk children early in life. Moreover, the significant nonshared environmental effects found may also suggest that unique experiences (e.g., influence from peers) may play an important role in the development of these behaviors—perhaps, intervention methods may also want to target and incorporate influence from peers.

Study Strengths and Limitations

The results of the current study should be examined while taking a few limitations into account. First, we may not necessarily be able to generalize the results from our twin study to the general population because there may be something innately different about twins compared with nontwins. However, our twins are no more or less disordered than any other population in terms of conduct or oppositional defiant disorder (Baker et al., 2006). The examination of psychopathic personality traits in children could also be questioned. Some researchers believe that it might not be developmentally appropriate to diagnose this syndrome in children and adolescents (Vincent & Hart, 2002); however, studies have shown that psychopathic personality traits can be seen in young children (Lynam, 1997; Viding et al., 2005). Moreover, one of the purposes of investigating personality disorders including psychopathic-like traits is to increase the understanding of these traits (and increase the genetic and environmental underpinnings of these traits). Additionally, results from the current study demonstrate the variance proportions associated with psychopathic personality traits and aggressive behaviors as well as the covariation of these two complex constructs; however, as with many behavior genetic studies, they do not inform us about the underpinning biological mechanisms associated with complex behaviors or disorders.

Finally, our study is cross-sectional involving a sample of 9- to 10-year-old twins, and therefore does not consider any developmental changes in the different constructs, or whether one precedes the other. However, targeting youth at such a young preado-lescent age may also be considered a strength and provide insight into early behavior problems.

Conclusions and Future Directions

In conclusion, the results from the current study may aid in the understanding of the etiology of psychopathic personality traits and aggressive behaviors early in life during childhood. Additionally, we found that the twins may be more aware of their motivations for different forms of aggressive behaviors (proactive–reactive) compared to their caregivers who may see the behavior but not necessarily the motivation. This study may further be extended in the future to explore the longitudinal stability or change (both phenotypically and etiologically) of these traits throughout adolescence and adulthood.

Acknowledgments

The authors wish to thank the USC Twin Project staff for assistance in data collection and scoring, and the twins and their families for their participation in this research. This study was supported by grants to Serena Bezdjian from NIMH (F31-MH068953) and NIDA (T32-DA07313), to Laura Baker from NIMH (R01 MH58354), and to Adrian Raine from NIMH (Independent Scientist Award K02 MH01114-08). Catherine Tuvblad was supported by postdoctoral stipends from the Swedish Council for Working Life and Social Research (Project 2006-1501) and the Sweden-America Foundation.

References

- Achenbach TM, McConaughy SH, Howell CT. Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situation specificity. *Psychological Bulletin*. 1987; 101:213–232. [PubMed: 3562706]
- Akaike AC. Factor analysis and AIC. *Psychometrika*. 1987; 52:317–332.
- 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]
- Baker LA, Jacobson KJ, Raine A, Bezdjian S. Genetic and environmental bases of childhood antisocial behavior: A multi-informant twin study. *Journal of Abnormal Psychology*. 2007; 116:219–235. [PubMed: 17516756]
- Baker LA, Raine A, Liu J, Jacobson KC. Differential genetic and environmental influences on reactive and proactive aggression in children. *Journal of Abnormal Child Psychology*. 2008; 36:1265–1278. [PubMed: 18615267]
- Barratt ES, Stanford MS, Dowdy L, Liebman MJ, Kent TA. Impulsive and premeditated aggression: A factor analysis of self-reported acts. *Psychiatry Research*. 1999; 86:163–173. [PubMed: 10397418]
- Bartels M, Hudziak JJ, Boomsma DI, Rietveld MJH, Van Beijsterveldt TCEM, Van Den Oord EJCG. A study of parent ratings of internalizing and externalizing problem behavior in 12 year old twins. *Journal of American Academy of Child and Adolescent Psychiatry*. 2003; 42:1351–1359.
- Berkowitz, L. The experience of anger as a parallel process in the display of impulsive, “angry” aggression. In: Green, RG.; Donnerstein, EI., editors. *Aggression: Theoretical and empirical views*. New York: Academic Press; 1983. p. 103-134.
- Berkowitz, L. *Aggression: Its causes, consequences, and control*. Philadelphia: Temple University Press; 1993.
- Bezdjian S, Raine A, Baker LA, Lynam DR. Psychopathic personality traits in children: Genetic and environmental contributions. *Psychological Medicine*. 2011; 41:589–600. [PubMed: 20482945]
- Blair RJR. Neurocognitive models of aggression, the antisocial personality disorders, and psychopathy. *Journal of Neurology, Neurosurgery and Psychiatry*. 2001; 71:727–731.
- Blair RJR. Aggression, psychopathy and free will from a cognitive neuroscience perspective. *Behavioral Sciences and the Law*. 2007; 25:321–331. [PubMed: 17393398]
- Blonigen DM, Hicks BM, Krueger RF, Patrick CJ, Iacono WG. Continuity and change in psychopathic traits as measured via normal-range personality: A longitudinal-biometric study. *Journal of Abnormal Psychology*. 2006; 115:85–95. [PubMed: 16492099]
- Brendgen M, Vitaro F, Boivin M, Dione G, Perusse D. Examining genetic and environmental effects on reactive versus proactive aggression. *Developmental Psychology*. 2006; 42:1299–1312. [PubMed: 17087562]
- Brendgen M, Vitaro R, Tremblay RE, Lavoie F. Reactive and proactive aggression: Predictions to physical violence in different contexts and moderating effects of parental monitoring and caregiving behavior. *Journal of Abnormal Child Psychology*. 2001; 29:293–304. [PubMed: 11523835]
- Cornell D, Warren J, Hawk G, Stafford E, Oram G, Pine D. Psychopathy in instrumental and reactive offenders. *Journal of Consulting and Clinical Psychology*. 1996; 64:783–790. [PubMed: 8803369]
- Crick NR, Dodge K. Social information processing mechanisms in reactive and proactive aggression. *Child Development*. 1996; 67:993–1002. [PubMed: 8706540]
- Dodge, K. The structure and function of reactive and proactive aggression. In: Pepler, DJ.; Rubin, KH., editors. *The development and treatment of childhood aggression*. Hillsdale, NJ: Erlbaum; 1991. p. 201-218.
- Dodge KA, Coie JD. Social-information processing factors in reactive and proactive aggression in children’s peer groups. *Journal of Personality and Social Psychology*. 1987; 53:1146–1158. [PubMed: 3694454]
- Eley TC, Lichtenstein P, Moffitt TE. A longitudinal behavioral genetic analysis of the etiology of aggressive and non-aggressive antisocial behavior. *Development and Psychopathology*. 2003; 15:383–402. [PubMed: 12931834]

- Enebrink P, Andershed H, Langstrom N. Callous-unemotional traits are associated with clinical severity in referred boys with conduct problems. *Nordic Journal of Psychiatry*. 2005; 59:431–440. [PubMed: 16316895]
- Falkenbach DM, Poythress NG, Heide KM. Psychopathic features in a juvenile diversion population: Reliability and predictive validity of two self-report measures. *Behavioral Sciences and the Law*. 2003; 21:787–805. [PubMed: 14696031]
- Fite PJ, Stoppelbein L, Greening L. Proactive and reactive aggression in a child psychiatric inpatient population: Relations to psychopathic characteristics. *Criminal Justice and Behavior*. 2009; 36:481–493.
- Fite PJ, Stoppelbein L, Greening L, Gaertner AE. Further validation of a measure of proactive and reactive aggression within a clinical child population. *Child Psychiatry and Human Development*. 2009; 40:367–382. [PubMed: 19296214]
- Flight JJ, Forth AE. Instrumentally violent youths: The roles of psychopathic traits, empathy, and attachment. *Criminal Justice and Behavior*. 2007; 34:739–751.
- Frick PJ. *Conduct disorders and severe antisocial Behavior*. New York: Plenum; 1998.
- Frick PJ, Bodin SD, Barry CT. Psychopathic traits and conduct problems in community and clinic-referred samples of children: Further development of the Psychopathy Screening Device. *Psychological Assessment*. 2000; 12:382–393. [PubMed: 11147105]
- Frick PJ, Cornell AH, Barry CT, Bodin SD, Dane HE. Callous-unemotional traits and conduct problems in the prediction of conduct problem severity, aggression and self-report of delinquency. *Journal of Abnormal Child Psychology*. 2003; 31:457–470. [PubMed: 12831233]
- Grann M, Langstrom N, Tengstrom A, Kullgren G. Psychopathy (PCL-R) predicts violent recidivism among criminal offenders with personality disorders in Sweden. *Law and Human Behavior*. 1999; 23:205–217. [PubMed: 10333757]
- Hare, RD. *The revised psychopathy checklist*. Toronto, Canada: Multi-Health Systems; 1991.
- Hare, RD.; Jutai, J. Criminal history of the male psychopath: Some preliminary data. In: Van Dusen, KT.; Mednick, SA., editors. *Prospective studies of crime and delinquency*. Boston: Kluwer-Nijhoff; 1983. p. 225-236.
- Hare, RD.; Neumann, CS.; Patrick, CJ. The PCL-R Assessment of Psychopathy: Development, structural properties, and new directions. In: Patrick, CJ., editor. *Handbook of psychopathy*. New York: Guilford; 2006. p. 58-88.
- Harpur TJ, Hare RD. Assessment of psychopathy as a function of age. *Journal of Abnormal Psychology*. 1994; 103:604–609. [PubMed: 7822561]
- Hart SD. The role of psychopathy in assessing risk for violence: Conceptual and methodological issues. *Legal and Criminological Psychology*. 1998; 3:121–137.
- Hemphill, JF.; Templeman, R.; Wong, S.; Hare, RD. Psychopathy and crime: Recidivism and criminal careers. In: Cooke, D.; Forth, A.; Hare, RD., editors. *Psychopathy: Theory, research, and implications for society*. Dordrecht, The Netherlands: Kluwer; 1998. p. 374-399.
- Hudziak JJ, van Beijsterveldt CE, Bartels M, Rietveld MJ, Rettew DC, Derks EM, et al. Individual differences in aggression: Genetic analyses by age, gender and informant in 3-, 7-, and 10- year-old Dutch twins. *Behavior Genetics*. 2003; 33:575–589. [PubMed: 14574134]
- Kruh IP, Whittemore K, Arnaut GLY, Manley J, Gage B, Gagliardi G. The concurrent validity of the psychopathic personality inventory and its relative association with past violence in a sample of insanity acquittees. *International Journal of Forensic Mental Health*. 2005; 4:135–145.
- Lamarche V, Brendgen M, Boivin M, Vitaro F, Dionne G, Périus D. Do friends' characteristics moderate the prospective links between peer victimization and reactive and proactive aggression? *Journal of Abnormal Child Psychology*. 2007; 35:665–680. [PubMed: 17503177]
- Larsson H, Andershed H, Lichtenstein P. A genetic factor explains most of the variation in the psychopathic personality. *Journal of Abnormal Psychology*. 2006; 115:221–230. [PubMed: 16737387]
- Lynam DR. Pursuing the psychopath: Capturing the fledgling psychopath in a nomological net. *Journal of Abnormal Psychology*. 1997; 106:425–438. [PubMed: 9241944]
- Lynam DR. Fledgling psychopathy: A view from personality theory. *Law and Human Behavior*. 2002; 26:255–259. [PubMed: 11985302]

- Lynam DR, Caspi A, Moffitt TE, Raine A, Loeber R, Loeber-Stouthamer M. Adolescent psychopathy and the Big Five: Results from two samples. *Journal of Abnormal Child Psychology*. 2005; 33:431–443. [PubMed: 16118990]
- Neale, MC.; Boker, SM.; Xie, G.; Maes, H. Mx: Statistical modeling. Richmond: Department of Psychiatry, Medical College of Virginia; 2003.
- Neale, MC.; Cardon, LR. Methodology for genetic studies of twins and families. Dordrecht, The Netherlands: Kluwer Academic; 1992.
- Patrick CJ, Zempolich KA. Emotion and aggression in the psychopathic personality. *Aggression and Violent Behavior*. 1998; 3:303–338.
- Plomin, R.; DeFries, JC.; McClearn, GE.; McGuffin, P. Behavioral Genetics. New York: Worth; 2001.
- Porter, S.; Woodworth, M. Psychopathy and aggression. In: Patrick, CJ., editor. *Handbook of psychopathy*. New York: Guilford; 2006. p. 481–494.
- Posthuma D, Beem AL, de Geus EJC, van Baal CM, von Hjelmberg JB, Iachine I, et al. Theory and practice in quantitative genetics. *Twin Research and Human Genetics*. 2003; 6:361–376.
- Poulin F, Boivin M. Reactive and proactive aggression: Evidence of a two-factor model. *Psychological Assessment*. 2000; 12:115–122. [PubMed: 10887757]
- Pulkkinen L. Proactive and reactive aggression in early adolescence as precursors to anti- and prosocial behaviors in young adults. *Aggressive Behavior*. 1996; 22:241–257.
- Raftery AE. Bayesian model selection in social research. *Sociological Methodology*. 1995; 25:111–163.
- Raine A, Dodge K, Loeber R, Gatzke-Kopp L, Lynam D, Reynolds C, et al. The reactive-proactive aggression questionnaire: Differential correlates of reactive and proactive aggression in adolescent boys. *Aggressive Behavior*. 2006; 32:159–171. [PubMed: 20798781]
- Salekin RT, Brannen DN, Zalot AA, Leistico AM, Neumann CS. Factor structure of psychopathy in youth: Testing the applicability of the new four-factor model. *Criminal Justice and Behavior*. 2006; 33:135–157.
- Salekin RT, Rogers R, Sewell KW. A review and meta-analysis of the Psychopathy Checklist and Psychopathy Checklist-Revised: Predictive validity of dangerousness. *Clinical Psychology: Science and Practice*. 1996; 3:203–215.
- Salmivalli C, Helteenvuori T. Reactive, but not proactive aggression predicts victimization among boys. *Aggressive Behavior*. 2007; 33:198–206. [PubMed: 17444526]
- Salmivalli C, Nieminen E. Proactive and reactive aggression among school bullies, victims, and bully-victims. *Aggressive behavior*. 2002; 28:30–44.
- Schwartz D, Dodge K, Coie JD, Hubbard JA, Cillessen AHN, Lemerise EA, et al. Social-cognitive and behavioral correlates of aggression and victimization in boys' play groups. *Journal of Abnormal Child Psychology*. 1998; 26:431–440. [PubMed: 9915650]
- Serin RC, Amos NL. The role of psychopathy in the assessment of dangerousness. *International Journal of Law in Psychiatry*. 1995; 18:231–238.
- Taylor A, Loney BR, Bobadilla L, Iacono WG, McGue M. Genetic and environmental influences on psychopathy trait dimensions in a community sample of male twins. *Journal of Abnormal Child Psychology*. 2003; 31:633–645. [PubMed: 14658743]
- Tuvblad C, Raine A, Zheng M, Baker AL. Genetic and environmental stability differs in reactive and proactive aggression. *Aggressive Behavior*. 2009; 35:437–452. [PubMed: 19688841]
- 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; 46:592–597. [PubMed: 15877765]
- Vincent, GM.; Hart, SD. Psychopathy in childhood and adolescence: Implications for the assessment and management of multi-problem youths. In: Corrado, Raymond R.; Roesch, Ronald; Hart, Stephen D.; Gierowski, Jzef K., editors. *Multi-problem violence youth: A foundation for comparative research on needs, interventions and outcomes*. Vol. 324. Amsterdam, The Netherlands: IOS Press; 2002. p. xivSeries I: Life and behavioural sciences, 150–163
- Vitaro, F.; Brendgen, M. Proactive and reactive aggression: A developmental perspective. In: Tremblay, RE.; Hartup, WM.; Archer, J., editors. *The developmental origins of aggression*. New York: Guilford Press; 2005. p. 178–201.

- Vitaro F, Gendreau PL, Tremblay RE, Oligny P. Reactive and proactive aggression differentially predict later conduct problems. *Journal of Child Psychology and Psychiatry*. 1998; 39:377–385. [PubMed: 9670093]
- Youngstrom E, Loeber R, Stouthamer-Loeber M. Patterns and correlates of agreement between parent, teacher, and male adolescent ratings of externalizing and internalizing problems. *Journal of Consulting and Clinical Psychology*. 2000; 68:1038–1050. [PubMed: 11142538]

Figure 1.

Standardized squared estimates from the best fitting one-factor independent pathway model are displayed.

Note. For simplicity, only one twin in a pair is shown. In the model, genetic and nonshared environmental effects are represented in two ways: general (common among subscales) and specific (unique to each subscale) effects. Latent variables are depicted in circles, A is common additive genetic factor and E is common nonshared environmental factor; A_s (additive genetic) and E_s (nonshared environmental effects) are residual variances specific to each measure. Measured variables are depicted in rectangles (manipulative & deceitful, callous & disinhibited, reactive and proactive aggression). One-factor independent pathway model of psychopathic personality traits and aggression in both caregiver reports and child's self-reports (child self-report parameter estimates are presented in parentheses).

* $p < .05$.

Table 1

Number of Participants (*N*), Means and Standard Deviations for Child Psychopathy Scale (CPS) Factors, Reactive and Proactive Aggression, by Zygosity and Sex

	Callous & disinhibited			Manipulative & deceitful			Reactive aggression			Proactive aggression		
	M	SD	N	M	SD	N	M	SD	N	M	SD	N
Caregiver report												
MZ male	1.23	0.17	277	1.29	0.18	277	0.70	0.32	277	0.09	0.15	277
DZ male	1.27	0.19	168	1.32	0.17	168	0.75	0.36	168	0.14	0.22	168
MZ female	1.20	0.14	278	1.26	0.17	278	0.61	0.30	278	0.08	0.13	278
DZ female	1.19	0.16	195	1.27	0.18	195	0.65	0.36	195	0.09	0.16	195
DZ OS male	1.26	0.18	147	1.30	0.19	147	0.69	0.30	147	0.10	0.15	147
DZ OS female	1.18	0.14	151	1.28	0.20	151	0.66	0.33	151	0.08	0.12	151
Twins' self-report												
MZ male	1.28	0.14	273	1.16	0.15	273	0.67	0.35	277	0.09	0.14	277
DZ male	1.28	0.14	168	1.16	0.17	168	0.65	0.34	168	0.10	0.20	168
MZ female	1.13	0.14	277	1.25	0.13	277	0.60	0.32	278	0.06	0.13	278
DZ female	1.25	0.13	195	1.14	0.14	195	0.59	0.31	193	0.05	0.11	193
DZ OS male	1.29	0.14	146	1.18	0.16	146	0.66	0.30	146	0.09	0.01	146
DZ OS female	1.26	0.13	148	1.13	0.15	148	0.62	0.32	151	0.06	0.01	151

Note. Means and standard deviations displayed are on raw (untransformed) data. CPS scales range from 1 to 2, and Reactive Proactive Questionnaire scales range from 0 to 2. DZ = dizygotic; MZ = monozygotic; OS = DZ opposite sex.

Table 2

Phenotypic and Twin Correlations Between the Two Child Psychopathy Scale Factors and Reactive and Proactive Aggression (Caregiver Reports)

Phenotypic correlations				
	Callous & disinhibited		Manipulative & deceitful	
Reactive aggression		.51 *		.50 *
Proactive aggression		.43 *		.47 *
Intraclass and cross-twin cross-trait correlations				
	Callous & disinhibited	Manipulative & deceitful	Reactive aggression	Proactive aggression
MZ male				
Callous & disinhibited	<u>.63</u> *	.24 *	.20 *	.22 *
Manipulative & deceitful		<u>.50</u> *	.31 *	.24 *
Reactive aggression			<u>.48</u> *	.33 *
Proactive aggression				<u>.61</u> *
DZ male				
Callous & disinhibited	<u>.11</u>	-.07	.13	.10
Manipulative & deceitful		<u>.02</u>	.07	.08
Reactive aggression			<u>.35</u> *	.17 *
Proactive aggression				<u>.34</u> *
MZ female				
Callous & disinhibited	<u>.53</u> *	.23 *	.24 *	.20 *
Manipulative & deceitful		<u>.58</u> *	.35 *	.30 *
Reactive aggression			<u>.60</u> *	.41 *
Proactive aggression				<u>.57</u> *
DZ female				
Callous & disinhibited	<u>.30</u> *	.21 *	.26 *	.18 *
Manipulative & deceitful		<u>.35</u> *	.37 *	.32 *
Reactive aggression			<u>.46</u> *	.38 *
Proactive aggression				<u>.48</u> *
DZ OS				
Callous & disinhibited	<u>.26</u> *	.18 *	.26 *	.25 *
Manipulative & deceitful		<u>.27</u> *	.23 *	.24 *
Reactive aggression			<u>.50</u> *	.35 *
Proactive aggression				<u>.55</u> *

Note. Intraclass correlations (presented with an underline) for parent reports of psychopathic personality traits and aggression have previously been reported on these data, please see text. DZ = dizygotic; MZ = monozygotic; OS = DZ opposite sex.

^{*} $p < .05$.

Table 3

Phenotypic and Twin Correlations Between the Two Child Psychopathy Scale Factors and Reactive and Proactive Aggression (Twin Self-Reports)

Phenotypic correlations				
	Callous & disinhibited		Manipulative & deceitful	
Reactive aggression		.45 [*]		.39 [*]
Proactive aggression		.46 [*]		.46 [*]
Intraclass and cross-twin cross-trait correlations				
	Callous & disinhibited	Manipulative & deceitful	Reactive aggression	Proactive aggression
MZ male				
Callous & disinhibited	<u>.39⁻</u>	.36 [*]	.31 [*]	.38 [*]
Manipulative & deceitful		<u>.42⁻</u>	.21 [*]	.38 [*]
Reactive aggression			<u>.39⁻</u>	.49 [*]
Proactive aggression				<u>.46⁻</u>
DZ male				
Callous & disinhibited	<u>.17</u>	.58 [*]	.48 [*]	.59 [*]
Manipulative & deceitful		<u>.11</u>	.35 [*]	.58 [*]
Reactive aggression			<u>.28⁻</u>	.56 [*]
Proactive aggression				<u>.26⁻</u>
MZ female				
Callous & disinhibited	<u>.34⁻</u>	.37 [*]	.38 [*]	.45 [*]
Manipulative & deceitful		<u>.25⁻</u>	.28 [*]	.37 [*]
Reactive aggression			<u>.35⁻</u>	.47 [*]
Proactive aggression				<u>.12</u>
DZ female				
Callous & disinhibited	<u>.08</u>	.39 [*]	.42 [*]	.33 [*]
Manipulative & deceitful		<u>.23⁻</u>	.47 [*]	.52 [*]
Reactive aggression			<u>.40⁻</u>	.47 [*]
Proactive aggression				<u>.17</u>
DZ OS				
Callous & disinhibited	<u>.13</u>	.52 [*]	.46 [*]	.42 [*]
Manipulative & deceitful		<u>.04</u>	.43 [*]	.46 [*]
Reactive aggression			<u>.06</u>	.57 [*]
Proactive aggression				<u>.11</u>

Note. Intraclass twin correlations are underlined. Twin correlations for reactive and proactive aggression have been reported on these data (see Baker, Raine, Liu, & Jacobson, 2008). DZ = dizygotic; MZ = monozygotic; OS = DZ opposite sex.

*
p < .05.

Table 4

Genetic and Environmental Correlations Between Child Psychopathy Scale Factors and Reactive and Proactive Aggression, Caregiver and Twin Self-Reports

	Caregiver reports		Twin self-reports	
	Genetic correlation (r_g)	Nonshared environmental correlation (r_e)	Genetic correlation (r_g)	Nonshared environmental correlation (r_e)
Callous & disinhibited—reactive aggression	.49 (.06–.81)	.47 (.38–.55)	.59 (.42–.73)	.37 (.28–.45)
Callous & disinhibited—proactive aggression	.41 (.00–.72)	.37 (.27–.47)	.76 (.58–.94)	.30 (.21–.39)
Manipulative & deceitful—reactive aggression	.57 (.17–1.0)	.34 (.23–.43)	.57 (.39–.74)	.29 (.20–.38)
Manipulative & deceitful—proactive aggression	.35 (–.20–.62)	.41 (.31–.50)	.81 (.64–.98)	.29 (.20–.38)

Note. The 95% confidence intervals are within parentheses.

Model Comparisons for Multivariate Genetic Analyses for the Two Child Psychopathy Scale Factors With Reactive and Proactive Aggression, Caregiver and Twin Self-Reports

Table 5

Model	Overall fit						Model comparison				
	-2LL	df	AIC	BIC	χ^2	df	p	$\Delta\chi^2$	Δdf	p	Comparison model
Caregiver reports											
1	Saturated model ^a	11,708.90	4,644	2,420.90	-9,045.20						
2	Cholesky $m \neq f$	11,858.89	4,796	2,266.89	-9,457.88	149.99	152	.531	—	—	—
3	One-factor IP $m \neq f$	11,878.18	4,808	2,262.18	-9,486.73	169.28	164	.354	19.29	12	.08 Model 2
3a	One-factor IP $m = f$ All (common + specifics)	11,909.33	4,832	2,245.33	-9,548.16	200.43	188	.254	31.15	24	.15 Model 3
3b	One-factor IP $m = f$ All (common + specifics; drop C in common)	11,921.60	4,836	2,249.60	-9,554.86	212.70	192	.146	12.27	4	.02 Model 3a
3c	One-factor IP $m = f$ All (common + specifics); drop C in common + specifics	11,923.91	4,840	2,243.91	-9,566.54	215.01	196	.167	14.58	8	.07 Model 3a
3e	One-factor IP $m = f$ Drop A in common	11,921.46	4,836	2,249.46	-9,554.93	212.56	192	.147	12.13	4	.02 Model 3a
3f	One-factor IP $m = f$ Drop A in common + specifics	11,970.15	4,840	2,290.15	-9,543.42	261.25	196	.001	60.82	8	<.01 Model 3a
Twin self-reports											
1	Saturated model ^a	12,181.83	4,620	2,941.83	-8,731.74						
2	Cholesky $m \neq f$	12,340.69	4,772	2,796.69	-9,139.98	158.86	152	.34	177.56	164	.22 —
3	One-factor IP $m \neq f$	12,359.39	4,784	2,791.39	-9,169.13	177.56	164	.22	—	—	Model 1
3a	One-factor IP $m = f$ All (common)	12,379.87	4,796	2,787.87	-9,197.39	198.04	176	.12	20.48	12	.08 Model 3
3b	One-factor IP $m = f$ All (common + specifics)	12,412.47	4,808	2,796.47	-9,219.59	230.64	188	.02	230.64	188	.02 Model 1
3c	One-factor IP $m = f$ All (common); drop C in common	12,383.08	4,800	2,783.08	-9,208.62	201.25	180	.13	201.25	180	.13 Model 1
3d	One-factor IP $m = f$ All (common); drop C in common + specifics	12,389.03	4,808	2,773.03	-9,231.31	207.20	188	.16	9.16	12	.10 Model 3a

Model	Overall fit				Model comparison				Comparison model
	-2LL	df	AIC	BIC	χ^2	df	p	$\Delta\chi^2$	p
3f One-factor IP $m = f$ Drop A in common	12,393.06	4,800	2,793.06	-9,203.63	211.23	180	.055	13.19	<.001
								4	Model 3a

Note. Best fitting models are presented in bold. $-2LL = -2(\log\text{-likelihood})$; AIC = Akaike's information criterion; BIC = Bayesian information criterion; IP = independent pathway; $\Delta\chi^2$ = difference in log-likelihoods between nested model.

^a All models compared with Model 1 unless otherwise specified.

Appendix

Cross-Report (Phenotypic) Correlations Among the Two Psychopathy Factors and Reactive and Proactive Aggression

Twin self-reports	Caregiver reports			
	Callous and disinhibited	Manipulative and deceitful	Reactive aggression	Proactive aggression
Callous and disinhibited	.24**	.06*	.15**	.12**
Manipulative and deceitful	.18**	.14**	.10**	.13**
Reactive aggression	.16**	.16**	.22**	.12**
Proactive aggression	.18**	.15**	.16**	.20**

Note. Correlations presented are on nontransformed data.

*
 $p < .05$.

**
 $p < .01$.