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# Psychopathic Personality Traits and Environmental Contexts: Differential Correlates, Gender Differences, and Genetic Mediation

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## Abstract

Theorists have speculated that primary psychopathy (or Factor 1 affective-interpersonal features) is prominently heritable whereas secondary psychopathy (or Factor 2 social deviance) is more environmentally determined. We tested this differential heritability hypothesis using a large adolescent twin sample. Trait-based proxies of primary and secondary psychopathic tendencies were assessed using Multidimensional Personality Questionnaire (MPQ; Tellegen & Waller, 2008) estimates of Fearless Dominance and Impulsive Antisociality, respectively (Benning et al., 2005). The environmental contexts of family, school, peers, and stressful life events were assessed using multiple raters and methods. Consistent with prior research, MPQ Impulsive Antisociality was robustly associated with each environmental risk factor, and these associations were significantly greater than those for MPQ Fearless Dominance. However, MPQ Fearless Dominance and Impulsive Antisociality exhibited similar heritability, and genetic effects mediated the associations between MPQ Impulsive Antisociality and the environmental measures. Results were largely consistent across male and female twins. We conclude that gene-environment correlations rather than main effects of genes and environments account for the differential environmental correlates of primary and secondary psychopathy.

### Keywords

primary psychopathy; secondary psychopathy; environmental risk; genetics; heritability

Theory and empirical research has identified meaningful variants of psychopathy as reflected in subtypes (e.g., primary and secondary psychopathy; Karpman, 1941) and structural models that have identified distinguishable factors underlying psychopathic personality features (e.g., Factor 1 [F1] affective-interpersonal features and Factor 2 [F2] social deviance; Benning et al., 2003; Harpur et al., 1989). A prominent hypothesis based on

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this research is that psychopathy variants differ in their etiological underpinnings, specifically, that F1/primary psychopathy[1] is more heritable whereas environmental factors contribute more to F2/secondary psychopathy as evidenced by the latter's stronger associations with putative environmental risk factors (for a review see Skeem et al., 2003). However, few studies have directly tested this hypothesis, and substantial behavioral genetic research indicates that (1) measures presumed to reflect environmental risk exhibit robust heritability (Kendler & Baker, 2007; Plomin & Bergeman, 1991), and (2) genetic effects account for much of the covariance between measures of environmental risk and indices of personality and psychopathology (Plomin, 1995; Saudino et al., 1997). Using trait-based proxies of primary and secondary psychopathy, we utilized a large, mixed-gender sample of adolescent twins to test whether common genetic factors could account for the differential associations between psychopathic traits and environmental correlates.

## Psychopathy Variants

Theories of primary and secondary psychopathy variants trace back to Karpman (1941) and have since been elaborated on by several investigators (Blackburn, 1975; Fowles, 1980; Lykken, 1995; Mealey, 1995; Porter, 1996). The two variants are similar in terms of exhibiting irresponsible, conning, aggressive, and generally antisocial behavior, but differ in terms of etiology and motivation. Primary psychopathy is conceptualized as stemming from a constitutional (i.e., heritable) affective deficit resulting in an incapacity for normal human emotions and attachments, reflected in callousness and absence of guilt, limited feelings of fear or anxiety, and predatory or instrumental antisocial behavior. In contrast, secondary psychopathy is often conceptualized as a disturbance in emotional and behavioral control arising from adverse environmental influences such as abuse, poor parenting, rejection, or neglect. Secondary psychopaths are described as being hostile, impulsive, and reactive in their antisocial and violent behavior, and as experiencing heightened levels of anxiety and negative emotions. Recent empirical studies of male and female prisoner samples have identified subgroups that are broadly consistent with these theoretical accounts of primary and secondary psychopathy (Hicks et al., 2004, 2010; Poythress et al., 2010a; Skeem et al., 2007).

In contrast to subtype models, the majority of research has relied on trait-based models to examine heterogeneity in psychopathy. While different instruments have been developed to assess psychopathic features in somewhat different ways, most psychopathy measures can be organized along the primary versus secondary distinction. For example, both the Psychopathy Checklist-Revised (PCL-R; Hare, 2003), an interview-based clinical rating primarily used in correctional settings, and the Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996), a self-report inventory designed for community samples, emphasize a two-factor structure whose content and correlates are consistent with descriptions of primary and secondary psychopathy. PCL-R Factor 1 (F1) incorporates the interpersonal and affective features most closely associated with primary psychopathy, while PCL-R Factor 2 (F2) reflects the impulsivity, reactive aggression, and negative emotionality associated with secondary psychopathy (Harpur et al., 1989). Alternatively, the PPI factors of Fearless Dominance (FD) and Impulsive Antisociality (IA) serve as markers of primary and secondary psychopathy, respectively (Benning et al., 2003), though FD is limited to

[1]We recognize there are several partially overlapping conceptualizations of psychopathy as well alternative measurement strategies. The term "F1/primary psychopathy" refers to a broad content domain of symptoms and traits associated with the affective and interpersonal features of psychopathy including callousness, lack of remorse, superficial charm, narcissism, conning and manipulative interpersonal behavior, social dominance, low anxiety, fearlessness, and descriptions of the primary psychopathy subtype. The term "F2/secondary psychopathy" refers to a related but distinct set of symptoms and traits associated with the social deviance features of psychopathy including impulsivity, irresponsibility, child and adult antisocial and criminal behavior, aggression, disagreeableness, rebelliousness, and descriptions of secondary psychopathy.

interpersonal traits (social dominance, stress immunity, fearlessness) and does not include the affective features (callousness, lack of remorse) of primary psychopathy. Additionally, several investigators have shown that normal range personality inventories such as the Multidimensional Personality Questionnaire (MPQ; Benning et al., 2005) and Big Five measures (Ross et al., 2008; Witt et al., 2009) can provide valid measures of FD and IA.

Both PCL-R and PPI factors exhibit distinct patterns of external correlates consistent with primary and secondary psychopathy subtypes (especially when controlling statistically for the common variance between PCL-R factors; Poythress et al., 2010b). PCL-R F1 and FD show selective relations with interpersonal dominance and narcissism, and negative associations with measures of anxiety, fear, internalizing psychopathology, and suicidal behavior (Benning et al., 2003, 2005; Blonigen et al., 2005, 2010; Hall et al., 2004; Harpur et al., 1989; Hicks & Patrick, 2006; Kennealy et al., 2007; Poythress et al., 2010b; Verona et al., 2001, 2005). Conversely, PCL-R F2 and IA show selective positive relations with impulsivity, measures of negative emotionality (distress, fear, and anger), alcohol and drug abuse, externalizing psychopathology, reactive aggression, and suicidal behavior (Benning et al., 2003, 2005; Blonigen et al., 2005, 2010; Hall et al., 2004; Harpur et al., 1989; Kennealy et al., 2007; Patrick et al., 1997, 2006; Poythress et al., 2010b; Verona et al., 2001, 2005).

## Environmental Risk Factors for Antisocial Behavior and Psychopathy

In addition to personality and clinical correlates, a large literature has documented numerous environmental correlates of antisocial behavior and psychopathy (for a recent review see Farrington et al., 2010). Patterson and colleagues (Granic & Patterson, 2006; Patterson et al., 1989) have proposed a prominent theory that helps account for the emergence and persistence of antisocial behavior and many its environmental correlates; in particular, noting that the development of chronic antisocial behavior is often marked by a typical sequence. First, ineffective parenting and family management practices coupled with child temperament characteristics result in child conduct problems. Next, conduct problems contribute to academic failure and rejection by prosocial peers. Failure to integrate with these socializing agents then increases the risk for depressed mood and deviant peer affiliation that then increases the likelihood of involvement in drug use and delinquency. These person-level risk factors are also correlated with broader contextual variables such as family money and legal problems, residential instability, parental conflict and divorce, and neighborhood characteristics such as poverty and crime (Appleyard et al., 2005; Deater-Deckard et al., 1998; Hawkins et al., 1992). These risk factors continue to build upon one another over time such that an outcome in one developmental stage can serve as a risk factor at a later stage. For example, academic failure is an outcome of conduct problems in childhood, which then creates a context and serves as a risk factor for deviant peer affiliation and continued antisocial behavior in adolescence. The accumulating interplay of environmental and person-level risk then begins to limit the available contexts for antisocial youth; a dynamic process referred to as “cascading constraints” (Granic & Patterson, 2006) or “canalization” (Waddington, 1966). In turn, these constraints then greatly increase the probability of poor outcomes such as school expulsion and educational disengagement, unemployment, criminal activity, and arrest.

Interestingly, PCL-R and PPI factors exhibit differential associations with these environmental correlates. PCL-R F2 and IA show consistent associations with childhood abuse and trauma, negative parenting, delinquent peers, and negative associations with quality of family life, income, SES, educational attainment, and father’s occupational status (Benning et al., 2003; Hall et al., 2004; Hare, 2003; Hare, McPherson, & Forth, 1988; Harpur et al., 1989; Lynam et al., 2010; Patrick et al., 1997; Poythress et al., 2006; Viding et

al., 2009). In contrast, PCL-R F1 and FD are uncorrelated with many environmental risk factors and have exhibited modest positive correlations with some including SES and educational attainment (Benning et al., 2003; Hall et al., 2004). Consequently, some investigators have interpreted the stronger associations between environmental risk factors and PCL-R F2 as being consistent with conceptions that secondary psychopathy is more environmentally determined while primary psychopathy is mostly a function of genetic factors (Skeem et al., 2007).

## Genetic Mediation of the Association between Psychopathy Facets and Environmental Risk Factors

Contrary to theories of greater heritability of primary psychopathy, however, twin studies using FD and IA measures have reported comparable heritability estimates in the .40 to .50 range (Blonigen et al., 2005). Moreover, other constructs strongly linked to secondary psychopathy such as the impulsivity and irresponsible behavior factor of the Youth Psychopathy Inventory (Larsson et al., 2006), antisocial behavior/criminality (Rhee & Waldman, 2002), and externalizing psychopathology (Krueger et al., 2002) have reported heritability estimates that are comparable to or larger than indices of traits related to primary psychopathy.

The concept of gene-environment (G-E) correlation may help to reconcile the finding of comparable heritability for different facets psychopathy along with their differential associations with putative environmental risk factors. G-E correlations refer to the phenomenon of non-independence between a person's genotype and their environmental experiences, that is, a person's genotype influences the degree of exposure to environmental risk factors (Plomin et al., 1977; Scarr & McCartney, 1983). G-E correlations can be *passive* as in the case of parents providing a child with both the genes and environment that favor the development of a particular trait. For example, the genetic risk factors associated with antisocial behavior are also associated with poor parenting (Wade & Kendler, 2000). As such, children of antisocial parents receive a "double whammy" in terms of inheriting both genes and a rearing environment that increases risk for antisocial behavior (Jaffe et al., 2003). G-E correlations can also be *active* (person seeks out certain environments) and *evocative* (person elicits certain responses from the environment). As already discussed, child conduct problems can instigate a cascade of events that increases exposure to environmental risk factors including increased conflict and weakened attachment with parents, academic failure and disengagement, stronger affiliation with deviant peers, early substance use, and involvement in criminal activity (Granic & Patterson, 2006).

Incorporating genetic risk and G-E correlations helps to expand these theories by stipulating factors that contribute to individual differences in both child and parent characteristics that underlie initial child management problems and ineffective parenting practices. G-E correlations also help to account for why nearly every putatively "environmental" measure exhibits heritability (Kendler & Baker, 2007; Plomin & Bergeman, 1991). That is, genetically-influenced characteristics such as personality, intelligence, and interests help to shape a person's environment, including their exposure to environmental risk. This fundamental non-independence between person-level and putative environmental variables further emphasizes the arbitrary nature of what is a risk factor (e.g., parent-child relationship) or an outcome (e.g., academic failure, arrest) as both are influenced by common genetic and environmental factors.

Our hypothesis then is that G-E correlations might also account for the differential associations between environmental risk factors and psychopathy facets. That is, the unique genetic risk factors associated with each psychopathy facet might result in differential

exposure to environmental risk factors. Put another way, certain genetic effects might act as common risk factors for both psychopathic personality traits and environmental risk. This would be a case of genetic mediation and can be tested by estimating the genetic correlation between psychopathic personality traits and an environmental risk factor. For example, in a twin design, this is done by correlating the psychopathic personality traits of twin A with the environmental risk exposure of twin B. If the correlation is greater for monozygotic (MZ) twins (who share all their genes) compared to dizygotic (DZ) twins (who share on average 50% of their segregating genes) then it could be inferred that common genetic risk factors account for the association between psychopathic personality traits and the environmental variable.

## Gender Differences

Another topic we wished to address was the potential role of gender in the etiology of psychopathy, as suggested by several previous findings. First, there are consistent and relatively large mean-level gender differences in psychopathy and antisocial behavior (Bolt et al., 2004; Moffitt et al., 2001). Second, highly antisocial women (e.g., female prisoners) exhibit greater levels of environmental deprivation, victimization, and mental health problems relative to their male counterparts (Maden et al., 1994; McClellean et al., 1997; Mulder et al., 1994; Teplin et al., 2002). This has lead some investigators to speculate that women require a greater “loading” of risk factors to exhibit psychopathic features or that environmental factors play a greater role in female relative to male psychopathy and antisocial behavior (Jordan et al., 1996; McClellan et al., 1997; Teplin et al., 2002; Warren et al., 2002). In contrast, Moffitt et al. (2001) analyzed data from a large epidemiological sample and concluded that the lower rates of antisocial behavior in women were primarily attributable to women experiencing lower levels of the risk factors for antisocial behavior (at least at the population level). Also, in terms of correlates, previous studies have found that the associations between psychopathy facets and various criterion variables are largely consistent across men and women (Benning et al., 2005; Blonigen et al., 2005; Kennealy et al., 2007).

Though the study of female psychopathy has increased dramatically in recent years (Verona et al., 2010), few studies have been able to make direct gender comparisons by virtue of having comparably and sufficiently sized samples of males and females to test these hypotheses in a single study; an advantage of our large, community-based sample. Specifically, we examined (1) mean-level gender differences in psychopathic traits and exposure to environmental risk factors, (2) gender differences in the heritability of psychopathic traits and genetic control of exposure to environmental risk, (3) gender differences in the associations between psychopathic traits and environmental risk factors, and (4) gender differences in the genetic and environmental contributions to the associations between psychopathic traits and environmental risk factors.

## Current Study

Our goal was to extend current etiological models of psychopathy variants by incorporating mechanisms of gene-environment interplay; specifically, by testing whether genetic risk could account for the differential pattern of correlates between psychopathy facets and putative environmental risk factors. To do so, we utilized a large, mixed-gender sample of adolescent twins. Adolescence is an interesting developmental period to examine this question as people are taking an increasingly active role in shaping their environments, but are still strongly influenced by the environmental context of their family of origin. We focused on environmental contexts that have been most consistently associated with antisocial behavior and psychopathy—family, school, and peers—using multiple informants

and methods. We also examined associations with stressful life events, as personal characteristics such as personality and psychopathology are associated with an increased likelihood of experiencing such events (Kendler et al., 2003; Saudino et al., 1997). Stressful life events include family-level stressors such as divorce, money and legal problems, as well as life events that are to some extent dependent on a person's behavior such as school (e.g., suspension, expulsion) and legal (e.g., arrest) problems. These latter events are important criterion outcomes in defining the constructs of psychopathy and antisocial behavior. Given the nature of our sample (i.e., adolescent twins from the community) and availability of measures, psychopathy variants were assessed using MPQ estimates of FD and IA. Although these constructs represent a variable-centered approach to measuring primary and secondary psychopathy subtypes, their pattern of external correlates suggest that they capture a similar nomological network as these variants and thus may be considered viable trait-based proxies of primary and secondary psychopathy. Our primary hypotheses were the following:

1. MPQ-IA would exhibit a robust association with each environmental risk factor. MPQ-FD would either be uncorrelated or exhibit a modest positive association with environmental advantage.
2. Common genetic risk factors would primarily account for the association between MPQ-IA and each environmental risk factor.

## Method

### Participants

The sample consisted of male and female twins participating in the Minnesota Twin Family Study (MTFS), an epidemiological-prospective study investigating the development of substance use disorders (Iacono et al., 1999; Iacono et al., 2006). The MTFS includes two age cohorts with participants entering the study at either age 11 or 17. Participants are then given the opportunity to return for follow-up assessments every 3–4 years. To maximize our sample size, we focused on data collected at the age 17 assessment for both cohorts (i.e., the intake assessment for the 17-year old cohort and the second follow-up assessment for the 11-year old cohort). In terms of recruitment, all families that included a twin birth in the state of Minnesota between 1972 and 1984 were identified using publicly available birth records and databases. Over 90% of families were successfully located for each target birth year. The only exclusionary criteria were that families had to live within a one-day drive of the University of Minnesota laboratories and neither twin could have an intellectual or physical disability that would preclude full participation in the assessment. Seventeen percent of eligible families declined participation. Based on a survey completed by over 80% of non-participating families, parents in the participating families differed only slightly in terms of educational attainment (i.e., 0.25 years more education), but did not differ in terms of history of mental health problems or treatment. Consistent with the demographics of Minnesota for the target birth years, 96% of the participants are non-Hispanic White. The final sample for the age 17 assessment included 2604 twins (1239 male, 1365 female).

### Assessment

**Psychopathic personality traits—**As part of the age 17 assessment, each twin completes the 198-item version of the MPQ (Tellegen & Waller, 2008). Regression weights from Benning et al. (2003) were applied to scores on the 11 primary scales of the MPQ to derive estimates of FD and IA. Correlations between MPQ and PPI FD and IA factors are typically > .70 (Benning et al., 2003; Witt et al., 2009). Previous reports using the MTFS twin sample have demonstrated that MPQ-FD and IA scores exhibit a theoretically coherent pattern of associations with internalizing and externalizing disorders (Benning et al., 2005;

Blonigen et al., 2005), developmental change and stability (Blonigen et al., 2006), and heritability (Blonigen et al., 2005). Results were unchanged when item sums were used rather than regression weighted scores (Blonigen et al., 2006).

**Family Environment: Parent-Child Relationship—**The Parental Environment Questionnaire (PEQ; Elkins et al., 1997) was used to assess parent-child relationship problems. The PEQ is a 50-item self-report questionnaire that assesses multiple dimensions of the parent-child relationship (e.g., conflict, involvement parent's regard for child, child's regard for parent; scale α's range from .82 to .69). Each twin completes separate PEQ ratings describing their relationship with each parent. Parents also rate the quality of their relationship with each twin as well as the quality of the relationship between each twin and the other parent (e.g., mother rated the relationship between each twin and their father). As such, up to 3 ratings were available for the mother-child and father-child relationship. The PEQ scales exhibit a dominant 1st factor that resembles the warmness/responsiveness dimension typically identified in measures of parenting and parent-child relationship measures (the other dimension being control/demandingness; Maccoby & Martin, 1983). Composites of mother-child and father-child relationship problems were calculated by taking the mean of the 3 informant ratings on the first principal component of the PEQ scales (mean correlation across informants was .41).

**Social Environment: Peer Affiliation—**Twin and teacher reports were used to assess peer affiliation. Twins completed a 19-item questionnaire assessing antisocial (e.g., my friends smoke, drink alcohol, steal, get in fights; α = .85) and prosocial (e.g., my friends work hard in school, popular with other kids, liked by teachers; α = .78) peer affiliation. Up to 3 teachers nominated by the twin also completed similar ratings regarding the twin's antisocial (α = .85) and prosocial (α = .87) peer affiliation (average inter-rater reliability was .71 across teacher ratings). The mean z-score of the twin and teacher reports ($r$ = .40) was used to calculate composite measures of antisocial and prosocial peer affiliation.

**School Environment: Academic Achievement and Engagement—**A composite of academic achievement and engagement was calculated using twin and mother reports of cumulative grade point average (GPA; $r$ = .80 between twin and mother reports and $r$ = .89 with school transcripts), self and maternal ratings of expectations regarding each twin's ultimate educational attainment (e.g., complete high school, bachelor's degree; $r$ = .64 between twin and mother ratings), and a 7-item scale (α = .83) completed by the twin and mother assessing each twin's attitudes and engagement in school (e.g., good attitude about school, enjoys attending school). The academic engagement and achievement composite was calculated by taking the mean z-score for ratings of GPA, academic expectations, and academic attitudes across twin and mother reports ($r$ = .77).

**Stressful Life Events: School and Legal Problems—**The Life Events Interview (Bemmels et al., 2008) was used to assess a number of potentially stressful experiences in the life of each twin. We focused on domains most relevant to psychopathic personality traits during late adolescence, namely, events related to school and legal problems. School problems included failing a class, being held back a year in school, being required to attend summer school, worried about how he or she was doing in school, and being suspended or expelled. Legal problems included getting into trouble because of the use of alcohol or drugs, trouble with police for traffic violations, trouble with police other than for traffic violations, had to go to court, and sent to a juvenile detention center. Life events such as these are called dependent life events, that is, the occurrence of these events is to some extent dependent on the behavior of the person who experiences them (Masten et al., 1994).

School and legal problems were summed to calculate an overall index of dependent life events. Results were unchanged when school and legal problems were analyzed separately.

We also calculated an index of family-level or independent stressful life events that included 18 items covering parental discord and divorce and family money, legal, and mental health problems. Because twins are necessarily concordant on these items, the family-level stressful life events can not be used in the heritability analyses, but do provide an additional measure to validate the differential associations between the environmental measures and psychopathy facets.

## Data Analysis

For the phenotypic analyses, MPQ-FD and MPQ-IA were entered into a regression model predicting each environmental measure. The models were multigroup regression models based on gender, and fit in Mplus 5 (Muthen & Muthen, 2007) using a maximum likelihood estimator with robust standard errors and the cluster option to account for the correlated twin observations while also accommodating missing data. First, regression coefficients were estimated and allowed to vary across groups. Second, the regression coefficients were constrained to be equal for MPQ-FD and MPQ-IA (but allowed to vary across gender) with the change in model fit (distributed as a $\chi^2$ difference test) used to test whether there was a significant difference in their association with each environmental variable. Finally, regression coefficients were constrained to be the same across gender (but allowed to vary for MPQ-FD and MPQ-IA) to test for gender differences. Because our large sample size provided substantial power to detect effects, only effects with $p < .001$ are reported as statistically significant for the phenotypic analyses.

Genetic and environmental influences on the variance and covariance among psychopathic personality traits and the environmental measures were estimated by fitting standard biometric models. These models assume that differences in the proportion of alleles shared between MZ and DZ twin pairs are reflected in differences in phenotypic similarity as measured by the MZ and DZ twin correlations for a given trait. Differences in the twin correlations are then used to decompose the variance of a trait into additive genetic ($a^2$), shared environmental ($c^2$), and nonshared environmental ($e^2$) components. Additive genetic influences refer to the summation of individual gene effects across loci. MZ twins share 100% of their additive genetic effects while DZ twins share, on average, 50% of these effects. Genetic effects are inferred if $r_{MZ} > r_{DZ}$ with $r_{MZ} = 2r_{DZ}$ consistent with twin similarity being solely due to additive genetic effects. Shared environmental effects refer to environmental influences that contributes to twin similarity and are inferred if $r_{DZ} > ½\ r_{MZ}$. Nonshared environmental effects refer to environmental influences that contribute to differences among members of a twin pair (including measurement error) and are inferred if $r_{MZ} < 1$.

Our primary goal was to determine the extent to which common genetic effects accounted for the association between psychopathic personality traits and the environmental variables. Biometric models called Cholesky decompositions were fit to each bivariate association between psychopathic personality traits and the environmental variables. This model parses both the individual variance of each phenotype and the covariance between phenotypes into their respective genetic and environmental components. Figure 1 provides a graphical representation of a bivariate Cholesky decomposition with MPQ-IA and antisocial peers as exemplary phenotypes. The latent $A_1$, $C_1$, and $E_1$ variables represent the additive genetic, shared and nonshared environmental effects on MPQ-IA with the paths $a_{11}$, $c_{11}$, and $e_{11}$ being factor loadings indicating the amount of variance of MPQ-IA attributable to $A_1$, $C_1$, and $E_1$. The paths $a_{21}$, $c_{21}$, and $e_{21}$ are estimates of the variance in antisocial peers attributable to $A_1$, $C_1$, and $E_1$, that is, the extent to which genetic and environmental risk

factors on MPQ-IA also contribute to variance in antisocial peers. The latent $A_2$, $C_2$, and $E_2$ variables are the additive genetic and environmental effects that are unique to antisocial peers. The paths $a_{22}$, $c_{22}$, and $e_{22}$ are factor loadings that estimate the variance in antisocial peers attributable to $A_2$, $C_2$, and $E_2$. The model also makes it possible to parse the phenotypic covariance between the two phenotypes into additive genetic and environmental effects. Further, the product of $a_{11}$ x $a_{21}$ is the genetic covariance and can be standardized on the genetic variance of the two phenotypes to calculate the genetic correlation ($r_A$). The genetic correlation indexes the amount of overlapping genetic variance across the two phenotypes. Analogous procedures can be used to calculate shared ($r_C$) and nonshared environmental ($r_E$) correlations. All biometric analyses were conducted using the computer program *Mx* (Neale et al. 2004) using full information maximum likelihood, which easily incorporates participants with missing data. Due to the modest age heterogeneity in the sample ($M$ = 17.83 years, $SD$ = 0.69, range 16.55 to 20.34 years), all variables were regressed on age and age$^2$ prior to all analyses.

## Results

The descriptive statistics for the psychopathic personality traits and environmental variables are provided in Table 1. All scores derived from questionnaires were converted to a T-score metric to facilitate interpretation. The life events measures are reported as simple count of stressful life events. We tested for gender differences using linear mixed models in SPSS with a random intercept at the family level to account for the non-independence of the twin observations. Effect sizes are also reported as Cohen's *d*. There was a moderate to large gender difference on MPQ-FD and MPQ-IA with boys scoring higher on both. Boys also scored higher on antisocial peers, mother-child relationship problems, and school and legal problems. Girls scored higher on academic achievement and engagement and family-level problems. Boys and girls did not differ on prosocial peers or father-child relationship problems.

Table 2 reports the standardized regression coefficients for models with MPQ-FD and MPQ-IA entered as predictors of each environmental variable. MPQ-FD and MPQ-IA were uncorrelated for boys and girls, $r$ = .04 and $r$ = −.03, respectively. For both boys and girls, MPQ-IA was robustly correlated with each environmental variable in the direction of greater adversity. MPQ-FD was weakly and inconsistently related to the environmental variables. For girls, MPQ-FD had a small but significant effect in the direction of less environmental adversity for 4 of 7 environmental variables. For boys, MPQ-FD was unrelated to the environmental variables with the exception of a modest positive association with antisocial peers. For both boys and girls, the effect of MPQ-FD and MPQ-IA was significantly different for each environmental variable (the one exception was family-level problems in boys, though the difference nearly reached the alpha-level for significance, $p$ = .0018). In terms of gender differences, there were no significant differences in the effects of MPQ-FD or MPQ-IA on the environmental variables with the exception of antisocial peers. This gender difference was primarily due to a stronger association between MPQ-FD and antisocial peers in boys compared to girls, $\chi^2(1) = 13.47$, $p < .001$.

We also tested whether mean-level gender differences in psychopathic traits could be accounted for by greater exposure to environmental risk factors in males. To do so, we fit regression models in which the psychopathy traits were regressed on the 7 environmental measures and saved the predicted values: $R$ = .26 and .53, both $p < .001$, for MPQ-FD and MPQ-IA, respectively. We then fit a stepwise regression model for each psychopathy trait, with gender entered in step 1, and the predicted values using the environmental measures entered in step 2. For MPQ-FD, the gender $\beta$ = .30, $p < .001$ in step 1, and $\beta$ = .27, $p < .001$ in step 2, indicating that gender differences in mean-levels of the environmental risk

measures could not account for mean-level gender differences in MPQ-FD. For MPQ-IA, the gender $\beta = .22$, $p < .001$ in step 1, and $\beta = .13$, $p < .001$ in step 2, indicating that mean-level differences in the environmental risk measures accounted for slightly less than half of the mean-level gender difference in MPQ-IA.

The twin correlations and univariate ACE parameter estimates are presented in Table 3. For each variable, the MZ correlation was greater than the DZ correlation indicative of heritable effects. Results of the univariate twin models were consistent with the twin correlations and showed that each variable exhibited significant heritable variance for both boys and girls. Mother-child and father-child relationship problems, prosocial peers, and antisocial peers also exhibited shared environmental effects though the effects on the peer affiliation variables were statistically significant for girls only. The only significant gender difference was that the heritability estimate for antisocial peers was significantly greater for boys, $\chi^2(1) = 6.32$, $p = .01$.

The phenotypic, genetic, and environmental correlations between MPQ-FD and each environmental variable are provided in Table 4. Because both psychopathic personality traits exhibited virtually no shared environmental variance, we fixed the shared environmental effects on the psychopathic traits to be zero in the bivariate Cholesky models (however, we included shared environmental effects on the environmental variables). For both boys and girls, phenotypic associations between MPQ-FD and the environmental variables were small with some failing to reach statistical significance. If the phenotypic correlation was not significant, we did not estimate the percent of covariance attributable to genetic effects as this often leads to estimation problems and results that are unreliable and difficult to interpret.

For girls, MPQ-FD exhibited a small positive association with academic achievement and engagement and prosocial peers, and a small negative association with mother-child and father-child relationship problems. MPQ-FD accounted for 3.6% to 16.8% (calculated by squaring the genetic correlation) of the genetic variance in these four environmental variables. Additionally, the genetic correlation between MPQ-FD and (lack of) father-child relationship problems was significantly greater for girls, $\chi^2(1) = 5.45$, $p = .02$. Also, the phenotypic association between MPQ-FD and these environmental variables was primarily attributable to genetic effects (mean = 84%). None of the nonshared environmental correlations were significant.

For boys, the phenotypic associations between MPQ-FD and the environmental variables were mostly negligible, although four were statistically significant (i.e., the 95% confidence interval did not include zero). The only notable finding was a small positive association between MPQ-FD and antisocial peers. MPQ-FD accounted for 14.4% of the genetic variance in antisocial peers, and the phenotypic association was entirely attributable to genetic effects. The finding is also of note due to a significant gender difference as the genetic correlation between MPQ-FD and antisocial peers was greater for boys and in the opposite direction relative to girls, $\chi^2(1) = 7.49$, $p = .006$.

The phenotypic, genetic, and environmental correlations between MPQ-IA and each environmental variable are provided in Table 5. For both boys and girls, MPQ-IA exhibited robust phenotypic associations with each environmental variable in the direction of greater exposure to environmental risk. MPQ-IA accounted for 10.2% to 51.8% (mean = 24.0%) of the genetic variance in the environmental variables. For both boys and girls, the nonshared environmental correlation was also significant though of smaller magnitude than the genetic correlation. Genetic effects accounted for the majority of the phenotypic association

between MPQ-IA and the environmental variables (mean = 76%). There were no significant gender differences in the associations between MPQ-IA and the environmental variables.

## Discussion

Utilizing a large adolescent twin sample, we tested whether genetic risk in the form of GE correlations could account for the differential pattern of environmental correlates exhibited by F1/primary and F2/secondary psychopathy variants. A notable strength of our design was the inclusion of multiple measures of environmental risk factors based on reports from multiple informants, yielding a relatively comprehensive assessment of environmental context. Consistent with previous research, psychopathy variants as measured by MPQ-FD and MPQ-IA exhibited differential associations with each environmental risk factor. Specifically, MPQ-IA was robustly associated (and to a significantly greater degree than MPQ-FD) with major domains of environmental risk in adolescence including the contexts of family, peers, school, and stressful life events. In contrast, MPQ-FD was either unrelated or had a modest negative association with exposure to environmental risk, especially for girls. The one exception was a modest positive association between MPQ-FD and antisocial peers in boys. Also consistent with previous research, but inconsistent with the differential heritability hypothesis, MPQ-FD and MPQ-IA exhibited comparable heritability for both boys and girls. Finally, we extended the existing research literature by demonstrating that the associations between MPQ-IA and MPQ-FD and the environmental measures were primarily attributable to common genetic risk factors.

### Role of G-E Correlations in Psychopathy

The last finding can be conceptualized within the theoretical framework of G-E correlations and helps to account for the different environmental correlates of F1/primary and F2/secondary psychopathy. G-E correlation reflects the fact that a person's genotype can increase or decrease their exposure to environmental risk factors. For both MPQ-FD and MPQ-IA, genetic effects primarily accounted for their associations with the environmental measures—a case of genetic mediation. MPQ-FD and MPQ-IA were uncorrelated, and so their genetic risk factors are also independent. Therefore, the genetic factors associated with MPQ-IA also increase risk for general exposure to multiple forms of environmental adversity. In contrast, the genetic factors associated with MPQ-FD are largely independent of exposure to environmental risk and may even reduce exposure to a modest extent.

These findings help extend current theories of psychopathy and antisocial behavior in important ways. For example, Patterson et al. (1989) applied coercion theory to describe the disrupted parenting practices that lead to a persistent pattern of mutually hostile and permissive interactions that reinforces a child's antisocial tendencies. This pattern of disrupted parent-child relationships and antisocial tendencies then initiates a sequence of developmental processes leading to academic failure, peer rejection, deviant peer affiliation, drug use, and delinquency. Less discussed are factors that underlie the initial disrupted parent-child relationship such as a child's undercontrolled temperament or the parent's own antisocial traits that then influence his or her parenting practices (Moffitt, 2005). We propose that genetic factors are a likely source of such individual differences that then influence other putatively environmental variables such as parent-child relationship problems.

It is important to note that a G-E correlation does not mean genes have a direct effect on the probability of exposure to environments. Rather the effects of genes on environmental context are mediated by some other variable. In the current analysis, we infer that a person's psychopathic traits influence his or her environmental context though we did not test the mechanism of these effects. However, most of the environmental variables were proximal

and malleable and depend on person-situation transactions; thus, suggesting active and evocative G-E interplay. For example, parent-child and peer relationships entail reciprocal processes between a person's perception of the relationship, the behavior and perceptions he or she evokes from other people, and how those responses then modify prior perceptions. Given a genetic endowment that lends itself to a personality structure characterized by suspiciousness, aggressiveness and disagreeableness, and the tendency to be impulsive, irresponsible, and rebellious, it is clear how such personal characteristics could hamper positive relationships. Selection effects also play a role as people tend to associate with others of similar interests and personality, especially in the case of antisocial behavior and deviant peer affiliation in adolescence (Granic & Patteron, 2006; Kendler et al., 2008). Such dynamics are also at work in broader environmental contexts such as school, whereby interplay occurs between personality characteristics and the experience of academic failure, disengagement, and reciprocal interactions with teachers and other students. Finally, these person-situation transactions can lead to conflict with institutional powers such as school administration or the criminal justice system. Such events can have important long-term consequences depending on a person's subsequent behaviors—behaviors that are influenced by heritable personality traits.

Contrast such dynamics with a personality structure characterized by MPQ-FD traits. While there is debate about how well MPQ-FD maps onto alternative operationalizations of F1/primary psychopathy (e.g., as indexed by the PCL-R) due to modest correlations with other measures (Malterer et al., 2009; Poythress et al., 2010), it appears to tap psychopathic traits related to the concept of boldness, that is, high self-confidence and social efficacy, ability to remain calm under conditions of stress or threat and recover quickly from stressful events, venturesomeness, and tolerance for uncertainty (Patrick et al., 2009). As already reviewed, these traits are associated with lower levels of internalizing psychopathology, anxiety, fear, and suicidal behaviors (Benning et al., 2005; Blonigen et al., 2005, 2010; Hicks & Patrick, 2006; Verona et al., 2001, 2005). These traits also appear to provide a modest reduction or at least do not increase exposure to environmental risk. Again, these findings can be conceptualized in terms of person-situation transactions: across multiple contexts, individuals high in MPQ-FD traits select different environments, evoke different responses, and have different reactions to their experiences relative to high MPQ-IA individuals. Importantly, MPQ-FD traits likely help to mitigate the consequences of certain negative experiences whereas MPQ-IA traits likely exacerbate these problems. As a consequence, the subjective experience of high MPQ-FD versus high MPQ-IA individuals will differ markedly across multiple domains of their environment, differences that are likely to accumulate over time.

## Gender Differences

We also analyzed our data separately for male and female twins to test for any gender differences in psychopathic traits and their associations with environmental risk factors. First, we found that male twins had higher mean-levels of both psychopathic traits with moderate to large effects. Second, male twins also exhibited higher mean-levels for 4 of 7 environmental risk measures with modest to moderate effects. Third, mean-level gender differences in the environmental risk measures failed to account for the mean-level difference in MPQ-FD, but did account for nearly half the mean-level difference in MPQ-IA. The latter is partially consistent with Moffitt et al.'s (2001) interpretation that greater rates of antisocial behavior in males are due to a greater exposure to risk factors, but it fails to account for gender differences in F1/primary psychopathy. Fourth, the heritability of both MPQ-FD and MPQ-IA was comparable for male and female twins suggesting environmental factors do not play a more important role in female relative to male psychopathy, at least at the population level (though women may still require a greater

loading of risk factors to exhibit especially severe antisocial behavior that would result in incarceration).

Lastly, the correlates of MPQ-FD and MPQ-IA were largely consistent across male and female twins at both the phenotypic and genetic level, though there were a few subtle distinctions in regards to MPQ-FD. For one, there was a trend for MPQ-FD to exhibit stronger associations with less environmental risk in female twins. This was especially evident in the higher genetic correlations between MPQ-FD and father-child relationship problems (−), academic achievement and engagement (+), and prosocial peers (+) (though only the difference for father-child relationship problems was statistically significant). Also, both the phenotypic and genetic correlations between MPQ-FD and antisocial peers were significantly higher for male twins. These findings suggest that while MPQ-IA traits are expressed similarity across males and females, gender moderates the expression and correlates of MPQ-FD traits. That is, MPQ-FD traits such as social assurance and low stress reaction seem to provide a slightly greater advantage to women in facilitating desirable outcomes in regards to achievement and positive peer and family relationships, with most of these associations attributable to genetic factors. In contrast, genetic factors underlying similar MPQ-FD traits along with fearlessness and venturesomeness in men may create an additional pathway (relative MPQ-IA traits) to antisocial behavior that is mediated by deviant peer affiliation. Such a process would be consistent with our previous finding of a significantly higher genetic correlation between MPQ-FD and externalizing disorder symptoms for male twins of the age 17 cohort of the MTFS (Blonigen et al., 2005).

## Limitations

While the current study provides novel findings, it is not without limitations, and additional follow-up research is clearly warranted. One limitation is that our large sample, although representative of its target population, is not ethnically or racially diverse, nor does it have the high levels of criminal deviance or the extremes of environmental deprivation often seen in prisoners and other clinical samples that have a high prevalence of psychopathy. Another is that MPQ-FD is only weakly indicative of the more pathological aspects of F1/primary psychopathy such as callousness and lack of remorse, and, at best, has modest associations with antisocial behavior (Benning et al., 2005). Therefore, our findings should be replicated using other measures that better assess these aspects of F1/primary psychopathy. However, it will be important in work of this type to distinguish any unique effects of this psychopathy variant from those that overlap with F2/secondary psychopathy (an advantage of using the uncorrelated MPQ-FD and MPQ-IA measures). Also, while the associations between MPQ-FD and the environmental measures were modest to null, it is possible that other environmental variables not included in this study might exhibit stronger associations with MPQ-FD, indicative of an important etiological process. As such, additional efforts should be made to identify such variables. It will also be important to directly test mechanisms of active and evocative G-E correlation as described in the current study, as well as examine the potential for passive G-E correlations in childhood that contribute to psychopathic personality traits and exposure to environmental risk. This can be accomplished using longitudinal data and discordant twin designs to better infer causal effects in G-E interplay. In addition, while we examined genotypic effects on exposure to environmental risk, we did not evaluate the possibility of G x E interaction, that is, genotypic effects that moderate the impact of environmental risk factors following exposure.

In conclusion, our work provides further evidence of differential correlates between psychopathy variants and environmental risk factors, and extends prior understanding of this phenomenon by demonstrating that observed links are a consequence of common genetic risk factors. Importantly, different genetic effects underlie the psychopathy variants, and these genetic and phenotypic differences contribute to markedly different environmental

contexts that can either increase or decrease the likelihood of negative outcomes. At the same time, while our results provide a novel contribution by testing the differential heritability hypothesis of psychopathy variants, other investigators have made important theoretical and empirical contributions to understanding G-E correlations in the emergence of antisocial behavior and reported findings similar to our own (e.g., Ge et al., 1996; Jaffee et al., 2004; Larsson et al., 2008; Schulz-Heik et al., 2009; for a review see Moffitt, 2005). Taken together, findings along these lines remind of us of the value of differing approaches when examining etiology, and the importance of conducting rigorous tests of genetic and environmental influences when drawing causal inferences.

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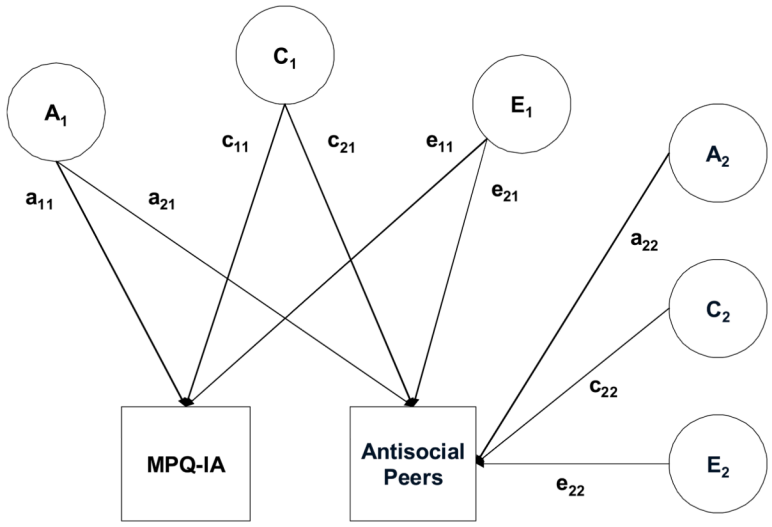


**Figure 1.**

Path diagram of a bivariate ACE Cholesky model for MPQ Impulsive Antisociality (MPQ-IA) and antisocial peers (AP). This model decomposes the variance into additive genetic ($A_1$, $A_2$), shared environmental ($C_1$, $C_2$), and nonshared environmental ($E_1$, $E_2$) effects. $a_{11}$, $c_{11}$, and $e_{11}$ are paths representing effects on MPQ-IA only. $a_{21}$, $c_{21}$, and $e_{21}$ are effects on MPQ-IA that also contribute to AP. $a_{22}$, $c_{22}$, and $e_{22}$ are effects that are unique to AP.

**Table 1**

Descriptive Statistics and Gender Differences in Psychopathic Personality Traits and Environmental Variables

| Psychopathic Personality Traits | Boys | | Girls | | | |
|---|---|---|---|---|---|---|
| | *n* | Mean SD | *n* | Mean SD | *F*-value | Cohen's *d* |
| MPQ-Fearless Dominance | 1115 | 53.2 8.9 | 1296 | 47.2 10.0 | $F(1,1242.38) = 179.42$ | .63* |
| MPQ-Impulsive Antisociality | 1115 | 52.3 9.3 | 1296 | 48.0 9.8 | $F(1,1228.50) = 87.04$ | .45* |
| Environmental Variables | | | | | | |
| Academic Achievement & Engagement | 1160 | 47.3 10.6 | 1293 | 52.4 8.8 | $F(1, 1257.12) = 108.22$ | −.52* |
| Prosocial Peers | 1072 | 50.5 10.2 | 1306 | 49.6 9.8 | $F(1, 1221.52) = 4.67$ | .09 |
| Antisocial Peers | 1025 | 52.3 11.0 | 1302 | 48.2 8.6 | $F(1, 1204.51) = 66.54$ | .42* |
| Mother-child Relationship Problems | 1169 | 51.1 10.0 | 1335 | 49.0 9.8 | $F(1, 1271.08) = 17.39$ | .21* |
| Father-child Relationship Problems | 1185 | 49.9 9.9 | 1314 | 50.1 10.0 | $F(1, 1258.35) = .03$ | −.02 |
| Dependent Stressful Life Events: School and Legal Problems | 1181 | 1.90 2.22 | 1332 | 1.38 1.53 | $F(1, 1275.84) = 33.45$ | .27* |
| Independent Stressful Life Events: Family-level Problems | 1184 | 2.64 2.89 | 1336 | 2.94 3.02 | $F(1, 1287.16) = 4.88$ | −.10* |

*Note.*

* $p < .001$.

Dependent stressful life events are a count of the number of events reported by the participant. All other scores were standardized to a T-score metric with mean of 50 and standard deviation of 10. The test statistics are adjusted for the non-independence of the twin observations, which results in non-integer values for the degrees of freedom of the *F* tests.

**Table 2**

Regression Coefficients between Psychopathic Personality Traits and Environmental Variables.

| | Predictor Variables | | | |
|---|---|---|---|---|
| **Dependent Variable** | **MPQ-Fearless Dominance (β)** | **MPQ-Impulsive Antisociality (β)** | **$\Delta\chi^2(1)$ MPQ-FD = MPQ-IA** | **$\Delta\chi^2(2)$ Boys = Girls** |
| Academic Achievement & Engagement | | | | |
| Boys | .10 | −.30* | 54.42* | |
| Girls | .17* | −.34* | 120.59* | 1.19 |
| Prosocial Peers | | | | |
| Boys | .10 | −.24* | 45.16* | |
| Girls | .19* | −.31* | 141.77* | 3.12 |
| Antisocial Peers | | | | |
| Boys | .14* | .37* | 20.84* | |
| Girls | −.04 | .41* | 103.25* | 15.00* |
| Mother-Child Relationship Problems | | | | |
| Boys | −.09 | .35* | 72.28* | |
| Girls | −.11* | .40* | 145.85* | .51 |
| Father-Child Relationship Problems | | | | |
| Boys | −.06 | .33* | 40.22* | |
| Girls | −.14* | .35* | 95.96* | 2.17 |
| Dependent Life Events: School & Legal Problems | | | | |
| Boys | .06 | .32* | 29.46* | |
| Girls | −.03 | .34* | 81.32* | 12.43 |
| Independent Life Events: Family-level Problems | | | | |
| Boys | .04 | .21* | 9.71 | |
| Girls | −.06 | .22* | 41.15* | 4.54 |

*Note.*

*$p < .001$.

**Table 3**

Twin Correlations and Univariate Estimates of Additive Genetic (A), Shared Environmental (C), and Nonshared Environmental (E) Variance Components with 95% Confidence Intervals.

| Variable | MZ | DZ | A | C | E |
|---|---|---|---|---|---|
| MPQ-Fearless Dominance | | | | | |
| Boys | .45 | .18 | .46 (.27, .53) | .00 (.00, .16) | .54 (.47, .62) |
| Girls | .47 | .16 | .45 (.26, .52) | .00 (.00, .17) | .55 (.48, .63) |
| MPQ-Impulsive Antisociality | | | | | |
| Boys | .52 | .22 | .52 (.31, .59) | .00 (.00, .19) | .48 (.41, .55) |
| Girls | .57 | .30 | .48 (.23, .61) | .08 (.00, .30) | .44 (.39, .51) |
| Academic Achievement & Engagement | | | | | |
| Boys | .76 | .41 | .76 (.54, .80) | .01 (.00, .22) | .23 (.20, .27) |
| Girls | .78 | .45 | .66 (.47, .81) | .12 (.00, .30) | .22 (.19, .26) |
| Prosocial Peers | | | | | |
| Boys | .65 | .40 | .51 (.26, .70) | .14 (.00, .37) | .35 (.29, .41) |
| Girls | .67 | .51 | .31 (.12, .52) | .36 (.16, .53) | .33 (.29, .39) |
| Antisocial Peers | | | | | |
| Boys | .74 | .47 | .60 (.37, .78)[a] | .15 (.00, .36) | .25 (.21, .30) |
| Girls | .65 | .57 | .22 (.04, .41) | .44 (.26, .60) | .34 (.29, .40) |
| Mother-Child Relationship Problems | | | | | |
| Boys | .83 | .57 | .52 (.37, .72) | .31 (.12, .46) | .17 (.14, .20) |
| Girls | .75 | .55 | .46 (.30, .65) | .29 (.12, .44) | .24 (.21, .28) |
| Father-Child Relationship Problems | | | | | |
| Boys | .85 | .75 | .27 (.16, .40) | .59 (.46, .70) | .14 (.12, .17) |
| Girls | .82 | .76 | .19 (.09, .30) | .64 (.53, .73) | .17 (.14, .20) |
| Dependent Life Events: School & Legal Problems | | | | | |
| Boys | .65 | .32 | .66 (.46, .71) | .00 (.00, .18) | .34 (.29, .40) |
| Girls | .62 | .41 | .58 (.38, .70) | .07 (.00, .26) | .35 (.30, .40) |

*Note*. MZ = monozygotic twin correlation; DZ = dizygotic twin correlation;

[a] = significant gender difference at $p < .05$. Independent life events was not included in the heritability analysis, because twins are necessarily concordant on family-level events.

**Table 4**

Phenotypic, Genetic, and Environmental Correlations (95% Confidence Intervals) between MPQ-Fearless Dominance and Environmental Variables

| | MPQ-Fearless Dominance | | | |
|---|---|---|---|---|
| **Environmental Variable** | ***r*** | $r_A$ | $r_E$ | **Percent of Covariance due to Genetic Effects** |
| Academic Achievement & Engagement | | | | |
| Boys | .07 (.01, .14) | .05 (−.08, .17) | .13 (.02, .24) | 39 |
| Girls | .18 (.12, .24) | .32 (.20, .45) | .02 (−.08, .12) | 96 |
| Prosocial Peers | | | | |
| Boys | .08 (.01, .14) | .10 (−.06, .26) | .06 (−.04, .17) | 63 |
| Girls | .19 (.14, .25) | .41 (.23, .66) | .07 (−.03, .16) | 85 |
| Antisocial Peers | | | | |
| Boys | .16 (.08, .22) | .38 (.23, .58)[a] | −.11 (−.23, .00) | 100 |
| Girls | −.05 (−.11, .01) | −.11 (−.39, .11) | −.04 (−.13, .06) | -- |
| Mother-Child Relationship Problems | | | | |
| Boys | −.07 (−.14, .00) | −.14 (−.29, .01) | −.01 (−.12, .10) | -- |
| Girls | −.12 (−.18, −.08) | −.19 (−.34, −.05) | −.09 (−.19, .01) | 72 |
| Father-Child Relationship Problems | | | | |
| Boys | −.01 (−.08, .05) | .00 (−.22, .22)[a] | −.05 (−.16, .07) | -- |
| Girls | −.14 (−.20, −.08) | −.37 (−.63, −.16) | −.08 (−.18, .01) | 81 |
| Dependent Life Events: School and Legal Problems | | | | |
| Boys | .08 (.02, .15) | .18 (.04, .32) | −.03 (−.14, .08) | 100 |
| Girls | −.03 (−.09, .03) | −.01 (−.14, .12) | −.06 (−.16, .03) | -- |

*Note.* $r$ = phenotypic correlation; $r_A$ = additive genetic correlation; $r_E$ = nonshared environmental correlation;

[a] = significant gender difference at $p < .05$. If the phenotypic correlation was not significant, we did not estimate the percent of covariance due to genetic effects. Independent life events were not included in the biometric analyses, because twins are necessarily concordant on family-level events.

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**Table 5**

Phenotypic, Genetic, and Environmental Correlations (95% Confidence Intervals) between MPQ-Impulsive Antisociality and Environmental Variables

| | MPQ-Impulsive Antisociality | | | |
|---|---|---|---|---|
| **Environmental Variable** | ***r*** | $r_A$ | $r_E$ | **Percent of Covariance due to Genetic Effects** |
| Academic Achievement & Engagement | | | | |
| Boys | −.29 (−.35, −.22) | −.32 (−.44, −.22) | −.25 (−.35, −.14) | 71 |
| Girls | −.34 (−.39, −.28) | −.42 (−.54, −.31) | −.28 (−.37, −.18) | 74 |
| Prosocial Peers | | | | |
| Boys | −.22 (−.28, −.15) | −.32 (−.50, −.18) | −.12 (−.22, −.01) | 78 |
| Girls | −.32 (−.37, −.26) | −.51 (−.73, −.36) | −.21 (−.30, −.12) | 74 |
| Antisocial Peers | | | | |
| Boys | .33 (.27, .39) | .42 (.30, .55) | .23 (.11, .34) | 76 |
| Girls | .37 (.31, .42) | .56 (.41, .78) | .30 (.20, .37) | 69 |
| Mother-Child Relationship Problems | | | | |
| Boys | .32 (.26, .38) | .52 (.38, .67) | .18 (.07, .28) | 84 |
| Girls | .41 (.35, .46) | .50 (.39, .62) | .38 (.30, .47) | 69 |
| Father-Child Relationship Problems | | | | |
| Boys | .31 (.25, .37) | .72 (.53, .94) | .16 (.05, .27) | 87 |
| Girls | .33 (.27, .38) | .68 (.52, .88) | .23 (.14, .32) | 80 |
| Dependent Life Events: School and Legal Problems | | | | |
| Boys | .32 (.26, .38) | .41 (.30, .53) | .21 (.11, .31) | 74 |
| Girls | .33 (.28, .39) | .49 (.36, .64) | .16 (.06, .25) | 81 |

*Note*. $r$ = phenotypic correlation; $r_A$ = additive genetic correlation; $r_E$ = nonshared environmental correlation. Independent life events were not included in the biometric analyses, because twins are necessarily concordant on family-level events.