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Psychopathic personality traits: heritability and genetic overlap with internalizing and externalizing psychopathology

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ABSTRACT

Background. Little research has examined genetic and environmental contributions to psychopathic personality traits. Additionally, no studies have examined etiological connections between psychopathic traits and the broad psychopathological domains of internalizing (mood and anxiety) and externalizing (antisocial behavior, substance abuse). The current study was designed to fill these gaps in the literature.

Method. Participants were 626 pairs of 17-year-old male and female twins from the community. Psychopathic traits were indexed using scores on the Multidimensional Personality Questionnaire (MPQ). Symptoms of internalizing and externalizing psychopathology were obtained via structured clinical interviews. Structural equation modeling was used to estimate genetic and environmental influences on psychopathic personality traits as well as the degree of genetic overlap between these traits and composites of internalizing and externalizing.

Results. Twin analyses revealed significant genetic influence on distinct psychopathic traits (*Fearless Dominance* and *Impulsive Antisociality*). Moreover, *Fearless Dominance* was associated with reduced genetic risk for internalizing psychopathology, and *Impulsive Antisociality* was associated with increased genetic risk for externalizing psychopathology.

Conclusions. These results indicate that different psychopathic traits as measured by the MPQ show distinct genetically based relations with broad dimensions of DSM psychopathology.

INTRODUCTION

Psychopathy is a disorder marked by a constellation of maladaptive personality traits. Within this literature, some scholars have postulated that the disorder comprises distinct facets including *interpersonal-affective* traits (e.g. superficial charm, manipulativeness, poverty of affect) and *antisocial* traits (e.g. impulsivity, aggression; Hare, 1991, 2003; Cooke & Michie, 2001). In terms of etiology, while many studies have investigated phenotypes related to psychopathy (e.g. delinquency, antisocial behavior), very little is known about the genetic and environmental structure of psychopathy *per se*, as virtually no studies have investigated the genetic and environmental contributions to the interpersonal-affective traits (see Blonigen *et al*. 2003 for an exception).

In the present investigation, we examined the biometric structure of the interpersonal-affective and antisocial traits of psychopathy using a community sample of male and female adolescent twins. Previous findings suggest that these traits exhibit divergent phenotypic relations with *internalizing* (Int) (mood and anxiety) and *externalizing* (Ext) (antisocial behavior and substance abuse) psychopathology (Patrick *et al.*

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2003; Benning *et al*. in press). Therefore, we also examined the degree of genetic overlap between psychopathic traits and these broader psychopathological domains to further clarify the underlying etiological bases of psychopathy and its relations with common mental disorders.

Psychopathy: description and behavior genetic investigations

The predominant view of psychopathy derives from the personality-based clinical conceptions of the syndrome (Cleckley, 1941/1976; Karpman, 1948; McCord & McCord, 1964) in which core interpersonal-affective deficits are emphasized (e.g. superficial charm, absence of nervousness, guiltlessness, pathologic egocentricity, dishonesty) rather than the commission of specific antisocial acts. Other theorists (Karpman, 1941; Lykken, 1957; Blackburn, 1975) have elaborated on this conceptualization of psychopathy to include *primary* and *secondary* subtypes, with the former characterized by low anxiety and antisocial behavior due to a lack of conscience, and the latter characterized by negative affect and impulsivity, and the expression of neurotic conflict as antisocial behavior.

Similar themes are embedded in the most prominent assessment instrument in the field, the Psychopathy Checklist – Revised (PCL-R; Hare, 1991, 2003), a semi-structured interview designed to assess these classic clinical descriptions of psychopathy in incarcerated settings. Factor analytic research (Harpur *et al*. 1989) has established that this measure captures two distinct facets of the syndrome: Factor 1 is marked by interpersonal-affective traits, and Factor 2 is marked behavioral indicators of antisocial deviance.[1]† Interestingly, the external correlates of Factors 1 and 2 strongly resemble clinical descriptions of primary and secondary psychopathy, respectively (Blackburn, 1975, 1996).

Although the interpersonal-affective and antisocial facets of psychopathy have received extensive empirical investigation, there is a lack of concomitant research exploring their genetic and environmental underpinnings. A primary reason for this is that the item content, use of collateral file information, and lengthy administration of the PCL-R were designed for incarcerated populations and are not conducive for use in epidemiological samples of twins or adoptees. Moreover, most behavior genetic studies have investigated behavior-based phenotypes [e.g. antisocial personality disorder (APD), juvenile delinquency, criminality; Mednick *et al*. 1984; Cloninger & Gottesman, 1987; DiLalla & Gottesman, 1989; Lyons *et al*. 1995; McGuffin & Thapar, 1998; Jacobson *et al*. 2000; Taylor *et al*. 2000; Goldstein *et al*. 2001] which preferentially index the antisocial traits of psychopathy and are, therefore, limited in their ability to address the etiology of the core interpersonal-affective features. This is a potentially critical limitation given that some researchers (e.g. Karpman, 1948; Mealey, 1995; Porter, 1996) have theorized that primary and secondary psychopathy may differ in their etiology with the former arising from a constitutional or genetic disposition, and the latter as a manifestation of environmental influences (e.g. parental rejection, abuse, poor socialization). However, such assertions have not been subject to empirical investigation with genetically informative data (see Skeem *et al*. 2003).

Assessment of psychopathy in the self-report domain

Given the limitations of the PCL-R, some scholars have turned to the self-report domain to investigate psychopathy within the general population. However, most self-report indices preferentially tap the antisocial traits of the disorder (Hare & Cox, 1978; Lilienfeld, 1994, 1998). A self-report measure that has shown potential as an index of both trait dimensions is the Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996). The PPI was designed to capture a range of psychopathic *personality* traits rather than overt antisocial behaviors. Based on evidence suggesting that it captures both the interpersonal-affective and antisocial traits of psychopathy (Lilienfeld & Andrews, 1996; Poythress *et al*. 1998), Benning *et al*. (2003) factor-analyzed PPI subscale scores from a community sample of adult men. Two dominant, orthogonal factors were extracted whose external correlates resembled those of the PCL-R factors. The first factor (PPI-I: *Fearless Dominance*) was marked by social potency, stress immunity, and fearlessness; core features

† The notes will be found on p. 646.

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of the interpersonal-affective traits and analogous to primary psychopathy. The second factor (PPI-II: *Impulsive Antisociality*) was marked by negative emotionality (aggression, alienation) and low behavioral constraint (impulsivity, sensation seeking), and is analogous to secondary psychopathy. Subsequent validation studies have demonstrated that *both* PPI factors demonstrate convergent *and* discriminant validity for the interpersonal-affective and antisocial traits of psychopathy (Benning *et al.* 2003; Lilienfeld & Skeem, 2004).

Measuring psychopathy via normal personality

A notable finding regarding the PPI factors is that they can be measured effectively using the Multidimensional Personality Questionnaire (MPQ, Tellegen, in press), a broadband measure of personality. Benning *et al.* (2003) found that the primary scales of the MPQ captures a substantial portion of variance in these factors ($R = 0{\cdot}89$ for *Fearless Dominance*; 0·84 for *Impulsive Antisociality*).[2] Recently, Benning *et al.* (in press) examined the criterion-validity of these factors as estimated from the MPQ in two community samples and a sample of male prisoners. *Fearless Dominance* was negatively related to social phobia and depression as well as self-reported fear, distress, and anxiety, and correlated positively with indices of fearlessness, sociability, thrill-adventure seeking, narcissism, and PCL-R Factor 1 (Benning *et al.* in press). Conversely, *Impulsive Antisociality* was positively associated with substance dependence, child and adult antisocial behavior, self-reported anxiety, disinhibition, and boredom susceptibility, and PCL-R Factor 2, and inversely related to socialization (Benning *et al.* in press).

Overall, the external correlates of these constructs are consistent across community and offender samples and largely resemble the external correlates of the PCL-R factors. This suggests that the MPQ-estimated psychopathy factors may index related constructs within the domain of normal personality. Given the feasibility of administering the MPQ in large epidemiological samples of twins or adoptees, the findings of Benning *et al.* (in press) provide a foundation for studying the genetic and environmental underpinnings of psychopathy within the general population. Accordingly, our first objective was to examine if there is a differential heritability to these psychopathic traits as has been theorized in the literature (see Skeem *et al.* 2003).

Phenotypic relations with psychopathology: Int and Ext

A notable finding regarding *Fearless Dominance* and *Impulsive Antisociality* is that they exhibit divergent phenotypic relations with Int and Ext psychopathology (Benning *et al.* in press). These dimensions represent systematic covariation among common DSM disorders with the former reflecting the covariation among unipolar mood and anxiety (Vollebergh *et al.* 2001) and the latter representing the covariance among child and adult antisocial behavior and substance dependence (Krueger *et al.* 2002). With respect to Int, self-reported fear and anxiety were inversely related to *Fearless Dominance*, whereas *Impulsive Antisociality* correlated positively with such measures (Benning *et al.* in press). Diagnostically, *Fearless Dominance* was negatively associated with social phobia, simple phobia, and major depression, whereas *Impulsive Antisociality* was positively associated with major depression (Benning *et al.* in press). With respect to Ext, *Impulsive Antisociality* was positively associated with child and adult antisocial behavior and substance dependence, while *Fearless Dominance* was relatively unrelated to these disorders with the exception of a positive correlation with adult antisocial behavior in a sample of adult men (Benning *et al.* 2003).

Despite these diverging phenotypic associations with indicators of Int and Ext, the extent to which psychopathic traits relate to these broad factors of psychopathology at a genetic level remains unclear. Thus, a second aim of the present investigation was to estimate the degree of genetic overlap between psychopathic traits and factors of Int and Ext. Given the inverse association between *Fearless Dominance* and indices of Int, and the positive association between *Impulsive Antisociality* and indices of Ext, we hypothesized a parallel genetic relationship among these variables.

METHOD

Participants

Participants were 17-year-old male and female twins from the Minnesota Twin-Family Study

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(MTFS). The MTFS is an ongoing epidemiological-longitudinal study examining the genetic and environmental factors that contribute to the development of substance abuse and related psychopathology in reared together, same-sex twins and their parents. A comprehensive description of the objectives and design of the MTFS has been provided elsewhere (Iacono *et al*. 1999; Iacono & McGue, 2002). The present investigation involved male twins born between the years 1972–1978, and female twins born between the years 1975–1979. Participating twins were predominately Caucasian (98 %), which is consistent with the demographics of Minnesota when the twins were born. Families were excluded from participation if they lived further than a day's drive from our laboratories in Minneapolis, or if either twin had a serious intellectual or physical disability that would preclude him or her from completing the day-long, in-person assessment. Following the intake assessment, the sample size consisted of 289 male ($n_{MZ}=188$, $n_{DZ}=101$) and 337 female ($n_{MZ}=223$, $n_{DZ}=114$) twin pairs.

Zygosity was determined by the agreement of three separate estimates: (1) a standard zygosity questionnaire completed by parents, (2) an evaluation by MTFS staff regarding the physical similarity between the twins, and (3) an algorithm comparing twins on ponderal and cephalic indices and fingerprint ridge counts. In situations in which the three estimates did not agree, a serological analysis was performed.

Assessment

Psychopathic personality traits

Because PPI data were not available for members of the current sample, factor scores on *Fearless Dominance* and *Impulsive Antisociality* were estimated using the 11 primary scales of the 198-item version of the MPQ. Specifically, regression weights derived from a community sample of adult men ($n=353$; Benning *et al*. 2003), were applied to MPQ primary scale scores to estimate factor scores for these psychopathy constructs.[3]

All families were mailed the MPQ prior to their intake assessment. Participants were asked to bring their completed MPQ with them to their in-person visit. If the MPQ was not completed either upon arrival for their intake assessment or by the end of the day-long visit, participants were asked to complete it at home and return it by mail. MPQ data were available for 1122 individuals (men = 502, women = 620). Consistent with previous investigations (Benning *et al*. 2003, in press; Patrick, 2004), *Fearless Dominance* and *Impulsive Antisociality* were uncorrelated in men and women, $r=0{\cdot}03$, N.S. and 0·00, N.S. respectively.

Int and Ext psychopathology

All twins were interviewed in-person for lifetime presence of several common mental disorders according to DSM-III-R criteria (the version of the diagnostic manual current at intake; APA, 1987) including: major depression, social phobia, simple phobia, conduct disorder, antisocial personality disorder (APD), and alcohol, nicotine, and drug dependence. Trained individuals with either a bachelor's or a master's degree in psychology conducted the interviews. A modified version of the *Structured Clinical Interview for DSM-III-R* (Spitzer *et al*. 1990) was used to assess major depression, social phobia, and simple phobia. Male twins were not assessed for Int disorders at intake, but were at the age 20 follow-up assessment ($n=$ 470–474). Therefore, for men, relations with Int disorders refer to lifetime symptoms assessed at age 20. A structured interview designed by MTFS staff (Holdcraft *et al*. 1998) was administered to assess symptoms of conduct disorder and adult antisocial behavior (the adult criteria for APD). The Substance Abuse Module of the Composite International Diagnostic Interview (Robins *et al*. 1987) was used to assess symptoms of alcohol, nicotine, and illicit drug dependence. The drug assessment included amphetamines, cannabis, cocaine, hallucinogens, inhalants, opioids, phencyclidine, and sedatives. The substance for which the participant evinced the maximal number of symptoms was used as their number of drug dependence symptoms. Additionally, mothers reported on symptom presence for conduct disorder and substance dependence disorders in both twins using the parent version of the Diagnostic Interview for Children and Adolescents – Revised (Reich & Welner, 1988). Symptoms were deemed present if reported by either the mother or the twin.

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Following the intake assessment, all diagnostic interview data were reviewed in a case conference by at least two graduate students with advanced training in descriptive psychopathology and differential diagnosis. All items scored positive, or about which there was ambiguity regarding scoring, were reviewed with the aid of audiotapes from the interviews in order to achieve a consensus regarding symptom presence. Excellent reliability regarding this consensus process has been previously reported (Iacono *et al*. 1999) with kappas ranging from 0·78 (social phobia) to 0·95 (adult antisocial behavior).

In order to examine the genetic overlap between psychopathic personality traits and psychopathology, an Int variable was calculated by taking the average standardized (via *z*-score transformation) symptom count score among major depression, social phobia, and simple phobia. An Ext variable was calculated in the same manner using symptoms of adult antisocial behavior, conduct disorder, and alcohol, nicotine, and drug dependence. Int and Ext were significantly correlated for women, $r = 0{\cdot}21$, $p < 0{\cdot}001$, but not men, $r = 0{\cdot}00$, N.S.

Data analysis

Model-fitting analyses in the present study involved twin methodology. Twin studies utilize the difference in proportion of alleles shared between monozygotic (MZ) and dizygotic (DZ) twin pairs to estimate the relative genetic and environmental contributions to observed phenotypes. This methodology typically involves the estimation of additive genetic (a^2), shared environmental (c^2), and non-shared environmental effects (e^2). Additive genetic influences involve the summation of individual genes across loci. MZ (identical) twins share 100% of their additive genetic effects while DZ (fraternal) twins share 50% of these effects, on average. Thus, MZ correlations will be roughly twice as large as the DZ correlations if the phenotype in question is primarily due to additive genetic contributions. Shared environmental effects (c^2) are environmental effects common to both members of a twin pair that produce similarities between them (e.g. family environment). Substantial c^2 effects on a phenotype would generate MZ and DZ correlations similar in magnitude. Non-shared environmental effects (e^2) are environmental factors unique to each member of a twin pair that tends to create differences between the twins.

To examine the etiological connections between psychopathic traits and Int and Ext, we fit a Cholesky decomposition to the data, which parses the individual variance of each phenotype as well as the covariance between phenotypes into genetic, shared, or non-shared environmental factors. Using this approach, the amount of shared variance between the psychopathy traits and Int or Ext was partitioned into its genetic and environmental components. This allows for the estimation of the proportion of phenotypic covariance between the psychopathy traits and Int or Ext that is due to genetic, shared, or non-shared environmental effects. Moreover, these effects can then be standardized on their respective variances to compute genetic, shared, or non-shared environmental correlations. These estimates indicate the degree to which the genetic, shared, or non-shared environmental effects of one variable are correlated with that same effect on another variable.

All model-fitting analyses were conducted using *Mx*, a structural-equation modeling program (Neale *et al*. 2002). We fit models to the raw data using maximum-likelihood ('all data') estimation, which corrects for potential biases due to missing data. Specifically, this technique uses all available information to estimate values for the missing data and then adjusts for the imprecision of the estimated values. When first fitting models to raw data, all means, variances, and covariances are freely estimated to get a baseline index of fit (minus twice the natural log-likelihood of the data; $-2 \ln L$). A comparison of the $-2 \ln L$ under this unrestricted model with the $-2 \ln L$ under more restrictive biometric models yields a likelihood-ratio χ^2 goodness-of-fit test ($\Delta\chi^2$). To guide selection of the best-fitting model, this $\Delta\chi^2$ was then converted to the Akaike information criterion (AIC $= \chi^2 - 2$ df; Akaike, 1987). The AIC is a measure of model fit relative to parsimony and is used to assess the comparative fit among a series of competing biometric models.

Generally, parameter estimates could be constrained across men and women without a significant decrement in fit. However, some significant gender differences were observed. Therefore, we present results separately for men

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Table 1. *Twin correlations for psychopathic personality traits, internalizing, and externalizing*

	Men		Women		Combined	
	MZ (95% CI)	DZ (95% CI)	MZ (95% CI)	DZ (95% CI)	MZ (95% CI)	DZ (95% CI)
Fearless Dominance	0·42 (0·29–0·54)	0·20 (−0·01 to 0·40)	0·45 (0·34–0·56)	0·21 (0·02 to 0·39)	0·44 (0·36–0·52)	0·20 (0·06–0·34)
Impulsive Antisociality	0·51 (0·39–0·61)	0·17 (−0·05 to 0·38)	0·49 (0·37–0·59)	0·28 (0·10 to 0·44)	0·50 (0·41–0·57)	0·24 (0·10–0·37)
Internalizing	0·48 (0·34–0·59)	0·19 (−0·04 to 0·40)	0·29 (0·17–0·41)	0·14 (−0·05 to 0·32)	0·35 (0·25–0·44)	0·15 (0·01–0·29)
Externalizing	0·70 (0·62–0·77)	0·44 (0·27 to 0·59)	0·68 (0·60–0·74)	0·26 (0·08 to 0·42)	0·68 (0·63–0·73)	0·37 (0·25–0·48)

MZ, Monozygotic twins; DZ, Dizygotic twins; CI, confidence interval.

All twin correlations were estimated by fitting models to the raw data using maximum-likelihood estimation, which corrects for any potential biases due to missing data. $n = 1252$ individuals from 626 twin pairs, some with missing data. Internalizing was calculated by taking the average standardized (via *z*-score transformation) symptom count score among major depression, social phobia, and simple phobia. Externalizing was calculated in the same manner using symptoms of adult antisocial behavior, conduct disorder, and alcohol, nicotine, and drug dependence. Combined refers to models in which parameters were equated across the genders.

Table 2. *Heritability estimates from best-fitting univariate twin models for psychopathic personality traits, internalizing, and externalizing*

	Men		Women		Combined	
	Heritability	(95% CI)	Heritability	(95% CI)	Heritability	(95% CI)
Fearless Dominance	0·46	(0·32–0·57)	0·45	(0·34–0·54)	0·45	(0·37–0·53)
Impulsive Antisociality	0·51	(0·39–0·62)	0·48	(0·37–0·57)	0·49	(0·41–0·56)
Internalizing	0·49	(0·35–0·60)	0·31	(0·18–0·43)	0·36	(0·27–0·45)
Externalizing	0·76	(0·70–0·81)	0·68	(0·60–0·74)	0·73	(0·68–0·77)

$n = 1252$ individuals from 626 twin pairs, some with missing data. CI, confidence interval. The heritability for all variables are due to additive genetic effects. Combined refers to models in which the unstandardized parameters were equated across the genders.

and women, as well as estimates from combined models in which the unstandardized parameters were equated across the genders.

RESULTS

Etiological structure of psychopathic traits

Twin correlations estimated from baseline models for univariate biometric models are presented in Table 1. For both men and women, the twin correlations for *Fearless Dominance*, *Impulsive Antisociality*, Int, and Ext are generally consistent with an additive model of inheritance (MZ correlations approximately twice that of DZ correlations). To test for mean differences across the genders, a model allowing the means to vary was compared to a model in which the means were constrained to be equal. Within this model, men exhibited significantly greater mean levels of psychopathic traits and Ext symptoms than women ($\chi^2_{(1)} = 107{\cdot}7$, 53·6, 30·0, all p's $< 0{\cdot}001$), for *Fearless Dominance*, *Impulsive Antisociality*, and Ext respectively, while women exhibited significantly greater mean levels of Int symptoms ($\chi^2_{(1)} = 59{\cdot}7$, $p < 0{\cdot}001$). Therefore, we allowed the means to vary across gender for all biometric models. Results of the univariate biometric models were consistent with impressions from the twin correlations. For both *Fearless Dominance* and *Impulsive Antisociality*, as well as for Int and Ext, the AIC was smallest for the AE model (additive genetic and non-shared environment).[4]

Table 2 provides the heritability estimates from the best-fitting models. In the combined sample, roughly half the variance in both *Fearless Dominance* and *Impulsive Antisociality* was due to additive genetic effects. Moreover, the heritability was moderate for Int and strong for Ext. The heritability estimates for all the variables was very similar for men and women, although a sex difference for Int approached significance, $\chi^2_{(1)} = 3{\cdot}72$, $p = 0{\cdot}054$. There was virtually no evidence of shared environmental contributions to any of the phenotypes. Even when shared environmental effects were included in the model, almost all parameter estimates were zero.

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Table 3. *Correlations between psychopathic personality traits and symptoms of common mental disorders*

	Men		Women	
Symptom counts	Fearless Dominance	Impulsive Antisociality	Fearless Dominance	Impulsive Antisociality
Major Depression	−0·11	0·15*	−0·09	0·21***
Simple Phobia	−0·13*	−0·02	−0·16**	0·05
Social Phobia	−0·21**	−0·02	−0·24***	0·04
Adult Antisocial Behavior	0·14*	0·36***	0·06	0·38***
Conduct Disorder	0·15*	0·30***	0·04	0·30***
Alcohol Dependence	0·09	0·23***	−0·02	0·29***
Nicotine Dependence	0·15*	0·25***	−0·02	0·36***
Drug Dependence	0·04	0·21**	0·10	0·26***
Internalizing	−0·25***	0·05	−0·26***	0·16**
Externalizing	0·15*	0·36***	0·04	0·40***

Correlations were estimated by fitting models to the raw data using maximum likelihood. $n = 1252$ individuals from 626 twin pairs, some with missing data. Significance levels were adjusted by weighting each twin one-half to account for the correlated nature of the observations.
* $p < 0.05$, ** $p < 0{\cdot}01$, *** $p < 0{\cdot}001$.

Phenotypic and genetic associations between psychopathic traits and psychopathology

Table 3 contains the phenotypic correlations between the psychopathy variables and symptom counts for each mental disorder as well as the Int and Ext composite variables. For both men and women, *Fearless Dominance* was negatively related to each Int disorder, whereas *Impulsive Antisociality* was positively correlated with symptoms of major depression. The Int composite was also negatively correlated with *Fearless Dominance* for both genders. In women, Int was significantly correlated with *Impulsive Antisociality*, but this gender difference was not significant. For both men and women, *Impulsive Antisociality* was positively correlated with each Ext disorder. In men, *Fearless Dominance* was also weakly but significantly correlated with symptoms of adult antisocial behavior, conduct disorder, and nicotine dependence. For both men and women, the Ext composite was positively correlated with *Impulsive Antisociality*. In men, but not women, Ext was also significantly correlated with *Fearless Dominance*, but this gender difference was not significant.

The genetic correlations between each psychopathy variable and Int and Ext are provided in Table 4. Consistent with the phenotypic findings, *Fearless Dominance* and *Impulsive Antisociality* evinced a divergent pattern of associations with Int and Ext. For both genders, *Fearless Dominance* exhibited a moderate negative genetic correlation with Int. Moreover, further analysis revealed that 66% of the phenotypic covariance between these variables was due to genetic contributions. Conversely, *Impulsive Antisociality* exhibited a moderate positive genetic correlation with Ext, with 76% of the phenotypic covariance between these variables due to genetic factors. In women, but not men, there was a moderate genetic correlation between *Impulsive Antisociality* and Int, and this gender difference was significant, $\chi^2_{(1)} = 5{\cdot}77$, $p = 0{\cdot}016$. Moreover, 89% of the phenotypic association between these variables in women was due to genetic effects. This indicates that for women, genetic effects that contribute to *Impulsive Antisociality* increase the risk for the expression of both Int and Ext symptoms. In men, but not women, there was a moderate genetic correlation between *Fearless Dominance* and Ext, and this gender difference was also significant, $\chi^2_{(1)} = 6{\cdot}89$, $p = 0{\cdot}009$, with 100% of the phenotypic covariance due to genetic factors. This indicates that for men, genetic effects that contribute to *Fearless Dominance* also contribute to the expression of Ext symptoms.[5]

DISCUSSION

Etiological structure of psychopathic traits: conceptual implications

Behavior genetic studies of psychopathy have been virtually absent in the literature with most investigations only addressing phenotypes related to antisocial behavior (i.e. criminality,

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Table 4. *Genetic correlations between psychopathic personality traits, internalizing, and externalizing*

	Internalizing						Externalizing					
	Men		Women		Combined		Men		Women		Combined	
	r_g	(95% CI)	r_g	(95% CI)	r_g	(95% CI)	r_g	(95% CI)	r_g	(95% CI)	r_g	(95% CI)
Fearless Dominance	**−0·40**	(−0·64 to −0·16)	***−0·39***	(−0·63 to −0·15)	**−0·40**	(−0·57 to −0·22)	***0·36***	(0·17 to 0·56)	0·01	(−0·16 to 0·19)	**0·16**	(0·04 to 0·29)
Impulsive Antisociality	−0·03	(−0·27 to 0·20)	***0·38***	(0·14 to 0·64)	**0·20**	(0·03 to 0·37)	**0·45**	(0·28 to 0·60)	**0·52**	(0·37 to 0·65)	**0·49**	(0·38 to 0·59)

r_g, Genetic correlation; CI, confidence interval.
Correlations in bold are significant (confidence intervals do not include zero). Correlations in bold and italicized are significant and significantly different for men and women. Combined refers to models in which the unstandardized parameters were equated across the genders.

juvenile delinquency, APD) rather than the interpersonal-affective traits of the syndrome. However, behavior genetic studies of both are vital given that some scholars (e.g. Karpman, 1948) have posited that primary psychopathy may reflect stronger genetic influences than secondary psychopathy. The present investigation is significant in that it examined the relative genetic and environmental contributions to *both* the interpersonal-affective (*Fearless Dominance*) and antisocial (*Impulsive Antisociality*) traits of psychopathy as measured by the MPQ. The results suggest that these traits are equally and substantially heritable with each accounting for roughly half of the total variance in both men and women. Furthermore, in accordance with their phenotypic independence, *Fearless Dominance* and *Impulsive Antisociality* were also genetically uncorrelated for both men ($r_g = 0{\cdot}12$, N.S.) and women ($r_g = -0{\cdot}05$, N.S.) in the present study.

Collectively, the current data suggest that *Fearless Dominance* and *Impulsive Antisociality* may derive from separate etiological processes that are substantially genetic in nature. On a theoretical level, these findings could have wider implications for conceptualizations of the psychopathy construct. Specifically, in the general population, psychopathy may consist of independently derived maladaptive personality traits that in certain individuals may combine to form a particularly virulent phenotype. Notably, this conceptualization is consistent with the ideas of some personality theorists who view personality disorders as a configuration of certain maladaptive traits (see Grove & Tellegen, 1991).

Associations between psychopathic traits and psychopathology

One of the central findings from the current study was the diverging genetic associations between psychopathic traits and broad factors of psychopathology. First, *Fearless Dominance* demonstrated a significant negative genetic correlation with an Int composite, a finding consistent with prior evidence of an inverse relationship between the interpersonal-affective facet of psychopathy and self-reported anxiety (Harpur *et al.* 1989; Patrick, 1994, 1995; Frick *et al.* 1999, 2000). The present findings are an extension of this work to DSM disorders and

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demonstrate that genetic influences on *Fearless Dominance* may provide a resiliency to developing a broad range of Int psychopathology.

Impulsive Antisociality, on the other hand, exhibited a positive genetic correlation with an Ext composite, which is consistent with previous data showing specific relations between PCL-R Factor 2 and substance dependence (Smith & Newman, 1990; Reardon *et al.* 2002), APD (Harpur *et al.* 1989), and a latent Ext factor (Patrick, 2003). Given the current findings, the relationship between *Impulsive Antisociality* and Ext appears to be largely mediated by genetic contributions such that heritable influences to these psychopathic traits may increase one's vulnerability to a spectrum of disorders marked by disinhibition.

It is also worth noting that sex differences were found in the genetic correlations between the psychopathic traits and psychopathology with *Fearless Dominance* genetically correlated with Ext in men only, and *Impulsive Antisociality* genetically correlated with Int in women only. This pattern of relations was roughly similar to the phenotypic associations among the variables and suggests that the etiological connections between psychopathic traits and Int and Ext may differ somewhat by gender. Despite the novelty of these findings, further replication of these gender differences is required before drawing any firm conclusions.

Overall, the genetic correlations from the present study help to elucidate the etiological boundaries of psychopathy. The findings suggest that future investigations on the etiology of the syndrome can be informed by an understanding of the common genetic risk factors for psychopathy and Int and Ext rather than simply examining the specific genetic influences on psychopathy. Although genetic links between personality and psychopathology have been repeatedly observed (Carey & DiLalla, 1994; Slutske *et al.* 2002; Krueger & Tackett, 2003), such findings are relatively novel with respect to the psychopathy literature and demonstrate that different psychopathic traits show distinct genetic relations with broad dimensions of psychopathology.

In terms of the classic clinical conceptions of psychopathy, the finding of divergent genetic relations with distinct psychopathological syndromes aligns closely with Cleckley's paradoxical description of the psychopath (1941/1976). Specifically, the clinical profile delineated by Cleckley includes several features of overt adjustment (e.g. superficial charm and good intelligence, absence of nervousness or psychoneurosis, suicide rarely carried out) which serve to mask a severe and underlying behavioral pathology in the psychopath (e.g. impulsive antisocial behavior, irresponsibility, promiscuity, failure to follow any life plan). The present findings of divergent genetic relations with distinct psychopathological domains suggests the possibility that this paradoxical presentation may reflect the confluence of two distinct etiological processes; one which serves as a protective factor to Int distress and is phenotypically expressed as adjustment, the other which confers a vulnerability to chronic behavioral deviance.

Limitations and future directions

Some limitations are worth noting. First, we utilized a normal range personality measure to index psychopathic traits rather than the PCL-R, the standard assessment tool for psychopathy in prison samples. However, there is compelling evidence to suggest that psychopathy can be captured well within structural models of personality (Patrick, 1994, 1995; Widiger & Lynam, 1998; Miller *et al.* 2001; Verona *et al.* 2001; Lynam, 2002; Hicks *et al.* 2004). Moreover, the use of an omnibus personality measure to index psychopathic traits has several advantages, most notably of which is the ability to investigate psychopathy within the general population as opposed to incarcerated or clinical samples. Nonetheless, Benning *et al.* (in press) do note that the MPQ psychopathy constructs are not intended to be isomorphic or synonymous with the PCL-R factors but instead represent an alternative and complementary conceptualization which relates closely to the personality-based clinical conceptions of psychopathy (cf. Cleckley, 1941/1976).

Second, despite the advantages of our sample (e.g. community sample, men and women), we utilized adolescents (17 years old) rather than adults to examine the heritability and underlying genetic relations of psychopathy. Thus, future investigations are needed to examine whether the present findings will generalize to an adult sample.

In summary, both *Fearless Dominance* and *Impulsive Antisociality* (analogous to primary and secondary psychopathy, respectively) have substantial genetic influences with the former conferring a genetic resiliency to Int disorders, and the latter reflecting a genetic vulnerability to Ext psychopathology. This provides construct validation for the notion of psychopathic traits as deriving from separate etiological processes (cf. Patrick, 2001) given that they exhibited convergent and discriminant relations with two distinct domains of psychopathology.

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DECLARATION OF INTEREST

None.

NOTES

[1] Alternative structural models of the PCL-R have also been proposed in the literature. Recently, Hare (2003) introduced the PCL-R (2nd edn), which consists of four facets labeled *interpersonal*, *affective*, *lifestyle*, and *antisocial*. However, this four-facet structure still retains the basic higher-order two-factor structure from the PCL-R (Hare, 1991). The *interpersonal* and *affective* facets load onto one factor that is consonant with Factor 1 from the PCL-R. Moreover, the *lifestyle* and *antisocial* facets cohere into one factor that is largely reminiscent of PCL-R Factor 2. Additionally, Cooke & Michie (2001) have suggested that the PCL-R is underpinned by a three-factor structure consisting of an *Arrogant and Deceitful Interpersonal Style*, *Deficient Affective Experience*, and an *Impulsive and Irresponsible Behavioral Style*.

[2] Previously, Benning *et al*. (2003) reported the multiple *R*'s for the prediction of PPI-I (*Fearless Dominance*) and PPI-II (*Impulsive Antisociality*) by the MPQ to be 0·70 and 0·67, respectively. However, given that these instruments were administered 4–6 years apart, these estimates were likely attenuated substantially by the test–retest unreliability inherent in administering personality instruments over such a period of time. Thus, the multiple *R*'s reported in this article represent disattenuated multiple *R*'s for *Fearless Dominance* and *Impulsive Antisociality* that were previously described in Benning *et al*. (in press). In that study, the authors used Spearman's (1904) formula ($R_{\text{disattenuated}} = R_{\text{observed}}/\sqrt{[\text{test–retest reliability}]}$) to correct the observed multiple *R*'s (0·70 and 0·67) for the unreliability of personality scores across time. However, we acknowledge that this correction may represent somewhat overly optimistic estimates of the PPI factors.

[3] In an effort to verify that using a regression-weighted approach to estimate psychopathy scores did not bias the findings, the data were re-analyzed using specific items from the MPQ rather than scale scores. The results were virtually identical to the findings based on the regression-weighted approach. Given that MPQ-estimated psychopathy scores have only been validated using regression weights (see Benning *et al*. in press), the results using this approach to estimate psychopathy scores were reported.

[4] In the present study, we also tested models that included non-additive genetic effects, because a previous twin study found that PPI *total scores* fit an epistatic model of inheritance (Blonigen *et al*. 2003). Notably, we found that PPI total scores, as predicted by the MPQ, might be influenced by non-additive genetic contributions (MZ $r = 0{\cdot}46$, DZ $r = 0{\cdot}08$, for the combined sample). However, given the lack of any previous studies examining the validity of estimating PPI total scores from the MPQ, as well as the fact that we may have limited power to detect such effects reliably, these findings are not emphasized here but are available upon request from the first author.

[5] We also examined the genetic correlations among the psychopathy variables and the specific disorders of which the Int and Ext composites were derived. For all disorders, the genetic correlations were consistent with the findings based on the composites. Further details are available from the first author.

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