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A twin study of self-reported psychopathic personality traits

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Abstract

Previous twin studies attempting to assess the origins of psychopathic personality traits have mainly focused on an overt behavioral conceptualization of the syndrome as defined by a history of chronic antisocial behaviors. This investigation instead focused on a personality-based approach which emphasizes maladaptive personality traits as central to the syndrome. Psychopathic traits were indexed by the Psychopathic Personality Inventory (PPI), a self-report measure designed to assess the personality domain of the disorder. Biometric parameters obtained from the responses of 353 male twins from the Minnesota Twin Registry revealed significant genetic influences, largely non-additive in nature. Although preliminary due to the modest sample size, the findings encourage a larger scale investigation with greater statistical power to evaluate competing models of genetic influence.
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1. Introduction

The construct of psychopathy has been the topic of extensive investigation. The disorder is defined by a conjunction of affective, behavioral, and interpersonal features including egocentricity, fearlessness (Lykken, 1982a), impulsivity (Zuckerman, 1978), shallow emotions, lack of empathy or guilt, manipulativeness, and recurrent violations of social norms (Cleckley, 1941/1988). However, psychopathy research has been plagued throughout its history by a lack of consensus regarding the conceptualization of the syndrome. In clarifying the conceptual boundaries of psychopathy, two prominent approaches have emerged. One group of scholars view psychopathy primarily from a personality-based approach (e.g. Hare, 1970; Lilienfeld, 1994;

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Lilienfeld & Andrews, 1996; Lykken 1995; McCord & McCord, 1964). This is exemplified by Cleckley's classic clinical description of psychopathy as a constellation of deviant personality traits. Other scholars, however, (e.g. Cloninger, 1978; Spitzer, Endicott, & Robins, 1975) conceptualize psychopathy as a behavioral syndrome that should instead be operationalized in terms of a history of chronic antisocial behaviors. Such behavioral, categorical conceptualizations continue to dominate the current version of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV [American Psychiatric Association, 1994]; Miller, Lynam, Widiger, & Leukefeld, 2001; Widiger, 1997; Widiger & Clark, 2000).

Although previous behavior genetic studies have attempted to ascertain the relative influence of genetic and environmental etiological factors to the syndrome by adopting a behavioral approach, they have not specifically tapped the core personality features as defined by Cleckley. Therefore, this investigation sought to determine via twin methodology the relative genetic and environmental contributions to the variance in the personality construct of psychopathy.

1.1. Behavioral genetic studies

A multitude of prior twin and adoption studies have examined genetic and environmental influences on criminality (Coccaro & McNamee, 1998; Eysenck & Eysenck, 1978; Goldsmith & Gottesman, 1996; Hutchings & Mednick, 1975; Tehrani & Mednick, 2001), juvenile delinquency (Jacobson, Prescott, & Kendler, 2000; Jacobson, Prescott, Neale, & Kendler, 2000; Rowe, 1983, 1986; Taylor, Iacono, & McGue, 2000), and adult antisocial behavior (Cadoret, Troughton, Bagford, & Woodworth, 1990; Cadoret, Troughton, & O'Gorman, 1987; Crowe, 1974; Dilalla & Gottesman, 1989; Krueger, Hicks, & McGue, 2001a; Lyons, True, Eisen et al., 1995; McGuffin & Thapar, 1998). These studies have for the most part utilized behavioral measures related to criminality as well as criteria and symptom counts for the diagnosis of Antisocial Personality Disorder (ASPD) from the DSM-III (APA, 1980), DSM-III-R (APA, 1987), or DSM-IV (APA, 1994).

Research of this kind has consistently revealed substantial genetic contributions to the antisocial phenotype (c.f. Dilalla & Gottesman, 1989). A review of the literature also suggests that while the shared or family environment may promote adolescence-limited delinquency (Moffitt, 1993), genetic influence is more prominent among those individuals who start young and continue to engage in a pattern of life-course persistent antisocial behavior (see also Dilalla & Gottesman, 1989; Lyons et al., 1995; Moffitt, 1993; Taylor Iacono, & McGue, 2000; for exceptions see Rowe, 1983, 1986). For example, Taylor, Iacono, and McGue (2000), reported probandwise concordance rates for twins selected as either early starter (i.e. life-course persistent) or late starter (i.e. adolescence-limited) antisocial phenotypes. With respect to the early starter phenotype, co-twins of monozygotic (MZ) pairs were at a substantially greater risk than co-twins in dizygotic (DZ) pairs (55 and 29% probandwise concordances, respectively). In contrast, for the late starter phenotype, there was relatively little differential risk to co-twins based on zygosity (MZ and DZ probandwise concordance, 43 and 39%, respectively).

Furthermore, in behavior genetic studies of adult criminality, MZ twins typically have higher concordance rates than DZ twins (Dilalla & Gottesman, 1989; Eysenck & Eysenck, 1978; McGuffin & Thapar, 1998; Tehrani & Mednick, 2001). In a review of genetic studies of adult criminality, Goldsmith and Gottesman (1996) calculated MZ and DZ pairwise concordance rates

of 52% and 23%, respectively, indicating that MZ twins are more than twice as likely to be concordant for criminal conduct than DZ twins. In another review, Dilalla and Gottesman (1989) noted an average MZ pairwise concordance of 51% and DZ pairwise concordance rate of 22% across several studies.

In addition, adoption studies of criminality have provided evidence for genetic contributions to the etiology of antisocial behavior. Several investigations and reviews have shown that adoptees with biological parents who were criminal had higher rates of criminal convictions than control samples of adoptees without such pedigrees (Crowe, 1974; Hutchings & Mednick, 1975; Tehrani & Mednick, 2001). Data from two sets of adult adoptees also support the importance of genetic influences on antisocial behavior as diagnosed by DSM- III criteria (Cadoret et al., 1987, 1990).

1.2. Psychopathy

Although these findings regarding heritable influences on antisocial behavior have appeared consistently across several studies and designs, their relevance to the construct of psychopathy is unclear. Essentially, these studies have all focused on the etiology of behaviors associated with ASPD, delinquency, and criminality. These investigations have not examined psychopathy in terms of a constellation of maladaptive personality traits as delineated by Cleckley (1941/1988).

A system that exists for assessing this alternative conceptualization of psychopathy is Hare's (1991) Psychopathy Checklist-Revised (PCL-R). Probably the best validated and most widely used measure of psychopathy, the PCL-R has been consistently utilized in forensic settings and has shown excellent psychometric properties. The PCL-R is a diagnostic inventory consisting of 20 items that is scored on the basis of a structured interview and review of collateral file information.

Factor analysis of the PCL-R has yielded a latent two-factor model of psychopathy (Harpur, Hare, & Hakstian, 1989). Factor 1, encompasses emotional detachment and affective-interpersonal features of psychopathy, including many of the core personality traits suggested by Cleckley (1941/1988) such as lack of guilt, shallow affect, and narcissism. Factor 2, in contrast, relates to an impulsive, antisocial lifestyle, and comprises many chronic behaviors typical of ASPD such as a parasitic dependence, juvenile delinquency, and irresponsibility. These two facets of psychopathy show differing patterns of relations with various external criteria, including personality trait measures (Harpur et al., 1989; Patrick, 1994; Verona, Patrick, & Joiner, 2001) and indices of emotional reactivity (Patrick, 1994; Patrick, Bradley, & Lang, 1993).

Based on the operational criteria used in the previously mentioned behavior genetic designs, these studies appear to have tapped the antisocial behaviors related to Factor 2 of the PCL-R, but not the core personality traits of Factor 1. Although the two factors are moderately correlated, it is important to determine whether there is differential heritability for the personality dimension of psychopathy. However, the PCL-R was constructed specifically for use within prison populations where researchers have access to file data. An alternative approach is to use a well-validated self-report personality inventory to investigate the etiology of psychopathy in non-criminal populations. This issue is vital given the fact that many psychopathic individuals may function successfully outside of prisons at a sub-clinical level (Ishikawa, Raine, Lencz, Bihrle, & Lacasse, 2001; Levenson, Kiehl, & Fitzpatrick, 1995; Widom, 1977).

While analysis of normal range personality traits relevant to psychopathy and other antisocial phenotypes has also revealed genetic contributions (Taylor, McGue, Iacono, & Lykken, 2000;

Tellegen et al., 1988; Zuckerman, Buchsbaum, & Murphy, 1980), well-known self-report psychopathy scales such as the Minnesota Multiphasic Personality Inventory (MMPI) Psychopathic Deviate (Pd) scale (McKinley & Hathaway, 1944) and the California Psychological Inventory (CPI) Socialization (So) scale (Gough, 1960), appear not to assess the core personality traits outlined by Cleckley. Empirically, these instruments correlate primarily with the antisocial deviance facet (Factor 2) of the PCL-R, rather than the affective-interpersonal facet (Factor 1; Hare & Cox, 1978; Lilienfeld & Andrews, 1996). Studies examining the Constraint superfactor of the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1978/1982), which is closely linked to externalizing and antisocial behavior (Krueger, 1999; Krueger, Caspi, Moffitt, Silva, & McGee, 1996; Krueger, McGue, & Iacono, 2001; Krueger et al., 1994), are insufficient as well. Evidence from an analysis by Verona et al. (2001), shows that normal range traits related to Constraint also correlate primarily with Factor 2 rather than Factor 1 of the PCL-R. Consequently, twin studies using these self-report indices of personality, although informative, may fail to measure important aspects of psychopathic personality per se.

To facilitate research aimed at comparing the validity of the personality and behavior-based approaches, Lilienfeld and Andrews (1996) introduced the Psychopathic Personality Inventory (PPI). The PPI was constructed specifically to assess the core personality traits of psychopathy rather than the antisocial behavior facet. The criterion validity of the PPI has been demonstrated through correlations with the PCL-R. Analyses from a study in which the PPI and PCL-R were administered to prison inmates (Poythress, Edens, & Lilienfeld, 1998) revealed that most of the unique variance in PPI scores was associated with the affective-interpersonal features (Factor 1) rather than the antisocial deviance features (Factor 2) of the PCL-R. This attests to the validity of the PPI as a measure of the core personality facet of psychopathy.

1.3. The present study

This synopsis of the behavioral genetic research on psychopathy leads us to the primary objectives of the present study. Previous behavior genetic research has addressed issues related to etiological influences on antisocial behavior as characterized by ASPD and chronic criminality. The current investigation, in contrast, seeks to determine the relative genetic and environmental contributions to variance in the core personality facet of psychopathy. We used the PPI to assess this dimension. As such, this study was the first behavior genetic study to examine the etiology of psychopathy using a personality-based approach.

We expected the results to reveal a substantial genetic influence. While it is difficult to predict the degree and type of genetic loading, twin studies of personality in some instances have suggested effects of genetic non-additivity for complex personality traits (Finkel & McGue, 1997; Tellegen et al., 1988). Non-additive genetic effects consist of dominance, epistasis and emergensis. The first two effects involve the interaction among genes within and across loci, respectively. Emergenesis, however, entails several traits, genetically determined independently from one another, combining in a configural manner.

In summary, this investigation sought to assess the relative genetic and environmental contributions to variance in the personality trait dimension of psychopathy. Previous investigations have repeatedly demonstrated the heritability of relevant maladaptive personality traits, as well as

antisocial behavior operationalized by criminality and criteria from the DSM. However, it is unclear whether these findings are applicable to the personality domain of psychopathy.

2. Method

2.1. Participants

Participants were 165 monozygotic (MZ) and 106 dizygotic (DZ) male twin pairs ($N = 542$ individuals) from the Minnesota Twin Registry (MTR) born between the years of 1961 – 1964. The MTR is a birth-record based registry of twins born in Minnesota. Ascertainment procedures for the MTR have been described previously (Lykken, Bouchard, McGue, & Tellegen, 1990). Twin pairs still living and intact were located and recruited by mail. During the original recruitment of twins, a biographical questionnaire completed by the twins provided information about zygosity. Specifically, five questions concerning twin similarity were included in the biographical questionnaire to determine zygosity. Serological analysis of blood samples from 74 twin pairs who had participated in an earlier lab study demonstrated that the overall validity of the questionnaire method of zygosity determination was 96% (Lykken et al., 1990).

The rationale for selecting this particular sample from the Registry was twofold. First, most existing empirical work on psychopathy has dealt with younger adult samples. Therefore, we chose this sample because it represents the youngest cohort from the Registry. Secondly, this limited age range should minimize any cohort or age effects that may spuriously influence the results. Because this specific cohort includes only male twins born between the years of 1961 – 1964, only male twin pairs were included in this investigation. Although it would be advantageous to include female twins in order to examine gender differences in the personality dimension of psychopathy, women tend to be lower than men in psychopathy-related traits such as aggression and fearlessness (Tellegen, 1978/1982). Although variance differences between the two groups are most relevant to biometrical analysis, Lilienfeld and Andrews (1996) note in their preliminary validation article on the PPI that males, ''may also exhibit greater variance on such traits'' (p. 493).

Three hundred and fifty-three of the 540 individual twins who were contacted and received the questionnaire (two individuals were never relocated) agreed to participate by returning the PPI, yielding a response rate of 65.4% of the individuals contacted. This final sample consisted of 89 intact MZ and 47 intact DZ twin pairs, plus 81individuals whose co-twin did not choose to participate. Scores on the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1978/1982) were available for the 542 individual twins targeted for inclusion in this study. The MPQ is an omnibus index of personality, consisting of 11 primary scales organized around three superfactors: Positive Emotionality (PEM), Negative Emotionality (NEM), and Constraint (CON; Krueger, 2000; Tellegen, 1985). MPQ scores for responders and non-responders were compared in order to determine if the personality profiles of the responders were representative of the target sample. Effect sizes comparing responders and non-responders were minimal, with the mean Cohen's $d = 0.15$ (range 0.04–0.31) for all subscales and superfactors, suggesting that the personalities of responders were representative of the larger target sample. Effect sizes comparing responders and non-responders were also minimal for socioeconomic status as measured by income level ($d = 0.22$) and for age ($d = 0.11$).

2.2. Measure

As noted earlier, the Psychopathic Personality Inventory (PPI), was used in the present study because of evidence supporting its validity as a self-report index of the affective-interpersonal facet of psychopathic personality rather than the antisocial behavior facet per se. The inventory contains 187 questions developed using an exploratory approach to test construction. Items are answered using a four-option (false, mostly false, mostly true, true) Likert-type scale. Items assessing antisocial behaviors were explicitly avoided in developing the PPI so as to provide a "purer" measure of the personality facet of psychopathy. The entire inventory is comprised of a total score, interpretable as a global index of psychopathy, as well as eight sub-scales assessing specific traits relevant to psychopathy (see Table 1 for scale descriptions).

The PPI shows satisfactory psychometric properties as well as evidence of validity based on studies of undergraduate and forensic samples (Lilienfeld & Andrews, 1996; Poythress et al., 1998). Internal consistencies for the PPI total score in these studies (estimated by Cronbach's alpha) range from 0.90 to 0.93; sub-scale consistencies range from 0.70 to 0.91. Test-retest reliability (with a mean test-retest interval of 26 days) for the PPI total score is 0.95, with reliabilities for the sub-scales ranging from 0.82 to 0.94. In terms of validity, the PPI shows substantial positive correlations with several indices of Cleckley's (1941/1988) classic clinical description of the psychopath, as well as appropriate discriminant and convergent relations with other measures of psychopathology (Lilienfeld & Andrews, 1996).

2.3. Procedure

The PPI was mailed to the aforementioned sample of MTR male twins together with a cover letter and a consent form describing the study and inviting participation. The questionnaire was titled "Personality Styles Inventory." Participants were requested to complete the measure in its entirety and return it in the self-addressed envelope provided. Protocols were screened for completeness, and data were double entered to ensure accuracy. Twins who did not return the inventory after a specified time were subsequently prompted via a postcard and telephone call.

Table 1
Reliability estimates and scale descriptions for PPI total score and sub-scales ($n = 353$)[a]

PPI Scale	α	Description of a high scorer
Total Score (163 items)	0.89	A global index of psychopathic personality encompassing all sub-scales.
Machievellian Egocentricity (30 items)	0.80	Is narcissistic and self-centered in interpersonal functioning.
Social Potency (24 items)	0.89	Charismatic; skilled at influencing and manipulating others.
Fearlessness (19 items)	0.81	A risk taker; lacking anticipatory anxiety concerning harm.
Coldheartedness (21 items)	0.78	Has a callous and guiltless emotional style; emotionally detached.
Impulsive Nonconformity (17 items)	0.75	Reckless and rebellious; indifferent towards societal conventions.
Blame Externalization (18 items)	0.89	Rationalizes transgressions; blames others for one's problems.
Carefree Nonplanfulness (20 items)	0.79	Is impulsive, does not plan; lacking in forethought.
Stress Immunity (11 items)	0.77	Lacks anxiety when faced with significant stressful events.

[a] The total PPI score is the sum of all items on all subscales, as well as three additional items that are not included in specific subscales.

2.4. Statistical methods

Twin methodology involves the examination of the differential effects of genotype and environment by comparing the phenotypic similarity within MZ twin pairs with the phenotypic similarity within DZ twin pairs. Genetic influences are composed of additive and non-additive effects. The former is the extent to which genes summate and contribute to an individual's phenotype (Plomin, Defries, McClearn, & Rutter, 1997). MZ twins share 100% of their additive genetic effects while DZ twins share, on average, 50%. Non-additive effects, in turn, may be conceived in terms of dominance, epistasis, and emergenesis (Lykken, 1982b; Lykken, McGue, Tellegen, & Bouchard, 1992).

Dominance is related to the interaction within genes at a given locus. If dominance occurs, average allelic effects do not add up in a simple linear fashion. In epistasis, a particular allele interacts not only with alleles at the same locus on the homologous chromosome, but also with alleles at other loci. Thus, the intra-locus interaction of dominance and the inter-locus interaction of epistasis represent genetic influences that do not breed true from parents to offspring (Loehlin, 1992; Plomin et al., 1997). Moreover, because MZ twins share the exact same genetic configuration while DZ twins do not, dominance yields predicted DZ correlations equal to one-quarter of the MZ estimates, while epistatic effects yield predicted DZ correlations no greater than for unrelated participants drawn at random from the population (i.e. $r = 0$). Emergenesis involves several independent traits combining in a configural fashion. It is typically inferred when DZ correlations are near zero for phenotypes putatively influenced by several traits (Lykken, 1982b).

Environmental effects can also contribute to an individual's phenotype. These influences can be parsed into shared and non-shared environmental influences. The former refer to any environmental factor that members of twin pairs have in common that produces similarities (e.g. social class, parenting styles), while the latter entails external factors whose effects are specific to one twin and not the other (e.g. peer groups, random accidents). The non-shared environmental component also reflects measurement error and state fluctuations.

To decompose the phenotypic variance into its relative genetic and environmental contributions, we estimated genetic and environmental parameters by the method of maximum likelihood using MX software (Neale, 1994). One index of model fit, the root mean square error of approximation (RMSEA), was used to evaluate the absolute fit of the model. This index is used to determine if a specific model fits the data, but not to select the most optimal model among several competing models. The Bayesian Information Criterion ($\mathrm{BIC} = \chi^2 - \mathrm{df} \ln N$; Raftery, 1995), was utilized to assess the comparative fit among competing models of genetic and environmental influence. BIC provides an index of the extent to which each model maximizes correspondence between the observed and model predicted variances and covariances, while minimizing the number of parameters. Better fitting models have more negative values (Raftery, 1995).

Several models were fit for the total score as well as each sub-scale. Fitting a full model, or ACE model, estimating parameters with additive genetic influences (A) as well as shared (C) and non-shared environmental (E) contributions began the process. Subsequently, some parameters were dropped and added in order to accommodate models estimating non-additive genetic parameters of dominance (D) and epistasis or emergenesis (I).

3. Results

3.1. Descriptive statistics

Table 2 lists the means, standard deviations and ranges of the PPI total score and sub-scales for all subjects as well as MZ and DZ twins separately. In order to determine if any differences existed due to birth order or zygosity, separate MANOVA were performed for each of these factors as an omnibus test across all PPI scales. No significant difference was found between the older and younger members of the twin pairs, $F(9, 343) = 0.591$, ns, or between MZ versus DZ twin membership, $F(9, 343) = 1.069$, ns.

Reliability estimates, as indexed by Cronbach's alpha are listed in Table 1. Internal consistency for the PPI total score was 0.89, with consistencies for sub-level trait scales ranging from 0.75 to 0.89. These results provide further evidence of the reliability of the PPI, supplementing that reported in its development and preliminary validation (Lilienfeld & Andrews, 1996).

3.2. Intraclass correlations

Prior to the final analyses, inspection of the frequency distributions for the total and eight subscale scores revealed some positive skewness. Therefore, scale scores were transformed using Blom's rank transformation method (Blom, 1958) in order to normalize their distributions. This procedure has been shown to optimize biometric model selection based on a variety of simulations (van den Oord et al., 2000).

Table 3 shows intraclass correlations for the MZ and DZ twins, along with 95% confidence intervals and *P*-values for the difference between MZ and DZ twin correlations on the total score and sub-scales of the PPI. The significance of the correlations was determined using a one-tailed *z*-test based on the a priori assumption that MZ resemblance is greater than DZ resemblance. MZ

Table 2
Descriptive statistics for PPI total score and sub-scales[a]

PPI scale	All subjects			MZ		DZ	
	M	SD	Min/max	*M*	SD	*M*	SD
TS	348.43	30.64	(266, 441)	350.56	30.73	344.64	30.23
McEgo	58.16	9.15	(36, 93)	58.46	9.00	57.63	9.42
SocP	60.94	11.15	(34, 91)	61.89	11.09	59.26	11.11
Frless	44.92	8.75	(19, 66)	45.30	8.97	44.24	8.33
Cldhrtd	49.17	7.70	(29, 74)	49.62	7.39	48.38	8.20
ImpNcf	32.25	6.27	(18, 50)	32.16	6.43	32.40	6.00
BlmExt	30.29	8.03	(18, 61)	30.12	8.06	30.60	7.99
CfNonp	35.07	6.46	(21, 55)	35.12	5.96	34.98	7.28
StrIm	32.29	4.93	(18, 43)	32.61	4.87	31.72	5.01

[a] $n = 353$ (all subjects), $n(\text{MZ}) = 226$, $n(\text{DZ}) = 127$ (includes both twin pairs and individuals whose co-twin did not respond); Min/Max = low and high scale score; TS = Total Score; McEgo = Machievellian Egocentricity; SocP = Social Potency; Frless = Fearlessnesss; Cldhrtd = Coldheartedness; ImpNcf = Impulsive Nonconformity; BlmExt = Blame Externalization; CfNonp = Carefree Nonplanfulness; StrIm = Stress Immunity.

Table 3
Intra-class correlations for PPI scale scores[a]

PPI scale	r_{MZ}	CI(95%)	r_{DZ}	CI(95%)	P(MZ/DZ)
Total score	0.46*	(0.28 – 0.61)	−0.26	(−0.51 –0.03)	0.00
Machievellian Egocentricity	0.28*	(0.08 – 0.46)	−0.09	(−0.37 –0.20)	0.02
Social Potency	0.54*	(0.37 – 0.67)	0.21	(−0.08 –0.47)	0.02
Fearlessness	0.54*	(0.37 – 0.67)	0.03	(−0.26 –0.31)	0.00
Coldheartedness	0.34*	(0.14 – 0.51)	−0.16	(−0.43 –0.13)	0.00
Impulsive Nonconformity	0.51*	(0.34 – 0.65)	−0.05	(−0.33 –0.24)	0.00
Blame Externalization	0.57*	(0.41 – 0.70)	0.16	(−0.13 –0.43)	0.00
Carefree Nonplanfulness	0.31*	(0.11 – 0.49)	−0.16	(−0.43 –0.13)	0.00
Stress Immunity	0.43*	(0.24 – 0.59)	−0.08	(−0.36 –0.21)	0.00

[a] All scores were transformed using Blom's rank transformation method; n(MZ) = 89 pairs; n(DZ) = 47 pairs; *P-values significant 0.05 (one-tailed); r_{MZ} = MZ intraclass correlation; r_{DZ} = DZ intraclass correlation; CI(95%) = 95% Confidence Intervals; P(MZ/DZ) = significance of difference between MZ and DZ correlations.

intraclass correlations were all statistically significant and ranged from 0.28 to 0.57 ($P < 0.05$). In contrast, none of the DZ intraclass correlations achieved significance. All DZ correlations were small and in most cases negative, albeit not significantly different from zero. Correlations for each scale were also found to be significantly different between the MZ and DZ twins (all P values $\leqslant 0.02$).

3.3. Biometrical modeling

Although intraclass correlations are often informative, they can be misleading if MZ and DZ variances differ. Therefore, MZ and DZ co-variance matrices were used to fit biometrical models. This procedure permitted examination of parameter estimates and comparisons of the relative fit of competing models of genetic and environmental influence. The best fitting model was selected as the model yielding the greatest negative value for BIC. Table 4 lists the results of all the models that were tested for each scale including parameter estimates with 95% confidence intervals using the method of maximum likelihood from the MX Software (Neale, 1994). The best fitting model for each scale is highlighted in bold. In general, models including only epistatic or emergenic (I) and non-shared environmental (E) parameters provided the best fit to the data. The exceptions were the Social Potency and Blame Externalization sub-scales, which were best represented by models containing additive genetic (A) and non-shared environmental influences[1]. Additionally, there was also substantial agreement across all fit indices, with χ^2, RMSEA, and BIC exhibiting agreement on the best fitting model for nearly every scale.

[1] During analysis of the Carefree Nonplanfulness scale of the PPI, none of the biometric models provided an adequate fit to the data (see Table 4). Upon further examination this was deemed attributable to different MZ and DZ variances as well as a negative DZ covariance. Subsequently, the MZ and DZ intraclass correlations for the scale were used in model fitting in place of the covariance matrices to rectify the matter. This yielded an acceptable fit on several of the models, the best of which was the IE model. In addition, parameter estimates for each model tested with intraclass correlations were comparable to the estimates from the analyses using the covariance matrices.

Table 4
Biometrical model fitting results for PPI total score and sub-scales[a]

Model[b]	Fit indices					Variance components				
	df	χ^2	*P*	BIC	RMSEA	a^2	d^2	i^2	c^2	e^2
Total score										
ACE	3	12.267	0.007	−2.47	0.207	0.395 (0.17)(0.56)	–	–	0.0 (0.0)(0.15)	0.605 (0.44)(0.80)
ADE	3	7.975	0.047	−6.76	0.137	0.0 (0.0)(0.38)	0.448 (0.04)(0.60)	–	–	0.552 (0.40)(0.74)
AE	4	12.267	0.015	−7.38	0.154	0.395 (0.20)(0.56)	–	–	–	0.605 (0.44)(0.80)
CE	4	20.142	0.000	0.49	0.256	–	–	–	0.228 (0.06)(0.38)	0.773 (0.62)(0.94)
E	5	27.261	0.000	2.698	0.202	–	–	–	–	1.000 (1.0)(1.0)
AIE	3	4.592	0.204	−10.15	0.085	0.0 (0.0)(0.26)	–	0.472 (0.19)(0.61)	–	0.528 (0.39)(0.70)
CIE	3	4.592	0.204	−10.15	0.085	–	–	0.472 (0.28)(0.61)	0.0 (0.0)(0.13)	0.528 (0.39)(0.70)
DIE	3	4.592	0.204	−10.15	0.085	–	0.0 (0.0)(0.50)	0.472 (0.0)(0.61)	–	0.528 (0.39)(0.70)
IE	**4**	**4.592**	**0.332**	**−15.06**	**0.064**	**–**	**–**	**0.472 (0.30)(0.61)**	**–**	**0.528 (0.39)(0.70)**
Machievellian Egocentricity										
ACE	3	2.983	0.394	−11.76	0.064	0.235 (0.0)(0.42)	–	–	0.0 (0.0)(0.25)	0.765 (0.58)(0.96)
ADE	3	1.768	0.622	−12.97	0.027	0.0 (0.0)(0.39)	0.269 (0.0)(0.45)	–	–	0.731 (0.55)(0.94)
AE	4	2.983	0.561	−16.67	0.042	0.235 (0.04)(0.42)	–	–	–	0.765 (0.58)(0.96)
CE	4	5.317	0.256	−14.33	0.062	–	–	–	0.149 (0.0)(0.31)	0.851 (0.69)(1.0)
E	5	8.329	0.139	−16.23	0.075	–	–	–	–	1.000 (1.0)(1.0)
AIE	3	0.852	0.837	−13.89	0.000	0.0 (0.0)(0.36)	–	0.288 (0.0)(0.46)	–	0.712 (0.54)(0.92)
CIE	3	0.852	0.837	−13.89	0.000	–	–	0.288 (0.02)(0.46)	0.0 (0.0)(0.20)	0.712 (0.54)(0.92)
DIE	3	0.852	0.837	−13.89	0.000	–	0.0 (0.0)(0.43)	0.288 (0.0)(0.46)	–	0.712 (0.54)(0.92)
IE	**4**	**0.852**	**0.931**	**−18.80**	**0.000**	**–**	**–**	**0.288 (0.08)(0.46)**	**–**	**0.712 (0.54)(0.92)**
Social Potency										
ACE	3	3.482	0.323	−11.26	0.050	0.538 (0.15)(0.67)	–	–	0.0 (0.0)(0.32)	0.462 (0.33)(0.63)
ADE	3	2.990	0.393	−11.75	0.008	0.183 (0.0)(0.66)	0.367 (0.0)(0.67)	–	–	0.450 (0.33)(0.61)
AE	**4**	**3.482**	**0.481**	**−16.17**	**0.000**	**0.538 (0.37)(0.67)**	**–**	**–**	**–**	**0.462 (0.33)(0.63)**

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Table 4 (*continued*)

Model[b]	Fit indices					Variance components				
	df	χ^2	*P*	BIC	RMSEA	a^2	d^2	i^2	c^2	e^2
CE	4	10.013	0.040	−9.64	0.155	–	–	–	0.407 (0.26)(0.54)	0.593 (0.46)(0.74)
E	5	34.233	0.000	9.67	0.221	–	–	–	–	1.000 (1.0)(1.0)
AIE	3	2.990	0.393	−11.75	0.008	0.367 (0.0)(0.66)	–	0.183 (0.0)(0.64)	–	0.450 (0.33)(0.61)
CIE	3	2.990	0.393	−11.75	0.008	–	–	0.367 (0.09)(0.64)	0.183 (0.0)(0.42)	0.450 (0.33)(0.61)
DIE	3	3.108	0.375	−11.63	0.024	–	0.553 (0.0)(0.67)	0.0 (0.0)(0.65)	–	0.447 (0.33)(0.61)
IE	4	4.813	0.307	−14.84	0.058	–	–	0.550 (0.39)(0.67)	–	0.450 (0.33)(0.61)
Fearlessness										
ACE	3	6.826	0.078	−7.91	0.142	0.494 (0.16)(0.63)	–	–	0.0 (0.0)(0.29)	0.506 (0.37)(0.67)
ADE	3	5.254	0.154	−9.48	0.093	0.0 (0.0)(0.60)	0.509 (0.0)(0.64)	–	–	0.491 (0.36)(0.65)
AE	4	6.826	0.145	−12.83	0.106	0.494 (0.33)(0.63)	–	–	–	0.506 (0.37)(0.67)
CE	4	13.438	0.009	−6.21	0.195	–	–	–	0.387 (0.23)(0.52)	0.613 (0.48)(0.77)
E	5	35.182	0.000	10.62	0.187	–	–	–	–	1.000 (1.0)(1.0)
AIE	3	4.855	0.183	−9.88	0.061	0.038 (0.0)(0.59)	–	0.475 (0.0)(0.64)	–	0.487 (0.36)(0.64)
CIE	3	4.855	0.183	−9.88	0.061	–	–	0.494 (0.15)(0.64)	0.019 (0.0)(0.34)	0.487 (0.36)(0.64)
DIE	3	4.855	0.183	−9.88	0.061	–	0.076 (0.0)(0.63)	0.437 (0.0)(0.64)	–	0.487 (0.36)(0.64)
IE	**4**	**4.867**	**0.301**	**−14.78**	**0.046**	**–**	**–**	**0.513 (0.36)(0.64)**	**–**	**0.487 (0.36)(0.64)**
Coldheartedness										
ACE	3	8.883	0.031	−5.86	0.166	0.279 (0.0)(0.47)	–	–	0.0 (0.0)(0.18)	0.721 (0.53)(0.93)
ADE	3	6.219	0.101	−8.52	0.114	0.0 (0.0)(0.39)	0.345 (0.0)(0.52)	–	–	0.655 (0.48)(0.87)
AE	4	8.883	0.064	−10.77	0.115	0.279 (0.07)(0.47)	–	–	–	0.721 (0.53)(0.93)
CE	4	12.856	0.012	−6.80	0.189	–	–	–	0.138 (0.0)(0.30)	0.862 (0.70)(1.00)
E	5	15.423	0.009	−9.14	0.144	–	–	–	–	1.000 (1.0)(1.0)
AIE	3	4.040	0.257	−10.70	0.076	0.0 (0.0)(0.28)	–	0.379 (0.05)(0.55)	–	0.621 (0.45)(0.83)

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Table 4 (*continued*)

Model[b]	Fit indices					Variance components				
	df	χ^2	*P*	BIC	RMSEA	a^2	d^2	i^2	c^2	e^2
CIE	3	4.040	0.257	−10.70	0.076	–	–	0.379 (0.15)(0.55)	0.0 (0.0)(0.14)	0.621 (0.45)(0.83)
DIE	3	4.040	0.257	−10.70	0.076	–	0.0 (0.0)(0.47)	0.379 (0.0)(0.55)	–	0.621 (0.45)(0.83)
IE	**4**	**4.040**	**0.401**	**−15.61**	**0.054**	**–**	**–**	**0.379 (0.17)(0.55)**	**–**	**0.621 (0.45)(0.83)**
Impulsive Nonconformity										
ACE	3	5.986	0.112	−8.75	0.118	0.469 (0.18)(0.61)	–	–	0.0 (0.0)(0.23)	0.531 (0.39)(0.70)
ADE	3	3.603	0.308	−11.14	0.048	0.0 (0.0)(0.56)	0.493 (0.0)(0.63)	–	–	0.507 (0.38)(0.67)
AE	4	5.986	0.200	−13.67	0.078	0.469 (0.30)(0.61)	–	–	–	0.531 (0.39)(0.70)
CE	4	13.359	0.010	−6.29	0.195	–	–	–	0.342 (0.18)(0.48)	0.658 (0.52)(0.82)
E	5	30.030	0.000	5.47	0.173	–	–	–	–	1.000 (1.0)(1.0)
AIE	3	2.453	0.484	−12.29	0.006	0.0 (0.0)(0.51)	–	0.501 (0.0)(0.63)	–	0.499 (0.37)(0.66)
CIE	3	2.453	0.484	−12.29	0.006	–	–	0.501 (0.21)(0.63)	0.0 (0.0)(0.26)	0.499 (0.37)(0.66)
DIE	3	2.453	0.484	−12.29	0.006	–	0.0 (0.0)(0.61)	0.501 (0.0)(0.63)	–	0.499 (0.37)(0.66)
IE	**4**	**2.453**	**0.653**	**−17.20**	**0.000**	**–**	**–**	**0.501 (0.34)(0.63)**	**–**	**0.499 (0.37)(0.66)**
Blame Externalization										
ACE	3	1.999	0.573	−12.74	0.023	0.556 (0.16)(0.68)	–	–	0.0 (0.0)(0.35)	0.444 (0.33)(0.60)
ADE	3	1.382	0.710	−13.36	0.000	0.084 (0.0)(0.67)	0.480 (0.0)(0.68)	–	–	0.435 (0.32)(0.58)
AE	**4**	**1.999**	**0.736**	**−17.65**	**0.000**	**0.556 (0.40)(0.68)**	**–**	**–**	**–**	**0.444 (0.33)(0.60)**
CE	4	8.822	0.066	−10.83	0.139	–	–	–	0.450 (0.30)(0.57)	0.550 (0.43)(0.70)
E	5	39.069	0.000	14.51	0.197	–	–	–	–	1.000 (1.0)(1.0)
AIE	3	1.382	0.710	−13.36	0.000	0.325 (0.0)(0.67)	–	0.240 (0.0)(0.67)	–	0.435 (0.32)(0.58)
CIE	3	1.382	0.710	−13.36	0.000	–	–	0.403 (0.10)(0.67)	0.162 (0.0)(0.43)	0.435 (0.32)(0.58)
DIE	3	1.400	0.706	−13.34	0.000	–	0.565 (0.0)(0.68)	0.0 (0.0)(0.67)		0.435 (0.32)(0.58)
IE	4	2.370	0.668	−17.28	0.000	–	–	0.565 (0.42)(0.68)	–	0.435 (0.32)(0.59)

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Table 4 (*continued*)

Model[b]		Fit indices				Variance components				
	df	χ^2	*P*	BIC	RMSEA	a^2	d^2	i^2	c^2	e^2
Carefree Nonplanfulness										
ACE	3	15.215	0.002	0.48	0.255	0.242 (0.0)(0.45)	–	–	0.0 (0.0)(0.17)	0.758 (0.55)(0.99)
ADE	3	12.490	0.006	−2.25	0.223	0.0 (0.0)(0.36)	0.330 (0.0)(0.52)	–	–	0.670 (0.48)(0.91)
AE	4	15.215	0.004	−4.44	0.211	0.242 (0.01)(0.45)	–	–	–	0.758 (0.55)(0.99)
CE	4	18.371	0.001	−1.28	0.240	–	–	–	0.093 (0.0)(0.26)	0.907 (0.74)(1.00)
E	5	19.541	0.002	−5.02	0.206	–	–	–	–	1.000 (1.0)(1.0)
AIE	3	10.053	0.018	−4.69	0.194	0.0 (0.0)(0.25)	–	0.377 (0.07)(0.55)	–	0.623 (0.45)(0.85)
CIE	3	10.053	0.018	−4.69	0.194	–	–	0.377 (0.14)(0.55)	0.0 (0.0)(0.13)	0.623 (0.45)(0.85)
DIE	3	10.053	0.018	−4.69	0.194	–	0.0 (0.0)(0.45)	0.377 (0.0)(0.55)	–	0.623 (0.45)(0.85)
IE	**4**	**10.053**	**0.040**	**−9.60**	**0.155**	**–**	**–**	**0.377 (0.15)(0.55)**	**–**	**0.623 (0.45)(0.85)**
Stress Immunity										
ACE	3	5.582	0.134	−9.16	0.113	0.378 (0.07)(0.54)	–	–	0.0 (0.0)(0.24)	0.622 (0.46)(0.81)
ADE	3	3.523	0.318	−11.22	0.049	0.0 (0.0)(0.49)	0.408 (0.0)(0.56)	–	–	0.592 (0.44)(0.77)
AE	4	5.582	0.233	−14.07	0.069	0.378 (0.19)(0.54)	–	–	–	0.622 (0.46)(0.81)
CE	4	10.721	0.030	−8.93	0.165	–	–	–	0.265 (0.10)(0.41)	0.735 (0.59)(0.90)
E	5	20.481	0.001	−4.08	0.139	–	–	–	–	1.000 (1.0)(1.0)
AIE	3	2.268	0.519	−12.47	0.005	0.0 (0.0)(0.44)	–	0.421 (0.0)(0.57)	–	0.579 (0.43)(0.76)
CIE	3	2.268	0.519	−12.47	0.005	–	–	0.421 (0.15)(0.57)	0.0 (0.0)(0.22)	0.579 (0.43)(0.76)
DIE	3	2.268	0.519	−12.47	0.005	–	0.0 (0.0)(0.53)	0.421 (0.0)(0.57)	–	0.579 (0.43)(0.76)
IE	**4**	**2.268**	**0.687**	**−17.38**	**0.000**	**–**	**–**	**0.421 (0.24)(0.57)**	**–**	**0.579 (0.43)(0.76)**

[a] df = degrees of freedom; χ^2 = chi-square; *P* = *P*-value; BIC = Bayesian Information Criterion; RMSEA = Root Mean Square Error of Approximation; a^2 = additive genetic variance; d^2 = dominance variance; i^2 = epistatic variance; c^2 = shared environmental variance; e^2 = non-shared environmental variance.

[b] Capital letters denoted under models heading are signified by the aforementioned variance components; Confidence Intervals are listed in parentheses underneath the respective variance components.

4. Discussion

The objective of this investigation was to evaluate genetic and environmental contributions to psychopathy defined in terms of a personality construct. Intraclass correlations and model fitting analyses revealed substantial evidence of genetic contributions to variance in the personality construct of psychopathy. Specifically, the wide disparity between the relatively large and significant MZ correlations and the non-significant (i.e. indistinguishable from zero) DZ correlations appears compatible with the idea of psychopathic personality traits being epistatic or emergenic in nature. While previous studies of antisocial and criminal behavior have typically revealed additive genetic contributions, the current results indicate a non-additive pattern that may be distinct for the personality-based conceptualization of psychopathy. Due to the relatively modest sample size, however, the results are preliminary and will require a larger study with greater statistical power to resolve competing models of genetic influence. Nonetheless, results from correlational analyses and biometric models appear to indicate that the etiology of the personality domain of psychopathy may derive largely from epistatic or emergenic and non-shared environmental contributions.

Emergenesis, although similar in nature to epistasis, is a distinct concept. As spelled out earlier, epistasis involves an interaction between genes across multiple loci as they contribute to a specific phenotype. Emergenesis (Lykken, 1982b; Lykken et al., 1992), on the other hand, refers to a situation in which several heritable traits combine in a configural, rather than additive, manner. Complex traits are considered emergenic if they reflect a non-additive aggregation of basic, metrical traits that are themselves genetically determined independently from one another. Lacking even one gene in such configurations could result in a qualitively different phenotype. Due to the genetic complexity of this mechanism, emergenic traits do not run in families. However, they are evident in studies of twins, since MZ pairs share the exact same genetic configuration. Thus, the large and robust MZ correlations in comparison to the small and insignificant DZ correlations from this investigation suggests a non-additive genetic influence, such as that observed in some previous research with twins on configural phenotypes, such as the human EEG (Lykken, 1982b; Lykken et al., 1992).

In addition, the finding of significant genetic contributions is compatible with previous reports regarding antisocial and criminal behavior reported earlier. Although several scholars would argue that ASPD and criminality do not adequately describe the construct of psychopathy (Lilienfeld, 1994; Lykken, 1995; Patrick, 2001), these findings are not surprising given the overlapping nature of the behavior and personality-based approaches. However, the data do provide preliminary evidence of a distinct genetic architecture for the personality component of the disorder. Moreover, these findings of non-additive genetic influences are consistent with other twin studies measuring normal range personality dimensions. Several studies employing the MPQ as well as other personality inventories indicate that the genetic architecture of the personality domain may be complex, requiring non-additive parameters to accommodate the data (Carey & Rice, 1983; Finkel & McGue, 1997; Tellegen et al., 1988).

Several other findings of this study are also worth noting. First, the PPI demonstrated satisfactory internal consistency as measured by Cronbach's alpha, thereby providing additional evidence for the reliability of the instrument. Secondly, the influence of shared environmental parameters appears to be negligible for all scales. This finding is consistent with evidence reported

across many behavioral genetic analyses of personality (e.g. Finkel & McGue, 1997; Loehlin, 1992; Loehlin & Nichols, 1976; Rowe & Plomin, 1981; Tellegen et al., 1988). Twin studies of juvenile and adult antisocial traits also indicate that contributions from shared environmental parameters diminish as individuals move into adulthood (e.g. Lyons et al., 1995).

4.1. Limitations

There are several limitations of the present investigation. First, the sample size was modest for a biometrical twin design, rendering inferences from the data tentative and preliminary. The small sample size may explain the contrast effects evident from the negative DZ correlations across several of the PPI scales. This bias derives more commonly from ratings given by mothers of young twins (Goldsmith, Buss, & Lemery, 1997; Spinath & Angleitner, 1998). In view of these issues, the results must be considered preliminary and future research with greater statistical power is encouraged to resolve competing models of genetic influence.

Secondly, the study sample included only male twins, and thus is not generalizable to females. It would be beneficial in future endeavors to include both male and female twins in order to assess sex differences within the psychopathy dimension. Such a design could shed light on the controversy over psychopathy's putative relation to histrionic personality disorder (HPD). HPD encompasses personality features analogous in nature to psychopathy yet typically manifests itself among females, leading several authors to speculate that HPD is a female-biased manifestation of psychopathy (Hamburger, Lilienfeld, & Hogben, 1996). A twin study assessing both personality disorders in both male and female twins could aid in clarifying this issue by differentiating the influences contributing to both syndromes.

Third, assortative mating (''like mating with like''), a factor that has been shown to be influential when estimating the relative genetic and environmental contributions to antisocial behavior (Krueger, Moffitt, Caspi, Bleske, & Silva, 1998), was not taken into account here. An assumption of a null effect for the trait in question when an effect does in fact exist leads to an underestimate of heritability (h^2) and an overestimate of shared (c^2) environmental influences (Krueger et al., 1998). However, in the present study, no such evidence was found for c^2 contributions on any of the PPI scales.

Fourth, no efforts were made to gauge the presence of genotype by environment interactions or gene-environment correlations. The presence of these effects could alter the interpretations of the analyses. Notably, in adoption studies, a design that allows for the assessment of interaction effects (since adoptees are usually exposed to various combinations of genetic and environmental factors), evidence has shown that the increase in the number of antisocial behaviors due to both genetic and environmental factors acting together is greater than the predicted increase from either factor acting alone (Cadoret, Cain, & Crowe, 1983; Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995).

Lastly, in addition to conducting a larger scale investigation with greater power to replicate these preliminary findings, it would be advantageous to examine how much genetic variance is shared across the sub-scales of the PPI. Such an approach would allow a glimpse into what scales or facets of the instrument share a common genetic etiology. Due to the modest sample size of this study we did not investigate this issue. Moreover, it is essential that future studies assess both the personality and behavioral aspects of psychopathy with parallel methods, in order to address

the differential heritability of these two factors of the psychopathy phenotype in a direct fashion. Nevertheless, the present results are promising, albeit preliminary, and are suggestive of a non-additive basis for the personality facet of psychopathy.

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