Insecure Attachment and Personality Disorder: A Twin Study of Adults

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Abstract

This study used 239 twin pairs from a volunteer community sample to investigate how anxious and avoidant attachment are related to personality disorder (PD). Factor analysis showed that self-reported anxious attachment and 11 PD scales from the Dimensional Assessment of Personality Problems loaded onto one factor (emotional dysregulation), and avoidant attachment and four PD scales loaded onto a second factor (inhibitedness). Biometric models indicated that 40% of the variance in anxious attachment was heritable, and 63% of its association with corresponding PD dimensions was attributable to common genetic effects. Avoidant attachment was influenced by the shared environment instead of genes. Correlations between avoidance and corresponding PD dimensions were attributable to experiences in the nonshared environment that influenced both variables. Copyright © 2006 John Wiley & Sons, Ltd.

INTRODUCTION

Although Bowlby (1969, 1973, 1980) originally sought to understand how insecure attachment influences affect regulation, personality development and psychopathology in children, his ideas also help to explain how these phenomena are woven together in close relationships between adults. When assessed with self-report questionnaires, insecure attachment in adults varies along two separate dimensions: *anxious attachment* reflects worry about being abandoned or rejected by others, and *avoidant attachment* reflects discomfort with close relationships and depending on others (Brennan, Clark, & Shaver, 1998; Mikulincer, Shaver, & Pereg, 2003). Anxiety and avoidance dimensions in adults are analogous to similar dimensions that have been observed in infants in the 'Strange

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Situation', a laboratory procedure that assesses how infants respond to separations from mother, exposure to an adult stranger and reunions with mother (Ainsworth, Blehar, Waters, & Wall, 1978, fig. 10, p. 102). Brennan et al. (1998) showed how the anxiety dimension in adults corresponds to continuous coding scales used to record infant distress and anger throughout the Strange Situation (e.g. when infants are left alone, joined by a stranger and reunited with mother). The avoidance dimension corresponds to Strange Situation scales used to record avoidance of mother, lack of closeness to mother and less distress in the presence of a stranger during mother's absence.

In keeping with the four-category model of adult attachment (Bartholomew & Horowitz, 1991), different combinations of anxious and avoidant attachment styles are classified as *secure attachment* (low anxiety and low avoidance), *preoccupied attachment* (high anxiety and low avoidance), *dismissing attachment* (low anxiety and high avoidance) and *fearful attachment* (high anxiety and high avoidance). Although researchers often use categorical labels for these four styles, people are actually distributed in a roughly bivariate normal way in the two-dimensional space defined by attachment anxiety and avoidance. There are no real categories or types within this conceptual space (Fraley & Waller, 1998). Also, attachment styles assessed with questionnaires differ in focus and method from the 'states of mind' measured in structured interviews that ask adults to recall and describe their attachment to parents (Bartholomew & Shaver, 1998; Shaver, Belsky, & Brennan, 2000). Nevertheless, a large body of literature has shown that self-reported attachment between adults predicts relationship variables, affect regulation and psychopathology as expected based on attachment theory (Carnelley, Pietromonaco, & Jaffe, 1994; Mikulincer et al., 2003; Nakash-Eisikovits, Dutra, & Westen, 2002; Noftle & Shaver, 2006). Furthermore, defensive strategies and unconscious processes expected in attachment theory have been associated with self-reported attachment styles in recent experimental studies (e.g. Mikulincer, Dolev, & Shaver, 2004; Mikulincer, Gillath, & Shaver, 2002; Shaver & Mikulincer, 2002).

Attachment and personality disorder

Anxious and avoidant attachment may provide a useful conceptual framework for understanding the interpersonal dysfunction that is salient in personality disorders (PDs; Bartholomew, Kwong, & Hart, 2001; Fonagy & Bateman, 2005; Meyer & Pilkonis, 2005). Dysfunctional interpersonal behaviours account for 45% of the diagnostic criteria for PD in the DSM-IV, far more than the remaining criteria devoted to maladaptive cognition (23%), affective disturbances (20%) or other behaviours (12%) (Pilkonis, 2002). Understanding how attachment and PD constructs are related could help to clarify how interpersonal problems take root during development and become central features in personality pathology (Lyddon & Sherry, 2001).

There have been few empirical studies investigating the relationship between attachment styles and PD. Brennan and Shaver (1998) used principal components factor analysis in a large college sample to show how two factors underlying DSM-III-R PD symptoms correspond closely with attachment styles hypothesised in the four-category model. One factor labelled 'insecurity' distinguished between secure and fearful attachment, and this dimension also loaded onto a PD factor that encompassed borderline, avoidant, paranoid and schizotypal symptoms. These Axis II disturbances are associated with marked worries about being abandoned (borderline PD), rejected (avoidant PD) or harmed by others (paranoid and schizotypal PD). As such, this alignment of PD and attachment constructs

fits well with attachment theory and emphasises how worries about close relationships are closely linked with emotional dysregulation. Brennan and Shaver found a second attachment dimension labelled 'defensive emotional style' that distinguished between dismissing and preoccupied styles, and this dimension loaded onto a factor that differentiated schizoid symptoms from dependent and histrionic symptoms.

By using attachment styles from the four-category model, these factor analyses identified broad associations between Axis II symptoms and attachment styles that are defined by different *combinations* of anxious and avoidant styles. As such, it is impossible to determine if PD symptoms were associated with the anxious or avoidant dimension, or both. Additional research is thus needed to determine how personality pathology aligns with the underlying anxious and avoidant attachment dimensions.

Although attachment theory views attachment and personality as related (Bartholomew et al., 2001; Lyddon & Sherry, 2001), the mechanisms linking attachment styles and PD symptoms remain unclear. It could be that attachment and PD overlap because both are influenced by a common third variable. For example, Brennan and Shaver (1998) showed that environmental factors such as parental divorce increased risk for the insecurity dimension and corresponding Axis II scores. Alexander (1992) hypothesised that insecure attachment and sexual abuse interact and together create developmental pathways leading to PD. Several studies link childhood abuse with subsequent PD (e.g. Cohen, Brown, & Smailes, 2001; Johnson et al., 2001) and insecure attachment (Allen, Hauser, & Borman-Spurrell, 1996; Mickelson, Kessler, & Shaver, 1997).

It also could be that the association between attachment style and PD is attributable to a shared set of genetic factors. Insofar as PD and insecure attachment are each linked with emotional dysregulation, for instance, both could be mediated by the same heritable differences in temperament or personality traits (Goldsmith & Harman, 1994). It is well established that genes account for 40–60% of the variability in normal personality traits (Plomin, DeFries, McClearn, & McGuffin, 2001), dimensionally defined PD trait scales (Jang, 2005) and to a lesser degree PD diagnoses in recent editions of the DSM (Nigg & Goldsmith, 1994). Although there is far less information on the heritability of attachment styles in adults, Brussoni, Jang, Livesley, and MacBeth (2000) found that genes accounted for 43, 25 and 37% of the variability in fearful, preoccupied and secure attachment assessed with the Relationship Scales Questionnaire (RSQ; Griffin & Bartholomew, 1994). Variability in dismissing attachment, in contrast, was found to be entirely attributable to environmental effects. But once again, these analyses did not assess the heritability of avoidant and anxious attachment, the defining dimensions in the four-category model of attachment.

The other available twin studies of attachment have all tested for heritable effects in children (Bakermans-Kranenburg, van IJzendoorn, Schuengel, & Bokhorst, 2004; Bokhorst et al., 2003; O'Connor & Croft, 2001). These studies used different assessment methods that include the Strange Situation (Ainsworth et al., 1978), the Attachment Q-Sort (Vaughn & Waters, 1990; Waters, 1995) and Cassidy and Marvin's (1992) preschool coding manual for behaviour in the Strange Situation. No significant genetic effects were found in these studies; instead, shared environmental influences explained between 32 and 59% of the variance. However, no firm conclusions can be drawn from these studies based on their small samples (ranging between 56 and 110 twin pairs) and corresponding limits to their power to detect genetic effects.

Given limited data on the heritability of adult attachment styles, the present study used a classic twins-reared-together design to estimate genetic and environmental effects

influencing avoidant and anxious attachment. The study then tested whether genetic and environmental effects associated with these underlying dimensions of attachment also influence corresponding dimensions of personality pathology. Anxious attachment and personality traits indexing emotional dysregulation were expected to share a common factor structure that might be linked by common etiological influences. This hypothesis was based on expectations that abandonment fears specific to attachment anxiety would be associated with other disturbances in emotion regulation. Avoidant attachment and personality traits indexing emotional inhibition and social avoidance were similarly tested for a shared factor structure and a common aetiology to account for links between corresponding attachment insecurity and PD.

METHOD

Participants

This study is based on 239 volunteer twin pairs from Vancouver, British Columbia, who were recruited in the mid-1990s through newspaper advertisements and media stories (Jang, Livesley, & Vernon, 2002). A total of 126 MZ twin pairs (78.6% female) and 113 DZ twin pairs (61% female pairs, 16% male pairs and 23% opposite-sex pairs) provided data for this study. Zygosity was determined by a questionnaire (Nichols & Bilbro, 1966) that is at least 95% accurate when compared to red blood cell polymorphism analyses (Kasriel & Eaves, 1976). The average age of co-twins was 30.9 years (SD = 12.1, range 16–79), and there were no group differences in age between MZ and DZ twins. To avoid systematic biases in twin analyses (e.g. based on birth order), siblings within each twin pair were randomly designated as Twin 1 and Twin 2.

Instruments

Attachment
Although anxious and avoidant attachment styles in adulthood are now commonly measured with the Experiences in Close Relationships Inventory (ECR; Brennan et al., 1998), this instrument was not available when the study sample was assessed. Internally reliable anxious and avoidant attachment scales were derived instead from a subset of items from the RSQ (Griffin & Bartholomew, 1994). Several items from the RSQ and ECR are identical and the remaining RSQ items were selected based on similarities with matching items on the ECR. For example, the RSQ item ('I worry that others do not value me as much as I value them') was selected for the anxious attachment scale based on its close correspondence with an ECR item ('I often wish that my partner's feelings for me were as strong as my feelings for him/her'). Anxious attachment was thus assessed with RSQ items 9, 11, 16, 18, 21, 23, 25 and 28. High scores on this eight-item scale (alpha = 0.88) indicate elevated worries about separation and fears of abandonment. Avoidant attachment was measured with RSQ items 1, 3, 10, 13, 15, 20, 24, 26 and 30. High scores on this nine-item scale (alpha = 0.85) reflect discomfort with intimacy and avoidance of emotional dependence on others. Respondents were instructed to rate individual items according to their experiences in close relationships using a five-point scale (1 = not at all like me, 5 = very much like me).

The anxious and avoidant scales derived from RSQ items have internal consistency (alpha > 0.85) that is more acceptable than the internal consistency of the standard RSQ

scales (where alphas range from 0.31 to 0.46; Griffin & Bartholomew, 1994). The newly created anxious and avoidant attachment scales were independently validated in a college sample ($N = 307$) by comparing them with scales on the ECR. Convergence between corresponding avoidant attachment scales ($r = 0.92$) and anxious attachment scales ($r = 0.91$) was quite satisfactory. The relationship between anxiety and avoidance on the newly constructed RSQ scales ($r = 0.37$) was similar to the relationship between these two dimensions on the ECR ($r = 0.29$). In other words, the alignment of anxious and avoidant dimensions was similar regardless of the measure used.

Personality disorder

The Dimensional Assessment of Personality Problems (DAPP; Livesley & Jackson, in press; Livesley, Jackson, & Schroeder, 1992) uses self-report items to measure 18 dimensions of personality disturbance: affective lability, anxiousness, callousness, cognitive distortion, compulsivity, conduct problems, identity problems, insecure attachment, intimacy problems, narcissism, oppositionality, rejection, restricted expression, self-harm, social avoidance, stimulus seeking, submissiveness and suspiciousness. Each dimension is assessed with 16 items (except self-harm and suspiciousness which have 14 items each) that are scored on a five-point scale (1 = very unlike me, 5 = very like me). The DAPP has demonstrated factorial validity and excellent psychometric properties: internal consistency alphas range from 0.81 to 0.94, and test–retest reliabilities over a 3-week period range from 0.81 to 0.93. Factor analysis of the DAPP scales has revealed four underlying dimensions that have been named emotional dysregulation, inhibitedness, dissocial behaviour and compulsivity (Livesley, 1991; Livesley, Jang, & Vernon, 1998).

Procedure

Twin pairs were instructed to complete self-report questionnaires at home independently from each other in a nondistracting setting and return completed questionnaires by mail. Informed consent was obtained in writing and assessment procedures were approved by the local Institutional Review Board. Because complete data were obtained for 99.2% for the variables investigated here, there were virtually no missing data in this study.

Data analysis

To determine how attachment and PD dimensions align with each other, these constructs were examined using principal components factor analysis with varimax rotation. Next, univariate biometric analyses were used to estimate genetic and environmental contributions to anxious and avoidant attachment, and multivariate models were used to determine whether genetic or environmental factors account for associations between attachment and PD dimensions. Raw data were used to generate the variance–covariance matrices needed for these statistical analyses. Although a variance–covariance matrix by itself could be entered into each statistical analysis instead, raw data are preferable because they can be used to manage potential problems arising from missing data. Age and gender were included in all twin models as covariates to remove possible influences that could bias estimates of genetic and environmental effects (see McGue & Bouchard, 1984). We controlled for linear effects of age because they were most pronounced on the DAPP (quadratic effects, in contrast, tended to be nonsignificant or weak in magnitude).

Univariate twin models

Structural equation models were used to estimate additive genetic, shared environmental and nonshared environmental effects on anxious and avoidant attachment. Additive genetic effects (A) passed directly from parents to children are broadly indicated when correlations between MZ twin pairs (who share 100% of their genes) are greater than correlations for DZ twin pairs (who share on average 50% of their genes). Interactive genetic effects may also contribute to estimates of A, but these are difficult to distinguish from simple additive effects in the sample size available here. Shared environmental effects (C) that have the same effects on twin pairs are indicated when correlations for MZ and DZ twins are similar in magnitude. To the extent that parental divorce, illness or death of a parent or family socioeconomic status affects co-twins in the same way, they would be attributable to shared environmental effects. Nonshared environmental influences (E) that cause twin pairs to differ from each other are estimated as the residual variance after the effects of A and C have been removed. As such, E also includes measurement error.

The relative contribution of the A, C and E variance components was estimated in a saturated ACE model, and the significance of individual components was tested in reduced models that removed the effects of (1) additive genetic variance (CE model), (2) shared environmental variance (AE model) and (3) both additive genetic and shared environmental variance (E model). When saturated and reduced models were compared, a significant increase in the likelihood-ratio χ^2 indicated a worse fit when a specific variance component was removed. Model fit was also evaluated using Akaike's Information Criterion (Akaike, 1987) ($AIC = \chi^2 - 2df$). Smaller AIC values indicate better fits than larger values. Reduced models that achieved parsimony by accounting for the observed variance with the fewest number of variance components were accepted when they yielded the smallest AIC value and did not significantly increase χ^2 values. All model fitting was conducted with the computer program *Mx* (Neale, 1997).

Multivariate twin models

The degree to which A, C and E effects are shared between attachment and personality pathology was estimated with genetic and environmental correlation coefficients. These coefficients are estimated by comparing MZ and DZ cross-twin correlations (e.g. between avoidant attachment in Twin 1 and intimacy problems in Twin 2). If MZ cross-correlations are greater than DZ cross-correlations, then genetic contributions to the covariance of measures are indicated. A standard Cholesky decomposition model (Neale & Cardon, 1992) was used to assess associations across twins between attachment and PD dimensions. This method yields genetic correlations (r_A) that estimate the extent to which variable *X* and variable *Y* are influenced by a common set of genes. Genetic correlations, however, do not reflect the magnitude of genetic contributions to the individual traits under investigation (which may be high or low). To obtain a more complete estimate of how much genetic factors influence phenotypic correlations, bivariate heritability estimates ($a_{BIV}^2 = a_X \times a_Y \times r_A$) are calculated to indicate the heritability of the two variables and the genetic correlation (Plomin & DeFries, 1979). Bivariate models also yield shared environmental correlations (r_C) and nonshared environmental correlations (r_E) that can be used to calculate bivariate shared environmental effects ($c_{BIV}^2 = c_X \times c_Y \times r_C$) and bivariate nonshared environmental effects ($e_{BIV}^2 = e_x \times e_Y \times r_E$). These bivariate estimates of heritability, shared environmental effects and nonshared environmental effects each contribute to the correlation between variables. Their sum equals the observed phenotypic correlation ($r_{PHEN} = a_{BIV}^2 + c_{BIV}^2 + e_{BIV}^2$).

Multivariate ACE models are normally compared with reduced models that delete parameter estimates for (1) common genetic effects (i.e. only r_C and r_E are specified), (2) bivariate shared environmental influences (only r_A and r_E are specified) and (3) bivariate nonshared environmental factors (only r_A and r_C are specified). Multivariate models yielding the lowest AIC values and nonsignificant changes in χ^2 statistics identify reduced models with the most parsimonious fit to the observed data.

RESULTS

Table 1 reports a zero-order correlation matrix for attachment and PD dimensions. All correlation coefficients greater than 0.09 are significant ($p < 0.05$). Table 2 reports results from principal components factor analysis of attachment and PD dimensions based on all 478 participants. Results in Table 2 were highly similar to factor structures obtained from MZ and DZ pairs separately. As expected, anxious attachment loaded on the DAPP emotional dysregulation factor. Personality pathology dimensions (and representative subscales) indexed on this factor include social avoidance (social apprehensiveness, defective social skills and worry about interpersonal hurt), identity problems (anhedonia, labile self-concept, chronic emptiness and boredom), anxiousness (guilt proneness, indecisiveness and trait anxiety), affective lability (affective over-reactivity, hypersensitivity and labile anger), cognitive distortion (depersonalisation, schizotypal cognition and brief stress psychosis), submissiveness (diffidence, suggestibility and need for advice), oppositionality (passivity, oppositional behaviour and lack of organisation), self-harm (ideas of self-harm and self-damaging behaviours), narcissism (need for adulation, attention seeking and grandiosity) and suspiciousness (hypervigilance and suspiciousness). Even though insecure attachment on the DAPP (made up of subscales for separation protest, secure base, feared loss, intolerance of aloneness and proximity seeking) seems to reflect both anxious and avoidant attachment, this dimension of personality pathology aligned most with anxious attachment.

The inhibitedness factor had loadings from avoidant attachment and PD dimensions measuring intimacy problems (desire for improved attachment relationships, inhibited sexuality and avoidant attachment), restricted expression (reluctant self-disclosure, restricted expression of anger and self-reliance) that define the DAPP inhibitedness factor along with identity problems and social avoidance dimensions. Convergence between avoidant attachment on the DAPP and modified RSQ scales, of course, is not surprising. Alignment between avoidant attachment and other forms of personality pathology is more noteworthy and reflects meaningful links with a wider range of psychopathology. However, neither avoidant nor anxious attachment dimensions showed any relationship to PD scales indexing dissocial behaviour (interpersonal violence, impulsivity, addictive behaviours and remorselessness) or compulsivity (orderliness, precision and conscientiousness), suggesting that these dimensions of personality pathology are unrelated to attachment styles.

Table 3 reports estimates of additive genetic (a^2), shared environmental (c^2) and nonshared environmental (e^2) effects on the two attachment dimensions. Approximately 40% of individual differences in anxious attachment were attributable to genetic influences. The AE model fit the data as well as the saturated ACE model, $\Delta\chi^2(df = 1) = 0.002$, ns, AIC $= -1.998$, but was preferred because it was more parsimonious. The CE model was rejected because its relative fit was worse when compared with the ACE model, $\Delta\chi^2(df = 1) = 3.104$, $p = 0.078$, AIC $= 1.104$. These

Table 1. Phenotypic correlations between anxious and avoidant attachment dimensions and DAPP personality disorder dimensions (N = 478)

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.
1. Anxious attachment	—																			
2. Avoidant attachment	0.34	—																		
3. Restricted expression	0.27	0.59	—																	
4. Intimacy problems	0.28	0.50	0.55	—																
5. Social avoidance	0.52	0.41	0.56	0.47	—															
6. Identity problems	0.57	0.39	0.50	0.47	0.70	—														
7. Anxiousness	0.55	0.28	0.39	0.34	0.68	0.76	—													
8. Affective lability	0.47	0.17	0.13	0.19	0.46	0.60	0.70	—												
9. Insecure attachment	0.47	-0.05	0.09	0.02	0.35	0.44	0.49	0.50	—											
10. Cognitive distortion	0.53	0.30	0.36	0.33	0.53	0.69	0.69	0.61	0.51	—										
11. Submissiveness	0.39	0.24	0.46	0.33	0.62	0.54	0.65	0.38	0.33	0.51	—									
12. Oppositionality	0.41	0.19	0.35	0.31	0.51	0.62	0.55	0.47	0.40	0.60	0.49	—								
13. Self-harm	0.37	0.25	0.27	0.30	0.34	0.53	0.43	0.41	0.28	0.50	0.23	0.31	—							
14. Narcissism	0.44	-0.01	0.13	0.11	0.37	0.44	0.52	0.48	0.46	0.43	0.32	0.52	0.26	—						
15. Suspiciousness	0.45	0.37	0.41	0.29	0.42	0.57	0.51	0.50	0.40	0.52	0.25	0.38	0.37	0.42	—					
16. Callousness	0.34	0.24	0.21	0.19	0.26	0.40	0.23	0.30	0.27	0.34	0.06	0.41	0.28	0.46	0.55	—				
17. Rejection	0.14	0.07	0.00	0.03	0.03	0.21	0.15	0.32	0.16	0.16	-0.09	0.29	0.12	0.44	0.41	0.52	—			
18. Conduct problems	0.23	0.22	0.14	0.13	0.17	0.30	0.15	0.32	0.15	0.33	0.06	0.38	0.30	0.27	0.37	0.59	0.37	—		
19. Stimulus seeking	0.16	0.02	0.06	-0.02	0.01	0.17	0.08	0.26	0.19	0.29	0.07	0.39	0.21	0.39	0.25	0.44	0.33	0.52	—	
20. Compulsivity	-0.06	0.04	0.06	-0.05	-0.03	-0.09	0.06	-0.02	-0.03	-0.07	0.04	-0.42	-0.09	-0.11	0.12	-0.11	0.00	-0.20	-0.26	—

Table 2. Alignment between anxious and avoidant attachment dimensions and four higher-order factors underlying DAPP personality disorder dimensions ($N = 478$)

	Emotional dysregulation	Inhibitedness	Dissocial behaviour	Compulsivity
Anxious attachment	**0.632**	0.249	0.185	0.004
Avoidant attachment	0.046	**0.817**	0.163	−0.097
Restricted expression	0.222	**0.807**	0.026	−0.000
Intimacy problems	0.174	**0.756**	0.013	0.084
Social avoidance	**0.648**	**0.533**	−0.046	0.050
Identity problems	**0.723**	**0.461**	0.194	0.085
Anxiousness	**0.864**	0.274	0.030	−0.065
Affective lability	**0.752**	0.026	0.290	−0.037
Insecure attachment	**0.745**	−0.194	0.154	−0.007
Cognitive distortion	**0.737**	0.276	0.211	0.123
Submissiveness	**0.670**	0.346	−0.233	0.123
Oppositionality	**0.587**	0.226	0.269	**0.540**
Self-harm	**0.421**	0.302	0.242	0.093
Narcissism	**0.625**	−0.116	**0.429**	0.110
Suspiciousness	**0.474**	0.332	**0.547**	−0.273
Callousness	0.193	0.199	**0.797**	0.063
Rejection	0.127	−0.082	**0.764**	−0.140
Conduct problems	0.093	0.183	**0.714**	0.278
Stimulus seeking	0.126	−0.076	**0.623**	**0.432**
Compulsivity	0.011	0.028	−0.066	**−0.883**
% Variance explained	27.5%	15.5%	15.2%	7.6%

combined results indicate little influence of shared environmental effects on attachment anxiety. In contrast, correlations for avoidant attachment in MZ and DZ twins (0.285 and 0.328, respectively) suggest no additive genetic influence. The CE model for avoidant attachment fit as well as the saturated ACE model, $\Delta\chi^2(\mathrm{d}f = 1) = 0.000$, ns, AIC = −2.000, thereby providing the most parsimonious explanation of the data. Overall, shared environmental effects accounted for 30% of the variance in avoidant attachment.

Table 4 reports correlations between anxious attachment and DAPP dimensions indexing emotional dysregulation and decomposes these correlations into genetic and environmental components. Although they share a common underlying structure, phenotypic correlations between attachment and PD dimensions (mean $r = 0.47$, range = 0.37–0.57) indicate that these variables were not redundant. Table 4 does not include bivariate shared environmental effects (c_{BIV}^2) because they explained little or no covariation between anxious attachment and personality pathology in multivariate models. This outcome largely reflects the fact that shared environmental factors accounted for little variance in these individual variables in univariate models. The mean genetic correlation (r_A) was 0.75 (range = 0.57–1.00), and 95% confidence intervals (CIs) ranged on average between 0.49 and 1.00. Because ACE models including r_C were not considered, we tested for significant differences between ACE models that specified both r_A and r_E versus those that specified only r_E. When the parameter for r_A was dropped, significant change statistics (mean $\Delta\chi^2 = 23.62$, range = 13.47–39.35; all $\Delta\mathrm{d}f = 1$, $p < 0.01$) indicated that reduced models fit significantly worse than when bivariate genetic effects were included.[1]

[1] We do not report AIC statistics in this context because they are effectively redundant with the $\Delta\chi^2$ statistic when only one reduced model is considered.

Table 3. Estimated genetic (a^2), shared environmental (c^2) and nonshared environmental (e^2) influences on anxious and avoidant attachment in 126 MZ and 113 DZ twin pairs

	r_{MZ}	r_{DZ}	a^2	c^2	e^2	$-2ll$	df	$\Delta\chi^2$	Δdf	p	AIC
Anxious attachment	0.442	0.242									
ACE (95% CI)			0.397 (0.000–0.531)	0.009 (0.000–0.356)	0.594 (0.469–0.754)	3206.885	476				
AE (95% CI)			0.407 (0.263–0.531)	— (0.000–0.000)	0.593 (0.469–0.737)	3206.887	477	0.002	1	ns	−1.998
CE (95% CI)			— (0.000–0.000)	0.306 (0.187–0.416)	0.694 (0.584–0.813)	3209.989	477	3.104	1	0.078	1.104
Avoidant attachment	0.285	0.328									
ACE (95% CI)			0.000 (0.000–0.342)	0.301 (0.001–0.411)	0.699 (0.576–0.818)	3182.636	476				
AE (95% CI)			0.330 (0.188–0.456)	— (0.000–0.000)	0.670 (0.544–0.813)	3186.654	477	4.018	1	0.045	2.018
CE (95% CI)			— (0.000–0.000)	0.301 (0.182–0.411)	0.699 (0.589–0.818)	3182.636	477	0.000	1	ns	−2.000

Table 4. Genetic and environmental correlations between anxious attachment and personality disorder dimensions in 126 MZ and 113 DZ twin pairs

	Genetic and environmental correlations with anxious attachment					
	r_{PHEN}	r_A	r_E	a^2_{BIV}	e^2_{BIV}	a^2_{BIV}/r_{PHEN}
Affective lability	0.47	0.62	0.33	0.28	0.18	0.60
Anxiousness	0.55	1.00	0.37	0.37	0.21	0.67
Cognitive distortion	0.53	1.00	0.31	0.33	0.18	0.62
Identity problems	0.57	0.81	0.34	0.39	0.13	0.68
Insecure attachment	0.47	0.75	0.32	0.28	0.17	0.60
Narcissism	0.44	0.57	0.29	0.23	0.17	0.52
Oppositionality	0.41	0.58	0.30	0.23	0.16	0.56
Self-harm	0.37	0.76	0.18	0.24	0.16	0.65
Social Avoidance	0.52	0.72	0.35	0.33	0.19	0.64
Submissiveness	0.39	0.81	0.20	0.30	0.12	0.77
Suspiciousness	0.44	0.64	0.31	0.26	0.18	0.59

r_{PHEN} = phenotypic correlations; r_A = additive genetic correlations; r_E = nonshared environmental correlations; a^2_{BIV} = bivariate heritability; e^2_{BIV} = nonshared environmental effects; a^2_{BIV}/r_{PHEN} = the proportion of covariance attributable to shared genetic effects.

Nonshared environmental correlations (r_E) were consistently lower (mean = 0.30, range = 0.18–0.37), and 95% CIs ranged on average between 0.15 and 0.44. When bivariate heritability was estimated as a proportion of total covariation (a^2_{BIV}/r_{PHEN}), common genetic effects explained on average 63% of the total covariation between anxious attachment and DAPP measures indexing emotional dysregulation.

As reported above, results from univariate analyses indicate no influence of genetic effects on avoidance and little or no influence of the shared environment on DAPP scales loading onto the inhibitedness factor. Logically, this leaves the nonshared environment as the only potential source for bivariate effects across avoidance and corresponding PD variables.

DISCUSSION

To our knowledge this article reports the first heritability estimates for self-reported anxious and avoidant attachment in adults. Whereas attachment anxiety was clearly influenced by genetic factors, avoidant attachment seemed influenced entirely by environmental effects. Based on these findings, we were able to show that the association between anxious attachment and personality pathology was largely explained by genetic factors. In contrast, it appears most likely that associations between avoidant attachment and personality pathology are attributable to nonshared environmental influences.

Anxious attachment includes abandonment fears and difficulties regulating worries about the availability of attachment figures. Given that heritable factors accounted for 40% of the phenotypic variance in anxious attachment, genetic effects probably establish different degrees of susceptibility to emotional difficulties in close relationships that heighten or attenuate these fears and worries. For example, people with high genetic susceptibility to attachment anxiety could be more prone to worry about relationships with parents or romantic partners who are inconsistently available. People with lower susceptibility to attachment anxiety may be less affected by these experiences. Shared environmental factors did not account for any similarity in attachment anxiety between

twins. Instead, nonshared environmental factors (e.g. unique relationships with family members, friends and romantic partners, plus measurement error) accounted for the remaining variation in anxious attachment. Twins may be especially likely to differ from each other on attachment anxiety based on differences in the close friends and romantic partners they each choose in adolescence and adulthood.

In marked contrast to anxious attachment, avoidant attachment seemed to be unaffected by genes. As in previously published studies, environmental factors accounted for the vast majority of the effects related to avoidance. For example, when Waller and Shaver (1994) investigated the heritability of adult 'romantic love styles'—probably proxy variables for attachment security—they found sizable shared environmental effects on a range of attitudes toward loving relationships and no evidence of genetic effects. In a preschool sample of 110 twin pairs, O'Connor and Croft (2001) used Strange Situation procedures adapted for preschool children to produce categorical classifications and a continuous measure of insecure attachment. When the continuous measure was used, shared environmental factors accounted for 31% of the variance, approximately the same amount attributable to the shared environment in avoidant attachment in adults. Evidence for genetic influences was weak regardless of how attachment was measured. Nevertheless, it remains difficult to interpret findings across studies because different measures were used, and it is unclear how well O'Connor and Croft specifically assessed avoidant attachment. As indicated above, other studies failed to find any genetic influences on attachment in children (Bakermans-Kranenburg et al., 2004; Bokhorst et al., 2003) but their samples may have lacked adequate statistical power.

Our finding that genetic factors have no influence on avoidant attachment is intriguing. At the same time that anxious attachment can be attributed to a mixture of heritable and environmental influences, the social environment alone appears to determine whether avoidant strategies for dealing with that anxiety are encouraged or not. Consistent with attachment theory, avoidance initially learned in parent–child relationships (Ainsworth et al., 1978) often continues to be expressed and reinforced in close relationships in adolescence and adulthood (Lyddon & Sherry, 2001). As avoidant youth become parents themselves, avoidant approaches to affect regulation often foster attachment avoidance in their children (Fonagy, Steele, & Steele, 1991). Although avoidant attachment may persist over time as the child develops into an adult, avoidance may also subside if young people develop significant relationships with friends or romantic partners who are more sensitive and responsive to their needs, thereby reducing abandonment fears and reinforcing more secure forms of attachment.

If avoidant attachment is in fact more influenced by shared environment than by genes, it remains unclear what nongenetic familial experiences might account for it. O'Connor and Croft (2001) hypothesised that parental sensitivity and responsiveness to the attachment needs of children might represent a shared environmental factor that could explain similarities in MZ and DZ twin pairs. Allowing for ways in which parents treat individual twins differently based on their specific needs, parental sensitivity and responsiveness are nevertheless influenced by 'internal working models'—i.e. the parents' mental representations of self and other—that are thought to have a relatively stable influence on close relationships over time (Bretherton & Munholland, 1999). If internal working models in parents reflect negative representations of others, they often manifest in an emotional distance or tendency to reject others, thereby producing a shared environmental effect. If internal working models in parents reflect negative self-representations instead, they create disturbances in affect regulation and inconsistent parenting behaviours that in turn reduce parental sensitivity and responsiveness. In meta-analyses of the intergenera-

tional effects of adult attachment representations and parental responsiveness (van IJzendoorn, 1995; van IJzendoorn & Bakersmans-Kranenburg, 1996), parents' internal working models assessed with the adult attachment interview (AAI; George, Kaplan, & Main, 1985) were reliably linked with insecure attachment in children. Parents' internal working models were also linked with sensitive and responsive parenting behaviours that play a key role in shaping children's attachment. However, parents' internal working models accounted for only about 12% of the variance in children's attachment orientations, thus indicating that more work is needed to clarify how avoidant attachment is transmitted across generations.

Insecure attachment and personality pathology

When attachment and PD dimensions were investigated with factor analysis, results were consistent with expectations. Anxious attachment loaded onto the DAPP emotional dysregulation factor, and avoidant attachment loaded onto the inhibitedness factor. Emotional dysregulation is most correlated with neuroticism in the five-factor model of personality (Schroeder, Wormworth, & Livesley, 2002), and corresponding PD dimensions and personality facets have been shown to share common genetic influences (Jang & Livesley, 1999). Anxious attachment is correlated with neuroticism (Crawford et al., 2006; Noftle & Shaver, 2006) and this association probably can be explained by common genetic effects. When inhibitedness is correlated with the big five dimensions, it is most associated with low extraversion and low openness to experience (Schroeder et al., 2002).

Avoidant attachment is harder to place within the framework of the five-factor model of personality. Correlations between avoidant attachment and the five higher order dimensions (neuroticism, extraversion, openness, conscientiousness and agreeableness) range between 0.19 and -0.29 (Crawford et al., 2006; Noftle & Shaver, 2006), thus suggesting that avoidant attachment is largely independent of normal personality traits. In comparison, correlations are higher between avoidant attachment and the four DAPP dimensions loading on the inhibitedness factor (mean $r = 0.47$, range $= 0.39$–0.59), thus suggesting that avoidance may be more closely related to this dimension of abnormal personality instead.

Avoidant attachment was most associated with PD dimensions labelled intimacy problems and restricted expression ($r_{PHEN} = 0.50$ and 0.59, respectively) and somewhat less correlated with social avoidance and identity problems ($r_{PHEN} = 0.41$ and 0.39, respectively). These results fit well with outcomes predicted by attachment theory. High scores on the avoidant attachment dimension reflect 'deactivating strategies' (Cassidy & Kobak, 1988) that are used to reduce anxiety and other negative emotions in close relationships. It thus makes sense that avoidant attachment would correlate with restricted expression and intimacy problems that each reflect ways in which people down-regulate emotion and also limit emotional and sexual intimacy in relationships.

Multivariate models suggest that covariation between avoidant attachment and inhibitedness is attributable to nonshared environmental influences. Associations between these variables thus appear to stem from experiences that are unique to individual twins (e.g. differential neglect or rejection of twins by caregivers or romantic partners) instead of experiences within the familial environment that are shared. However, this conclusion is based primarily on findings from univariate ACE models that show that avoidance had shared environmental but not genetic effects, and that DAPP dimensions indexing

inhibitedness had genetic but not shared environmental effects, thus leaving the nonshared environment as the only variance component available to be shared across constructs.

Anxious attachment was associated with a notable range of personality pathology indexed on the emotional dysregulation factor. People with elevated attachment anxiety often use 'hyperactivating strategies' (Cassidy & Kobak, 1988; Mikulincer & Shaver, 2003) to manage fears of being abandoned (i.e. when they do not contain those fears with avoidant strategies). Hyperactivating strategies lead people to seek as much proximity with attachment figures as possible and require constant vigilance for possible threats, separations or betrayals that might jeopardise important attachment relationships. Rather than reducing negative affect, these strategies usually elicit thoughts and expectations that exacerbate it instead. As such, it makes sense that anxious attachment loaded onto the emotional dysregulation factor. Etiologically, genetic factors accounted for approximately 63% of the covariation between the anxious attachment and PD measures loading on this factor. There was notably less influence from the nonshared environment and none from the shared environment.

It comes as no surprise, of course, that anxious attachment correlated with the DAPP insecure attachment dimension. It also makes sense that attachment anxiety correlates with the affective lability and self-harm that are prominent in borderline PD, which itself may be fundamentally defined by abandonment fears (Gunderson, 1996). It may be more notable that anxious attachment was associated with personality pathology that manifests in very different forms. Anxious attachment correlated not just with oppositionality but also with submissiveness. Similarly, anxious attachment correlated with the inflated self-satisfaction in narcissism and the lack of self-fulfilment associated with identity problems. Given how anxious attachment correlated with such different kinds of psychopathology, it could be that different sets of genes contribute to the specific associations observed between these variables. Eight of the eleven DAPP dimensions indexing emotion dysregulation have been shown to have additive genetic effects even when the variance from that higher order factor is controlled (Livesley et al., 1998), thus suggesting the presence of unique additive genetic effects among these variables. More work is needed, however, to clarify whether these unique effects are independent of each other and also whether they are associated with genetic influences in anxious attachment.

Based on the available data one could reasonably argue that anxious attachment, emotional dysregulation, and even their overlap can be explained by the higher order trait of neuroticism assessed in dimensional models of normal personality. As noted above, neuroticism is known to correlate with DAPP scales indexing emotional dysregulation (Jang & Livesley, 1999) and anxious attachment (Crawford et al., 2006; Noftle & Shaver, 2006). Even if neuroticism does provide an underlying genetic foundation for anxious attachment and emotional dysregulation, it is important to recognise that these constructs remain distinct. In theoretical terms, anxious attachment is expected in close relationships with parents or romantic partners but not in relationships that are less close. Anxious attachment may be absent in situations that do not involve close relationships (e.g. academic testing) where neuroticism might be relevant. Insofar as anxious attachment thus occurs under specific circumstances, it differs from the general predisposition to negative emotions measured as neuroticism. Differences between attachment anxiety and neuroticism are also apparent empirically. Correlations between anxious attachment and neuroticism rarely exceed 0.5 (Shaver & Mikulincer, 2004), and associations between anxious attachment and PD dimensions indexing emotional dysregulation are similar in magnitude. Furthermore, theoretically important effects of attachment anxiety are obtained

even when measures of neuroticism are statistically controlled (e.g. Mikulincer et al., 2002; Simpson, Rholes, Campbell, Tran, & Wilson, 2003).

Significance and limitations

As indicated above, this appears to be the first study to estimate the heritability of anxious and avoidant attachment. However, the use of a volunteer twin sample may affect the generalisability of the findings, especially insofar as self-selection biases in volunteer samples may inflate estimates of additive genetic effects (Kendler & Holm, 1985). Our sample size was larger than other twin samples used to investigate attachment (e.g. O'Connor & Croft, 2001), but it nevertheless lacked the statistical power needed to detect modest genetic or environmental effects. Although gender effects were statistically removed, women were over-represented in our twin sample, thus potentially limiting the generalisability of our findings. Given the degree to which women may be more focused on relationships (e.g. Cross & Madson, 1997), research is needed to test for potential gender differences in links between attachment dimensions and PDs. Additionally, our results were based on self-report measures and thus may not generalise to attachment constructs investigated with the AAI. This structured interview focuses on how adults remember and describe early attachment experiences with primary caregivers and thus differs from self-report scales that investigate close relationships between adults. Moreover, little is known about the degree to which adult attachment styles measured with self-report measures trace back to childhood treatment by parents, as implied by attachment theory.

This study showed how common genetic effects accounted for a substantial portion of the correlation between anxious attachment and PD dimensions associated with emotional dysregulation. Based on the available evidence it appears that bivariate nonshared environmental effects alone explained the association between attachment avoidance and inhibitedness. Attachment dimensions were unrelated to dissocial behaviour on the DAPP, a higher order factor indexing interpersonal violence, impulsivity, addictive behaviours, remorselessness and recklessness. Brennan and Shaver (1998) found that a similar PD factor labelled 'psychopathy' was unrelated to attachment constructs. Compulsivity on the DAPP represents an additional higher-order PD factor that also appears unrelated to attachment. Differences in attachment styles thus may contribute more to our understanding of the emotional dysregulation and inhibitedness PD factors than to dissocial behaviour and compulsivity factors.

The present study provides a useful starting point for future research by showing that genetic and environmental effects are both likely to influence attachment patterns and PDs. It also indicates that the relative contributions of genes and environment may differ for anxious and avoidant attachment and their association corresponding dimensions of personality pathology. Now that twin studies are beginning to investigate insecure attachment, behavioural genetic research promises to increase our understanding of how it is transmitted across generations, how it is reinforced or maintained in close relationships and how it influences and is influenced by related variables such as personality.

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